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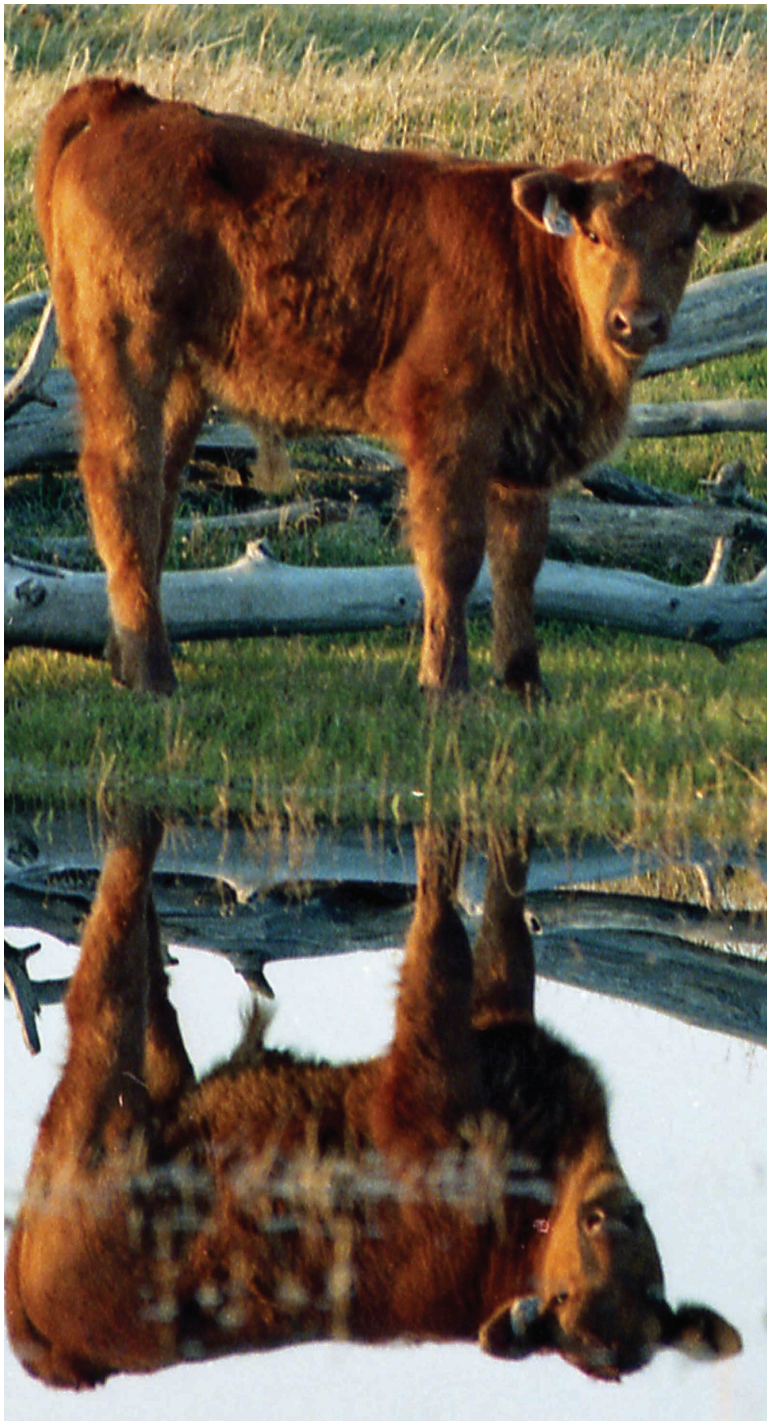

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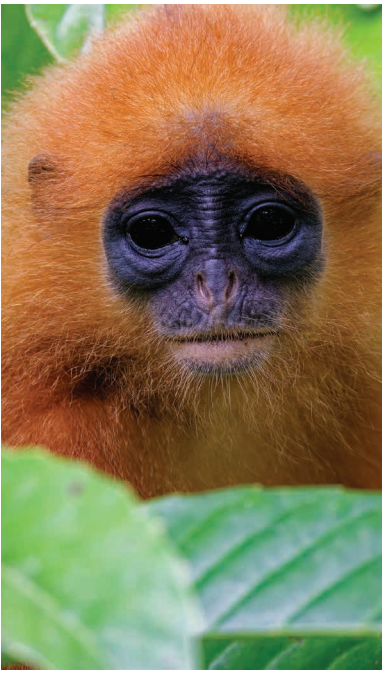

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06 2023

EDITORIAL
ÉDITORIAL

Mentorship has never been more important in the veterinary profession

Le mentorat n'a jamais été aussi important au sein de la profession vétérinaire

Dr./Dr John Kastelic

Dr./Dr Tim Ogilvie

For some veterinarians, especially those with much experience, "mentorship" meant getting keys to the practice vehicle, a gas card, and a county map. That really wasn't adequate at the time and is currently woefully insufficient.

The veterinary profession can provide an unprecedented range of services, our clients have increasing demands (and Dr. Google at their fingertips) and many practices are understaffed. Consequently, new graduates entering veterinary practice often have a mixture of exhilaration and trepidation! Having the support of a robust mentorship program is a high priority, with inadequate mentorship cited as an important factor by those leaving their first job (1). In addition, according to a recent American Animal Hospital Association (AAHA) survey, more than 30% of veterinary practitioners are considering changing jobs, with a desire for better career development part of their motivation (1). Therefore, mentorship programs are critical in recruiting new clinicians and retaining current ones. Typical mentees include new graduates, persons rejoining practice after an extended interval away, or those who are switching career paths (*e.g.,* changing focus to a different species or pursing a leadership role) (2).

A successful mentorship program has 3 key components: setting clear expectations, creating an action plan, and maintaining consistent communication (1). In brief: both mentor and mentee must discuss and understand what is needed and can be delivered; the action plan should include goals, timelines, and resources needed; and both parties must be committed to honest and open communications. Some useful suggestions include creating a written agreement outlining the mentorship program, having regularly scheduled meetings, and moving meetings offsite to minimize disruptions (1). Also, confidentiality, clear

Il y a un certain nombre d'années (les vétérinaires ayant beaucoup d'expérience s'en souviendront), le « mentorat » signifiait obtenir les clés du véhicule de la pratique, une carte pour payer l'essence et une carte routière de la région. Ce n'était pas adéquat à l'époque, et ce serait terriblement insuffisant aujourd'hui.

La profession vétérinaire offre une gamme de services sans précédent, nos clients sont de plus en plus exigeants (avec le Dr Google toujours accessible en quelques clics) et de nombreuses pratiques manquent de personnel. Les vétérinaires qui amorcent leur carrière ressentent souvent un mélange d'exaltation et de trépidation, et leur offrir le soutien d'un programme de mentorat solide devrait être une priorité. En effet, le manque de mentorat est cité comme un facteur important par ceux qui quittent leur premier emploi (1). Un récent sondage mené par l'American Animal Hospital Association (AAHA) révèle que plus de 30 % des vétérinaires envisagent de changer d'emploi, motivés notamment par le désir d'un meilleur épanouissement professionnel (1). Le mentorat s'avère essentiel pour le recrutement de nouveaux médecins vétérinaires et pour la

boundaries and regular feedback (both directions) are really important (2).

Both the mentor and mentee each need to contribute to make mentorship function well, and both should expect to derive benefits. In that regard, serving as an effective mentor should provide a sense of contribution and satisfaction (2), plus the mentor almost inevitably acquires some new information.

Although a mentor often provides advice, in some cases, they can act as a coach, discuss the situation with the mentee, and guide them to consider the situation and potential solutions. It has been stated that "enabling an individual to identify and solve their own challenges makes resolution more likely" (2).

In its classical form, mentorship of veterinarians focused on clinical skills, particularly for new graduates, who had much knowledge but lacked practical experience (3). In addition, good mentorship also addresses communication, leadership, and personal wellness, areas in which many new graduates feel very insecure (3). In that regard, a veterinarian with considerable experience mentoring new veterinary graduates commented that "90 percent of the topics worked through are non-clinical yet impact their working life significantly" (2).

In a practice with several veterinarians, more than 1 can serve as a mentor. Furthermore, a mentee may have more than 1 mentor (known as "mosaic mentoring"), which can be very valuable as mentors likely vary in knowledge, skills, and approaches. For example, 1 mentor may focus on surgery, another on internal medicine, and a third on communications and client interactions. Although having a member of the same practice as mentor is most common, a mentor could belong to another practice or mentorship can be delivered remotely (various kinds of virtual mentoring services are available). The Canadian Veterinary Medical Association (CVMA) website has a mentoring section with over 15 documents available for download, plus links to 2 online resources (4). In addition, the CVMA recently launched the pilot version of a mentorship program (5).

In summary, mentorship has never been more important, given the complexity of our profession, client demands and expectations, and challenges in hiring and retaining staff. "Upping your game" in mentoring is a key part of successful practice management. Although this editorial has focused on mentoring veterinarians, there are clearly benefits to mentoring Animal Health Technicians (AHT) and other clinic staff, and many resources are available.

REFERENCES

1. Trends [Internet]. Why your practice needs a mentorship program — and how to get started. [updated 2025]. Available from: https://www.aaha.org/newstat/publications/why-your-practice-needs-a-mentorship-program/. Last accessed January 30, 2025.
2. Moody C. Mentoring matters — mentorship in veterinary practice. Veterinary Practice, January 2024. Available from: https://www.veterinary-practice.com/article/mentoring-mentorship-veterinary-practice. Last accessed January 30, 2025.
3. Boatright K. The Evolution of Mentorship for Veterinary Professionals. Today's Veterinary Practice, June 15, 2022. Available from: https://todaysveterinarypractice.com/personal-professional-development/the-evolution-of-mentorship-for-veterinary-professionals/. Last accessed January 30, 2025.
4. Canadian Veterinary Medical Association [Internet]. Mentoring Resources. Available from: https://www.canadianveterinarians.net/veterinary-resources/cvma-mentoring-program/mentoring-resources/. Last accessed January 30, 2025.
5. Canadian Veterinary Medical Association [Internet]. CVMA Mentorship Pilot Program. Available from: https://www.canadianveterinarians.net/about-cvma/latest-news/cvma-mentorship-pilot-program/. Last accessed February 5, 2025.

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rétention des professionnels déjà en poste. Le mentorat est typiquement bénéfique pour les personnes qui ont récemment obtenu leur diplôme, celles qui reprennent la pratique après une longue période d'absence, et celles qui réorientent leur carrière (qui changent de domaine de pratique ou qui aspirent à un rôle de gestionnaire, par exemple) (2).

Un programme de mentorat fructueux comporte trois éléments clés : des attentes claires, un plan d'action et une bonne communication (1). Autrement dit, le mentor et le mentoré doivent discuter pour bien comprendre les besoins et les possibilités, établir un plan d'action qui décrit les objectifs, l'horizon temporel et les ressources requises, et s'engager à communiquer honnêtement et ouvertement. Il peut être utile de rédiger une entente décrivant le programme de mentorat, de planifier des réunions périodiques et de tenir ces réunions à l'extérieur de la clinique afin de réduire au minimum les interruptions (1). La confidentialité, des limites bien définies et une rétroaction régulière (dans les deux sens) sont d'autres éléments très importants (2).

Le mentor et le mentoré doivent tous deux contribuer au succès du mentorat, et devraient s'attendre tous deux à

en tirer des avantages. Être un bon mentor peut procurer un sentiment de contribution et de satisfaction (2), sans compter que le mentor acquiert presque inévitablement de nouvelles connaissances dans le processus.

Même si le mentor donne souvent des conseils, dans certains cas, il peut agir comme un coach, discuter de diverses situations avec le mentoré et l'accompagner dans sa réflexion visant à trouver des solutions. Certains estiment que le fait de permettre à une personne d'identifier et de résoudre ses propres problèmes rend la résolution plus probable (2).

Dans sa forme classique, le mentorat en médecine vétérinaire est axé sur les compétences cliniques, en particulier pour les nouveaux diplômés qui ont beaucoup de connaissances mais manquent d'expérience pratique (3). Toutefois, un bon mentorat couvre également la communication, le leadership et le bien-être personnel – des aspects de la profession dans lesquels de nombreux vétérinaires en début de carrière manquent d'assurance (3). Un médecin vétérinaire ayant une longue expérience d'accompagnement de collègues débutants a d'ailleurs déclaré que 90 % des sujets abordés avec ses mentorés n'étaient pas de nature clinique mais avaient tout de même un impact important sur leur vie professionnelle (2).

Plusieurs vétérinaires d'une même clinique peuvent jouer le rôle de mentor auprès d'un même mentoré, et un mentoré peut avoir plus d'un mentor. Un tel « mentorat mosaïque » peut être très utile, car les connaissances, compétences et approches varient d'un mentor à l'autre. Par exemple, un mentor peut se concentrer sur la chirurgie, un autre sur la médecine interne, et un troisième sur la communication et les interactions avec les clients. Bien que le fait d'avoir un collègue de la même pratique comme mentor soit très courant, un mentor peut aussi travailler dans une autre clinique ou le mentorat peut s'effectuer à distance (différents types de services de mentorat virtuel sont disponibles). Nous vous invitons à consulter le site Web de l'Association canadienne des médecins vétérinaires (ACMV), qui comporte une section sur le mentorat avec plus de 15 documents à télécharger ainsi que des liens vers deux ressources en ligne (4). De plus, l'ACMV a récemment lancé la version pilote d'un nouveau programme de mentorat (5).

Compte tenu de la complexité de notre profession, des exigences et des attentes des clients, et des défis liés à l'embauche et à la rétention du personnel, le mentorat n'a jamais été aussi important. La capacité d'offrir un mentorat de qualité est un élément clé de la gestion réussie d'une pratique vétérinaire. Cet éditorial est axé sur le mentorat des médecins vétérinaires, mais le mentorat des techniciens et techniciennes en santé animale (TSA) et d'autres membres du personnel des cliniques vétérinaires présente des avantages indéniables. N'hésitez pas à profiter des nombreuses ressources disponibles.

RÉFÉRENCES

1. Trends [Internet]. Why your practice needs a mentorship program — and how to get started. [Site mis à jour en 2025.] En ligne : https://www.aaha.org/newstat/publications/why-your-practice-needs-a-mentorship-program/ (dernière consultation le 30 janvier 2025).
2. Moody C. Mentoring matters — mentorship in veterinary practice. Veterinary Practice, janvier 2024. En ligne : https://www.veterinary-practice.com/article/mentoring-mentorship-veterinary-practice (dernière consultation le 30 janvier 2025).
3. Boatright K. The Evolution of Mentorship for Veterinary Professionals. Today's Veterinary Practice, 15 juin 2022. En ligne : https://todaysveterinarypractice.com/personal-professional-development/the-evolution-of-mentorship-for-veterinary-professionals/ (dernière consultation le 30 janvier 2025).
4. Association canadienne des médecins vétérinaires [Internet]. Mentoring Resources. En ligne : https://www.veterinairesaucanada.net/ressources-pour-les-medecins-veterinaires/programme-de-mentorat-de-l-acmv/ressources-mentorat/ (dernière consultation le 30 janvier 2025).
5. Association canadienne des médecins vétérinaires [Internet]. Projet pilote de mentorat de l'ACMV. En ligne : https://www.veterinairesaucanada.net/a-propos-de-l-acmv/dernieres-nouvelles/projet-pilote-de-mentorat-de-l-acmv/ (dernière consultation le 5 février 2025).

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VETERINARY MEDICAL ETHICS
DÉONTOLOGIE VÉTÉRINAIRE

ETHICAL QUESTION OF THE MONTH — JANUARY 2025

You are a seasoned veterinarian employed by a companion animal practice. The practice has a reputation of providing excellent care at a reasonable cost. The practice owner is seeking to retire and sells the business to a veterinary practice corporation. Assurances are made to both the practice owner and staff that the practice policies that have contributed to the success of the business will not change with the change in ownership. However, a year later you begin receiving directives from management requiring you to recommend and promote routine laboratory and other wellness testing. This makes you uncomfortable for 2 reasons — you do not wish to "upsell" to clients, especially as some are very vulnerable to pressure due to their attachment to their animal. You are also aware that some sick animals are waiting too long for appointments, so feel the practice should stay focused on that as a priority instead of optional wellness testing. **How do you navigate this disagreement in practice focus and priorities? Given the much-reduced competition between practices since the corporatization trend, how do we protect the public's access to affordable veterinary care?**

QUESTION DE DÉONTOLOGIE DU MOIS – JANVIER 2025

Vous êtes vétérinaire depuis longtemps et vous travaillez dans une clinique pour animaux de compagnie. Cette clinique a la réputation de fournir d'excellents soins à un coût raisonnable. Le propriétaire souhaite prendre sa retraite et vend la clinique à un conglomérat vétérinaire. L'acheteur garantit à votre patron et au personnel que les politiques qui ont contribué au succès de la clinique ne seront pas modifiées malgré le changement de propriétaire. Or, un an plus tard, vous commencez à recevoir des directives de la direction vous demandant de recommander et de promouvoir des analyses de laboratoire de routine et d'autres tests de bien-être. Cela vous met mal à l'aise pour deux raisons : premièrement, vous ne souhaitez pas faire de la « vente incitative » aux clients, d'autant plus que certains sont très vulnérables à la pression en raison de leur attachement à leur animal, et deuxièmement, vous savez que les délais d'attente pour les rendez-vous sont trop longs pour certains animaux malades et vous estimez que la pratique devrait se concentrer sur cet enjeu plutôt que sur des tests de santé facultatifs. **Comment résoudre ce désaccord sur l'orientation et les priorités de la clinique? Compte tenu de la concurrence fortement réduite entre les établissements vétérinaires depuis la tendance à l'acquisition par des conglomérats et la consolidation, comment protéger l'accès du public à des soins vétérinaires abordables?**

ETHICISTS' COMMENTARY ON INCREASING CORPORATIZATION IN VETERINARY PRACTICES

One major development in companion animal veterinary practice in Canada (as elsewhere) is increasing corporatization. A 2023 study reported that 20% of small animal clinics, and more than 50% of specialty and emergency clinics in Canada are owned by corporations.

All veterinary clinics are businesses, and shifting ownership does not alter this basic feature. However, corporate ownership typically brings significant changes. Some of these are plausibly advantageous to everyone: corporate size, for instance, allows economies of scale in procurement and administration. However, one significant change is that the direction of the business is no longer defined by a veterinarian working directly with the clients and their animals. Although this may be a relief to some veterinarians,

this raises the potential for a loss of veterinary autonomy, and (as in the case here) concern about working for a business that's primarily geared towards the profit of corporate investors.

The underlying worry behind this month's question is that the interests of the corporate veterinary clinic are out of line with the interests of its clients and patients. More specifically, management is directing veterinarians to promote "wellness" assessments that are not strictly required for the animals in question, and that may lead to financial manipulation of vulnerable clients and to wasting practice time.

The specifics can, of course, be debated. It might be that "wellness" treatments serve as a form of preventative medicine that can ultimately reduce the number of sick patients taking up practice time. And when it comes to the need for diagnostic testing and imaging there might be different views among veterinarians. Some may prefer to have more data before developing a treatment plan, and their clients may be happy to pay for this.

However, there is a genuine risk that corporate and client interests will not be aligned. For example, if corporate veterinary practices take over most of the market, they will have the power to set prices, limiting the ability of clients to seek lower prices elsewhere. The veterinarian, who has obligations both to the corporation and the client, is caught in the middle.

In Canada (as in almost everywhere else) the public has no established right to "affordable veterinary care." Pricing for goods and services is established in the marketplace, and the letter writer is likely correct in assuming that further consolidation is likely to financially benefit corporate investors more than individual clients.

The best way to counter the problems identified here must be an ongoing effort within the veterinary profession to empower front-line veterinarians to focus on the well-being of their clients and patients, even when this might be at odds with the short-term business interests of their employer. As many of the concerns raised are not unique to corporate practice, that employer could own several clinics or perhaps just a single clinic. Given the professional power imbalance, and the vulnerability of clients making decisions for their companions, we especially encourage front-line veterinarians to always make their clients aware of all the available options, how much they are likely to benefit the patient, and how much they will cost.

Drs. Clare Palmer, Peter Sandøe, and Dan Weary

Response from Co-Editors

The Co-Editors of The Canadian Veterinary Journal remind the readers that articles and features published in the journal reflect the opinions of the authors and do not necessarily reflect the opinion of the publisher. As the publisher, the CVMA respects the independence and editorial policies of the journal. We are very proud to oversee the journal and appreciate the various opinions expressed.

ETHICAL QUESTION OF THE MONTH — APRIL 2025

You are presented with a 5-year-old Labrador retriever by owners who are requesting euthanasia because the dog has bitten their granddaughter twice without warning. The owners are not clients of your clinic and made the appointment because their regular veterinarian and a veterinarian at an emergency clinic refused to euthanize, telling them that they do not believe in "convenience euthanasia" for behavioral reasons. They seem very distraught at their decision and tell you that they feel considerable guilt. You examine the dog who appears to be in excellent physical health and is a typical friendly Labrador with no signs of aggression. You have been told by more recent graduate veterinarians that the term "convenience euthanasia" has fallen out of favor: it is too judgmental, and requests for euthanasia solely for

QUESTION DE DÉONTOLOGIE DU MOIS – AVRIL 2025

Vous recevez en consultation les propriétaires d'un labrador de 5 ans qui vous demandent de l'euthanasier parce qu'il a mordu leur petite-fille à deux reprises sans avertissement. Ces gens ne sont pas des clients de votre clinique et ont pris rendez-vous parce que leur vétérinaire habituel et un vétérinaire d'un centre d'urgence ont refusé d'euthanasier leur chien, car ils n'acceptent pas de faire des « euthanasies de convenance » pour des raisons comportementales. Les propriétaires semblent profondément bouleversés par ces refus et vous disent qu'ils se sentent terriblement coupables. Vous examinez le chien qui a l'air d'être en excellente santé physique et qui est un labrador amical typique ne démontrant aucun signe d'agressivité. Des collègues vétérinaires ayant obtenu leur diplôme récemment vous ont dit que

the benefit of the owner are no longer made. These veterinarians have been advised to be more empathetic towards the owners requesting euthanasia for non-medical reasons, and by withholding judgment and asking more questions, they inevitably learn that the owner's reasons are valid, and it is not for owner convenience at all. **Do you proceed with euthanasia?**

Submitted by Cecily Grant, B.Sc. (Hons), D.V.M

l'expression « euthanasie de convenance » n'était plus utilisée, parce qu'elle est trop réprobatrice mais aussi parce que les vétérinaires ne se font plus demander de faire des euthanasies dans le seul intérêt des propriétaires. On aurait recommandé à ces jeunes vétérinaires d'être plus empathiques à l'égard des clients qui demandent l'euthanasie pour des raisons non médicales, car en ne portant pas de jugement et en posant plus de questions, ils finiraient inévitablement par constater que les raisons des clients sont valables et que leur décision n'est pas du tout motivée par la convenance. **Procédez-vous à l'euthanasie?**

Question soumise par Cecily Grant, B. Sc. (Hons), D.M.V.

Responses to the case presented are welcome. Please limit your reply to approximately 50 words and forward along with your name and address to: **Ethical Choices, c/o Canadian Veterinary Medical Association, Attn: Journals Department, 339 Booth Street, Ottawa, Ontario K1R 7K1; email (bettinadvm@gmail.com). A longer response may appear as a Letter to the Editor.**

Suggested ethical questions of the month are also welcome! All ethical questions or scenarios in the ethics column are based on actual events, which are changed, including names, locations, species, *etc.*, to protect the confidentiality of the parties involved.

Les réponses au cas présenté sont les bienvenues. Veuillez limiter votre réponse à environ 50 mots et nous la faire parvenir avec vos nom et adresse **par la poste (Choix déontologiques, Association canadienne des médecins vétérinaires, À l'attention de : Journals Department, 339 rue Booth, Ottawa, Ontario, K1R 7K1) ou par courriel (bettinadvm@gmail.com). Les réponses plus longues pourraient être publiées dans le courrier des lecteurs.**

Les propositions de questions déontologiques sont toujours bienvenues! Toutes les questions et situations présentées dans cette chronique s'inspirent d'événements réels dont nous modifions certains éléments, comme les noms, les endroits ou les espèces, pour protéger l'anonymat des personnes en cause.

QUIZ CORNER
TEST ÉCLAIR

1. A milk sample is sent into your clinic for culture from a third lactation Jersey cow with a history of decreased appetite, drop in milk production, and temperature of 39.7°C (normal temperature = 38.0 to 39.3°C).

There is bacterial growth on both the blood agar and MacConkey culture plates. Growth on the MacConkey looks like the image in Figure 1.

1. Un échantillon de lait est envoyé à votre clinique aux fins de culture. Il provient d'une vache jersey en troisième lactation qui présente une diminution de l'appétit, une baisse de la production de lait et une température de 39,7 °C (alors que la température normale est entre 38,0 et 39,3 °C).

Une croissance bactérienne est observée sur la gélose au sang et sur la gélose de MacConkey. La croissance sur la gélose de MacConkey ressemble à l'image de la figure 1.

FIGURE 1
Microbial growth on a MacConkey plate. (Image courtesy of Liz Brock, DVM, MS).
Croissance microbienne sur une gélose de MacConkey. (Image de Liz Brock, D.M.V., M. Sc.)

What is the best treatment recommendation for this cow?

A. Intramammary pirlimycin
B. Supportive therapy
C. Iodine teat dip
D. Topical insecticides
E. Vaccinate with bacterin

Quelle est la meilleure recommandation de traitement pour cette vache?

A. Pirlimycine intramammaire
B. Traitement de soutien
C. Bain de trayons à base d'iode
D. Insecticides topiques
E. Vaccination au moyen d'une bactérine

2. A 4-month-old Great Dane dog exhibits an acute onset of distal forelimb swelling and lameness.

The puppy is febrile and lethargic. The distal metaphyseal region of the femur and tibia of both limbs feel very warm. Radiographs are shown in Figure 1.

2. Un grand danois de 4 mois est examiné en raison de l'apparition soudaine d'une enflure de la partie distale des membres antérieurs et d'une boiterie.

Le chiot est fiévreux et léthargique. La région métaphysaire distale du fémur et du tibia des deux pattes est chaude au toucher. Les radiographies sont présentées à la figure 1.

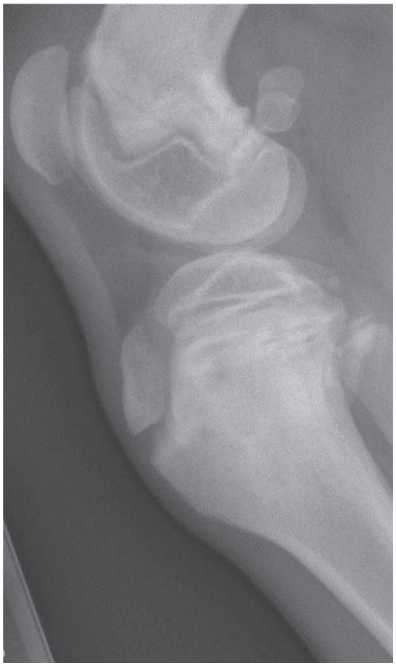

FIGURE 1
Lateral radiograph of the stifle.
(Image courtesy of Clara Moran, DVM, MS, DACVS-LA).
Radiographie latérale du genou.
(Image de Clara Moran, D.M.V., M. Sc., DACVS LA.)

Which one of the following choices is the most likely diagnosis?

A. Osteochondritis dissecans
B. Panosteitis
C. Osteosarcoma
D. Hypertrophic osteodystrophy
E. Amyotrophic lateral sclerosis

Quel est le diagnostic le plus probable?

A. Ostéochondrite disséquante
B. Panostéite
C. Ostéosarcome
D. Ostéodystrophie hypertrophique
E. Sclérose latérale amyotrophique

(See p. 456 for answers./Voir les réponses à la page 456.)

The questions and answers are provided by Zuku Review, online veterinary test prep.

Les questions et les réponses sont gracieusement fournies par le site de préparation aux examens vétérinaires Zuku Review.

CASE REPORT
RAPPORT DE CAS

Medical management of traumatic hepatic parenchymal emphysema in a dog

Adrien Privat, Céline Giron, Jo-Annie Letendre

ABSTRACT

A 4-year-old intact female dachshund dog was presented in shock after a traumatic event. The dog had sustained hemoperitoneum and pulmonary contusions. Veterinary point-of-care ultrasound revealed reverberation artifacts inside the liver. Abdominal ultrasonography and computed tomography identified these as lesions of hepatic emphysema. Biochemical analysis showed marked elevation of liver enzymes. Treatments included S-adenosylmethionine and broad-spectrum antibiotics, among others. Two weeks following trauma, alanine aminotransferase was near normal and there was almost complete resolution of the hepatic lesions on abdominal ultrasonography. This case report is the first to describe the medical management and time course of parenchymal hepatic emphysema in a dog following trauma. It also focuses on the diagnosis of hepatic emphysema using various imaging modalities.

Key clinical message:

Hepatic parenchyma emphysema can develop following blunt abdominal trauma and appears as bright, hyperechoic foci with reverberation artifacts that can be easily observed with veterinary point-of-care ultrasonography or complete abdominal ultrasonography. Hepatic emphysema does not always require surgical treatment. Monitoring biochemical abnormalities and lesions with various imaging modalities, along with providing supportive treatments, can sometimes be sufficient.

RÉSUMÉ

Prise en charge médicale d'un emphysème hépatique parenchymateux traumatique chez un chien

Une chienne teckel intacte de 4 ans a été présentée en état de choc après un événement traumatique. La chienne avait subi un hémopéritoine et des contusions pulmonaires. L'échographie au point de service vétérinaire a révélé des artefacts de réverbération à l'intérieur du foie. L'échographie abdominale et la tomodensitométrie ont identifié ces lésions comme étant celles d'un emphysème hépatique. L'analyse biochimique a montré une élévation marquée des enzymes hépatiques. Les traitements comprenaient notamment la S-adénosylméthionine et des antibiotiques à large spectre. Deux semaines après le traumatisme, l'alanine aminotransférase était proche de la normale et les lésions hépatiques avaient presque complètement disparu à l'échographie abdominale. Ce rapport de cas est le premier à décrire la prise en charge médicale et l'évolution temporelle de l'emphysème hépatique parenchymateux chez un chien après un traumatisme. Il se concentre également sur le diagnostic de l'emphysème hépatique à l'aide de diverses modalités d'imagerie.

Message clinique clé :

L'emphysème du parenchyme hépatique peut se développer à la suite d'un traumatisme abdominal contondant et se présente sous la forme de foyers hyperéchogènes clairs avec des artefacts de

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réverbération qui peuvent être facilement observés par échographie au point de service vétérinaire ou par échographie abdominale complète. L'emphysème hépatique ne nécessite pas toujours de traitement chirurgical. La surveillance des anomalies biochimiques et des lésions à l'aide de diverses modalités d'imagerie, ainsi que la mise en place de traitements de soutien, peuvent parfois être suffisantes.

(Traduit par Dr Serge Messier)

Can Vet J 2025;66:370–377

Hepatic emphysema is characterized by the presence of gas in the liver, either in the parenchyma, the portal venous system, or the biliary tract. In humans, causes of hepatic emphysema include infectious conditions, gastrointestinal tract diseases, other abdominal conditions, and various iatrogenic causes (1–4). In veterinary medicine, the condition has been reported in dogs and cats with hepatic abscess, emphysematous or suppurative hepatitis, liver lobe torsion, hepatic neoplasia, biliary tract infection, or gastrointestinal ulceration; and after oral administration of hydrogen peroxide, biliary tract or gastrointestinal surgery, or liver biopsy (5–10).

Historically, the identification of hepatic gas was thought to be a serious finding associated with a mortality rate as high as 75% (4). Because of the increased use of computed tomography (CT) and ultrasonography, hepatic emphysema is now more frequently detected and does not always appear to be associated with life-threatening conditions (11). In addition, the increasing use of imaging modalities has also challenged the belief that hepatic emphysema is always a sign of infection. When associated with a traumatic event, hepatic gas is suspected to be secondary to an acute change in intra-abdominal pressure that forces intraluminal gas into the bowel wall through mucosal injury. Hepatic emphysema can also be secondary to severe hepatic ischemia, hemorrhage, or sterile necrosis associated with liver injuries (3,6,12–14).

Because hepatic emphysema is infrequently encountered, selecting the optimal treatment, whether medical or surgical, can be difficult for veterinarians. This case report highlights the temporal progression of hepatic emphysema following trauma in a dog, providing valuable insights into the management of this uncommon condition in veterinary medicine. It also emphasizes how different imaging modalities can be useful in diagnosing and monitoring this type of lesion.

CASE DESCRIPTION

A 4-year-old intact female standard wire-haired dachshund was brought to a veterinary emergency service ~1 h after a road traffic accident. The dog was in excellent health before the accident, with no previous medical history. On initial examination (Day 1), the dog was in shock with depressed mentation, a temperature too low to read [< 32.0°C (< 89.6°F)], a heart rate of 80 bpm, pale mucous membranes with a capillary refill time of 2 s, and decreased femoral pulses. Her respiratory rate and effort were within normal limits. An electrocardiogram showed normal sinus rhythm. Oxygen saturation was 98% on room air. Blood pressure was initially unmeasurable with Doppler (Parks Medical, Aloha, Oegon, USA). Packed cell volume (PCV) was normal [52%; reference interval (RI): 30 to 55%] with a mild decrease in total plasma protein concentration [52 g/L; RI: 55 to 75 g/L (5.5 to 7.5 g/dL)]. Blood glucose was elevated [10.6 mmol/L (190.8 mg/dL); RI: 3.6 to 8.6 mmol/L (60 to 135 mg/dL)]. Veterinary point-of-care ultrasound (VPOCUS) imaging of the thorax and abdomen was obtained with a Sonosite Edge II (Fujifilm Sonosite, Bothell, Washington, USA) and a microconvex probe (C11x, 8-5.0 MHz ultrasound probe, Microconvex Model C11x; Fujifilm Sonosite). The B-lines observed bilaterally were interpreted as pulmonary contusions. A small volume of hypoechoic peritoneal effusion was observed. Hemoperitoneum was confirmed following abdominocentesis of hemorrhagic fluid with a PCV of 50%. Three-view thoracic radiographs were later obtained and showed a moderate multifocal bilateral and asymmetrical mixed pulmonary infiltrate characterized by a multifocal ill-defined unstructured interstitial lung pattern and moderate ill-defined cranial alveolar opacities (Figure 1). Other findings included a mild pleural effusion and a suspicion of focal cranial mediastinal effusion with discrete regional pneumomediastinum (Figure 1 A). A CBC and biochemical analysis revealed normal hematocrit (0.43 L/L; RI: 0.40 to 0.56 L/L), elevated nucleated red blood cell count [6 per 100 white blood cells (WBC); RI: 0/100 WBC], normal WBC count (11.74 × 10^9/L; RI: 5.10 to 14.20 × 10^9/L), normal platelet count (250 × 10^9/L; RI: 153 to 400 × 10^9/L), elevated phosphorus [1.86 mmol/L (5.76 mg/dL); RI: 0.79 to 1.51 mmol/L (2.45 to 4.68 mg/dL)]; increased

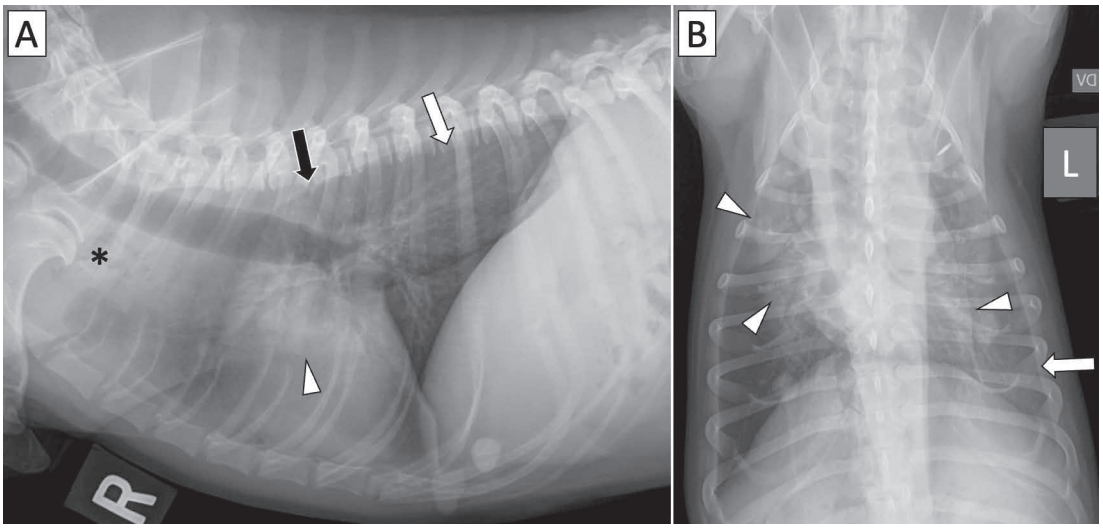

FIGURE 1. Right lateral (A) and dorsoventral (B) thoracic radiographs of a 4-year-old intact female standard wire-haired dachshund. Radiographs were obtained at admission following a road traffic accident. The mixed multifocal bilateral and asymmetrical pulmonary opacity characterized by an ill-defined unstructured interstitial lung pattern (white arrows) and moderate, ill-defined bilateral alveolar opacities (white arrowheads) were interpreted as pulmonary contusions or hemorrhage. Very mild pleural fissure lines were compatible with a minimal pleural effusion (black arrow). In the cranial mediastinum and ventral to the thoracic trachea, there was a heterogeneous area with a soft-tissue opacity accumulation and several small gas bubbles, representing most probably a focal mediastinal effusion and pneumomediastinum (black asterisk).

alanine aminotransferase (ALT) activity (1418 U/L; RI: 21 to 80 U/L) and gamma-glutamyltransferase (GGT) activity (10 U/L; RI: 1 to 8 U/L) suggestive of hepatic injury; and decreased albumin (24.4 g/L; RI: 26.7 to 35.4 g/L) and globulins (16.2 g/L; RI: 21.7 to 32.9 g/L), most likely secondary to hemoperitoneum.

Immediately upon admission, a cephalic IV catheter was placed, IV fluid boluses (Plasmalyte-A; Baxter, Mississauga, Ontario) were administered, and the dog was actively rewarmed. Mental state and vital signs improved. The dog was hospitalized, and treatments included tranexamic acid (Omega Laboratories, Montreal, Quebec), 10 mg/kg BW, IV, q8h); remifentanil (SteriMax, Oakville, Ontario), 5 μg/kg per hour continuous rate infusion (CRI); and IV fluid therapy. During the first night, the dog developed persistent ventricular tachycardia and received lidocaine (Lurocaine; Vetoquinol Canada, Lavaltrie, Quebec), 2 mg/kg BW, IV bolus; followed by a 25 μg/kg per minute CRI); which resolved the tachycardia, allowing discontinuation of antiarrhythmic therapy within 48 h.

Despite progressive anemia over the first few days of hospitalization (PCV: 30%, RI: 30 to 55%; total plasma protein: 40 g/L, RI: 55 to 75 g/L), the dog remained hemodynamically stable and did not require blood transfusion. Remifentanil CRI was replaced by methadone (Comfortan; Dechra Veterinary Products, Eurovet Animal Health, Bladel, Netherlands), 0.1 mg/kg BW, IV, q6h; and gabapentin (Summit Veterinary Pharmacy, Aurora, Ontario), 7 mg/kg BW, PO, q12h. Two days following admission (Day 3), a recheck VPOCUS showed a focal hypoechoic ill-defined hepatic nodule with reverberation artifacts and an improvement of the diffuse peritoneal effusion. Admission thoracic radiographs were retrospectively reviewed and no gas opacity in the visible portion of the liver silhouette was noted (Figure 1). Recheck biochemical analysis revealed a marked worsening of ALT (2569 U/L; RI: 21 to 80 U/L); slight GGT increase (12 U/L; RI: 1 to 8 U/L); elevated alkaline phosphatase (ALP) activity (328 U/L; RI: 10 to 113 U/L); and resolution of the hyperphosphatemia [1.15 mmol/L (3.56 mg/dL); RI: 0.79 to 1.51 mmol/L (2.45 to 4.68 mg/dL)], hypoalbuminemia (29.1 g/L; RI: 26.7 to 35.4 g/L), and hypoglobulinemia (28.2 g/L; RI: 21.7 to 32.9 g/L). The WBC count and differential remained within the reference range, except for a mild monocytosis (0.98×10^9/L; RI: 0.10 to 0.90×10^9/L). Concerns for severe hepatic injury such as necrosis or infection motivated the prescription of S-adenosylmethionine (Zentonil Advanced; Vetoquinol Canada), 14 mg/kg BW, PO, q24h; metronidazole (Baxter), 10 mg/kg BW, IV, q12h; and ampicillin (TEVA Canada, Toronto, Ontario), 22 mg/kg BW, IV, q8h.

A complete abdominal ultrasound examination was completed by a Board-certified radiologist on the following day (Day 4). In the left liver, there were 2 peripheral

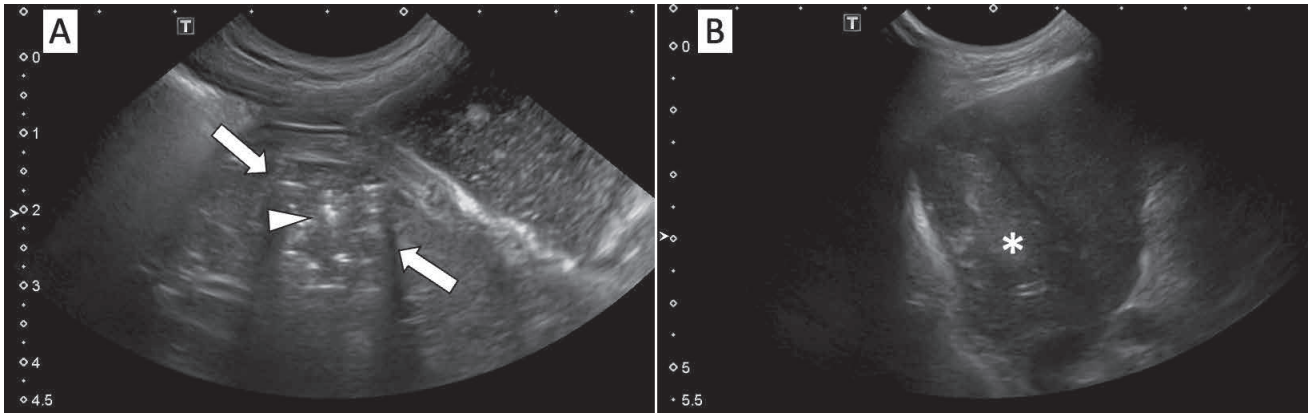

FIGURE 2. Ultrasound examination of a 4-year-old intact female standard wire-haired dachshund on Day 4 after a road traffic accident. A — In the left ventral hepatic parenchyma, a hypoechoic, heterogeneous, well-defined nodule (between white arrows) was present, with several hyperechoic foci inducing reverberation artifacts (white arrowhead), suggesting focal hepatic parenchymal emphysema. B — In the cranial hepatic parenchyma, there was an ill-defined, ovoid, hyperechoic hepatic nodule (white asterisk). This was nonspecific and could have represented a benign process (hyperplasia, extramedullary hematopoiesis, adenoma) or parenchymal hematoma/hemorrhage.

hepatic heterogeneous nodules slightly hypoechoic to the hepatic parenchyma, with well-defined margins. The largest (21 × 17 mm) contained several hyperechoic foci inducing reverberation artifacts (Figure 2 A). Other multifocal, ill-defined homogeneous hyperechoic hepatic nodules measuring a maximum of 21 × 10 mm were also present (Figure 2 B). The previous peritoneal effusion had mainly disappeared, but peritoneal fat was diffusely slightly hyperechoic, indicating residual peritonitis or edema secondary to the previous hemoperitoneum. In addition, an incidental small (2 mm) nephrolith was present in the left renal pelvis.

Three-view abdominal radiographs were obtained immediately after the abdominal ultrasound was completed. A well-defined gas cavity within the median part of the left liver was now present, measuring 16 × 20 × 16 mm (height × width × length) (Figure 3). Other minimal and more discrete gaseous cavities were present caudoventrally to this lesion. Given our main suspicion of a noninfectious process with the absence of medical differential diagnoses, the history of trauma, and the absence of specific abnormalities on the CBC, and after consideration of the associated risks, it was decided not to aspirate the gas-containing liver nodule.

To better characterize the hepatic lesions and make therapeutic decisions, an abdominal CT scan was undertaken on Day 5. The dog was sedated with butorphanol (Dolorex; Intervet, Kirkland, Quebec), 0.2 mg/kg BW, IV; and dexmedetomidine (Dexdomitor; Zoetis, Kirkland, Quebec), 1 μg/kg BW, IV bolus, followed by 1 μg/kg per hour CRI, and positioned in sternal recumbency. The CT evaluation included non-enhanced and contrast-enhanced dynamic CT with a 16-detector array helical unit (Lightspeed 16; General Electric Healthcare Medical Systems, Mississauga, Ontario). Images were acquired with 1.25-millimeter slice thickness in helical mode, using a pitch of 0.938, at 120 kVp and 200 mAs. Image acquisitions included a pre-contrast and a venous phase 2 min following a manual injection of iodinated contrast medium (Isovue 300; ER Squibb & Sons, Princeton, New Jersey, USA), 2 mL/kg.

On the left caudolateral part of the liver, 2 homogeneous liquid-filled cavitary masses measuring 24 × 22 × 20 mm and 17 × 12 × 3 mm (length × width × height) were present (Figure 4 A, B). The larger and more ventral lesion contained a large gas bubble (16 × 21 × 6 mm) and other smaller ones. At least 3 intrahepatic post-contrast heterogeneously enhanced and ill-defined lesions were noted (Figure 4 C). A subtle gas bubble was observed cranial to the cranial pole of the right kidney. Diffusely, mesenteric fat was enhanced and slightly heterogeneous. The final CT findings were hepatic parenchymal emphysema within hepatic hemorrhage, necrosis, or parenchymal ischemia, with multifocal hepatic hematoma or hemorrhage, resolution of the previously observed peritoneal effusion, persistent slight mesenteric edema, and a minimal retropneumoperitoneum. The hepatic emphysema seemed to represent necrosis or an ischemic hepatic parenchymal lesion secondary to blunt trauma. An infectious process such as a hepatic abscess caused by peritoneal/digestive bacterial translocation was considered less likely, even if it could not be excluded. Finally, a hepatic neoplastic infiltration was considered

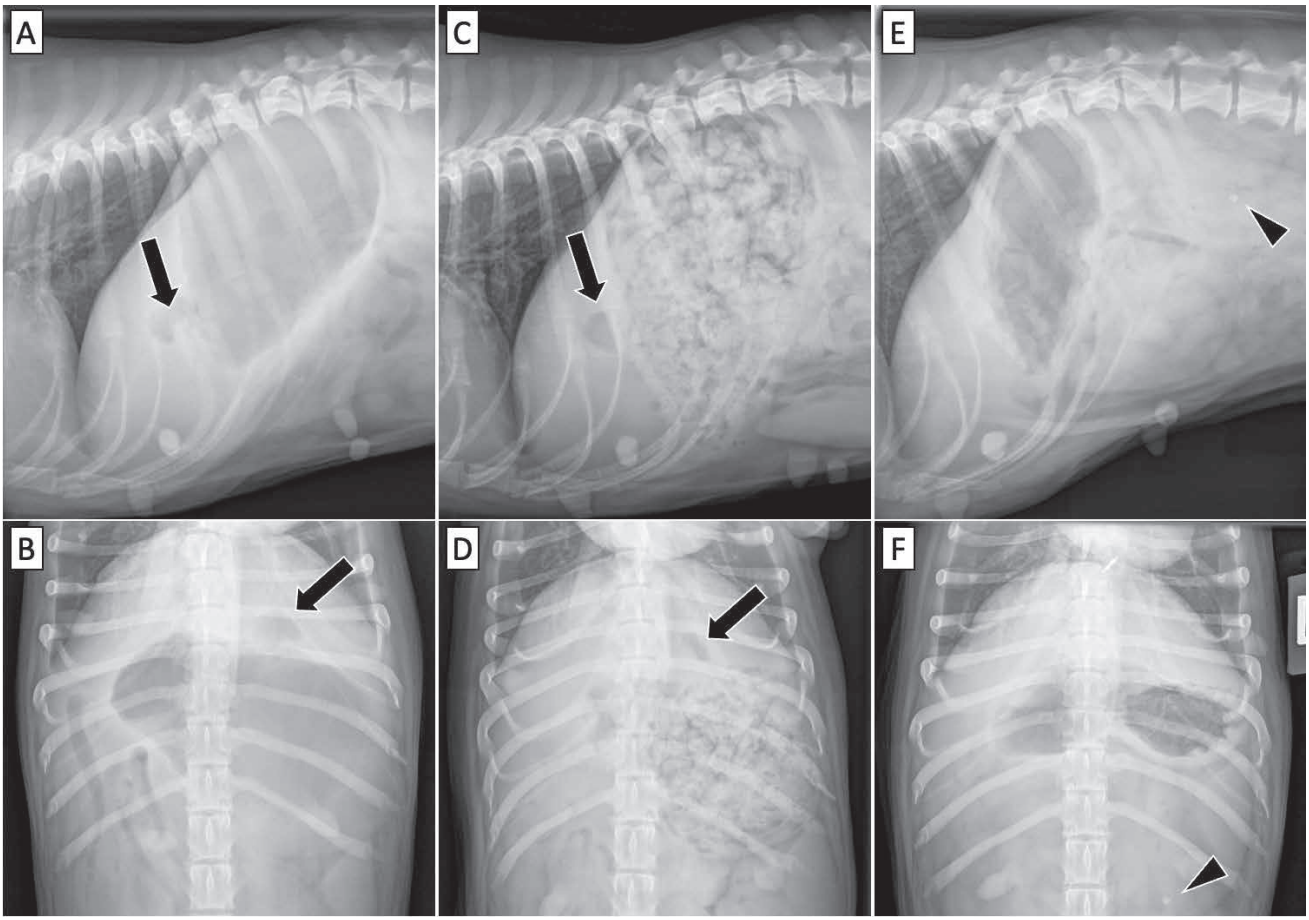

FIGURE 3. A to F — Follow-up abdominal radiographs with right lateral (A, C, E) and dorsoventral (B, D, F) views of a 4-year-old intact female standard wire-haired dachshund on Day 4 (A and B), Day 6 (C and D), and Day 13 (E and F) after a road traffic accident. A and B — At Day 4, a focal, moderate, gas-filled cavitary lesion was present in the left portion of the hepatic silhouette (black arrow), measuring a maximum of 16 × 20 × 16 mm (height × width × length). This lesion was not present on Day 1 (Figure 1). C and D — A mild size reduction of the gaseous cavitary lesions was visible on Day 6 (black arrow), measuring 11 × 15 × 8 mm. E and F — At Day 13, the gaseous cavitary lesion was not visible. The mentioned small left nephrolithiasis was visible (black arrowhead).

improbable given the dog's good health before the trauma. Based on these findings and the stable state of the animal, medical treatment was chosen over surgery.

On Day 6, the dog was doing clinically well, was comfortable, and had a good appetite. Follow-up biochemical analysis showed improvements in the ALT (1059 U/L; RI: 21 to 80 U/L), ALP (279 U/L; RI: 10 to 113 U/L), and GGT (10 U/L; RI: 1 to 8 U/L) elevations. On Day 7, the dog was discharged and prescribed gabapentin, 7 mg/kg BW, PO; q12h; Clavaseptin (Vetoquinol Canada), 14 mg/kg BW, PO, q12h; and Zentonil Advanced (Vetoquinol Canada), 14 mg/kg BW, PO, q24h. Before discharge, 3-view abdominal radiographs were taken and showed persistence of the liver cavity consistent with hepatic emphysema, with a mild decrease in size (Figure 3).

On Day 13, the dog was brought in for a follow-up appointment. She had been doing well at home. Recheck biochemical analysis showed improvements in the ALT (168 U/L; RI: 21 to 80 U/L) and ALP (150 U/L; RI: 10 to 113 U/L) elevations. Three-view abdominal radiographs showed near-resolution of the hepatic gas cavity (Figure 3). Only a discrete, ill-defined gas bubble 2 mm in diameter was observed. On abdominal ultrasound examination, a single, slightly heterogeneous hypoechoic lesion consistent with almost complete resolution of the previous cavitary lesions was visualized in the hilar portion of the left liver and now measured ~15 mm in diameter. The surrounding left liver parenchyma was slightly more echogenic and heterogeneous than the rest of the liver, which was normal in appearance. Treatment with Zentonil Advanced (Vetoquinol Canada) was continued for another 2 wk. Gabapentin was discontinued at the time of the recheck; Clavaseptin (Vetoquinol Canada) was discontinued after a total of 10 d of treatment.

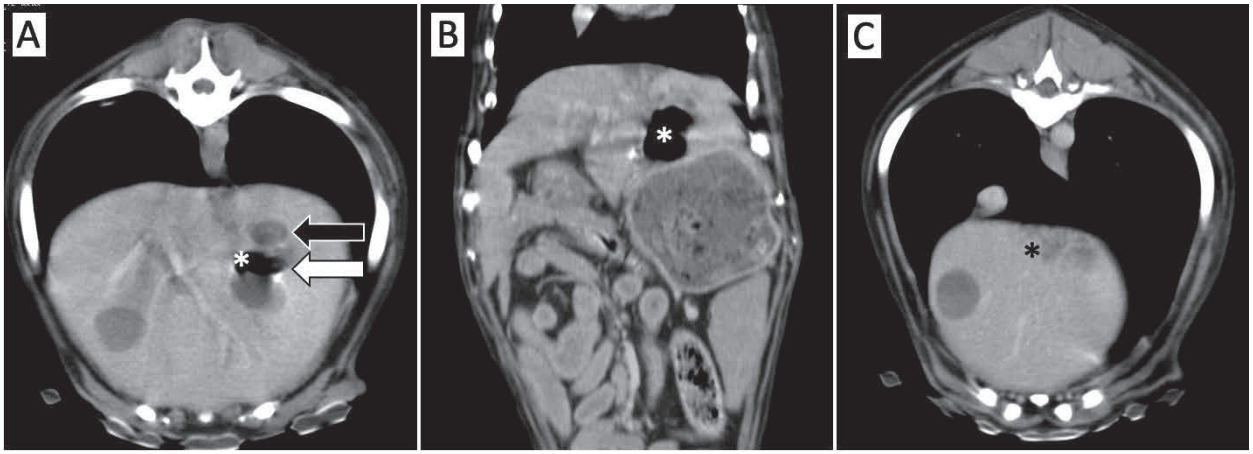

FIGURE 4. A to C — Contrast-enhanced computed tomographic imaging of the abdomen of a 4-year-old intact female standard wire-haired dachshund on Day 5 after a road traffic accident, with transverse (A and C) and sagittal (B) images. A and B — In the left cranial region of the liver, a hypoattenuating cavitary nodule containing fluid and a gaseous bubble (white asterisk) measuring 24 × 22 × 20 mm (length × width × height) was present and consistent with a hepatic parenchymal hemorrhagic or necrotic emphysema (white arrow). A second, smaller hypoattenuating fluid-filled cavitary lesion, measuring 17 × 12 × 3 mm, was present dorsally, representing a second hemorrhagic nodule (black arrow). C — In the craniodorsal region of the hepatic parenchyma, there was a peripheral, ill-defined, and heterogeneous contrast-enhancing intrahepatic lesion interpreted as a probable hepatic hematoma or hemorrhage (black asterisk).

Three months post-trauma, the dog was still doing clinically very well at home and appeared normal. Six months after the initial trauma and a few weeks before a planned follow-up checkup, the dog unfortunately died due to an unrelated traumatic event.

DISCUSSION

Hepatic emphysema is reported as a rare condition in veterinary literature, especially following trauma. In their study evaluating the clinical findings and outcomes associated with hepatic emphysema in cats and dogs, Manfredi *et al* reported only 4 dogs and 1 cat in which hepatic emphysema was secondary to trauma (6). In human medicine, when hepatic emphysema is reported in a context of trauma, the gas is most often located in the hepatoportal venous system (13,15–17). Mechanisms leading to gas in the hepatoportal venous system in the context of trauma include bowel wall disruption, ischemia or necrosis, entry of pathogenic gas-producing intestinal bacteria, and increase in intra-abdominal pressure secondary to trauma (15,17–18).

Thorough review of the human literature identified only 15 cases in which hepatic portal venous gas was reported, therefore occurring in < 1% of victims of blunt abdominal trauma (15). Of those 15 human patients, 7 underwent surgery and 8 were treated medically. In only 4 of the 7 patients who underwent surgery, identified intra-abdominal lesions explained the hepatic emphysema and required gastrointestinal resection (13,15–17). This suggests that, in most human cases, the presence of gas in the hepatoportal system following trauma is transient and self-limiting, as shown in the case presented herein.

In our case, hepatic gas was located in the liver parenchyma, within areas of presumptive hemorrhage, necrosis, or ischemia. In humans, traumatic parenchymal hepatic emphysema was described in only 2 cases (12). In both cases, the patients underwent explorative laparotomy that showed only hepatic contusion and necrosis. As mentioned, in the retrospective study by Manfredi *et al,* only 5 cases of hepatic emphysema were secondary to trauma: 4 were secondary to vehicular trauma and 1 to a dog attack. Among the 5 trauma episodes, 1 dog and 1 cat had hepatic parenchymal emphysema. The dog died from massive hemothorax, but the cat survived after complete resolution of the emphysema within 3 d without surgical intervention. In the other 3 cases, hepatic gas was located in the hepatoportal venous system (6).

In the present case, severe liver injury was suspected when biochemical analysis showed a marked increase in liver enzymes, particularly ALT, in parallel with the identification of hepatic emphysema by VPOCUS. Hepatic parenchymal emphysema appears as multiple bright, hyperechoic foci with reverberation artifacts that can be easily observed with VPOCUS. This has not been reported previously. Interestingly, in our case, hepatic emphysema was not identified with VPOCUS until Day 3. It is possible that the ultrasound lesions were missed on the first 2 d; however, it

is also possible that they were not present until then. This hypothesis was supported by the absence of air bubbles in the liver region on the radiographs obtained on Day 1, and the appearance on follow-up radiographs obtained on Day 4. This delay in the development of hepatic emphysema was similar to what was reported on a human case in which radiographs taken shortly after blunt abdominal trauma were normal and hepatic emphysema was only revealed in radiographs taken 2 d post-trauma (12).

In the current case, CT imaging was done to better characterize the extent of liver injuries and to inform further management of our case. Because no signs of peritonitis, pneumoperitoneum, pneumatosis intestinalis, or bowel necrosis were observed, we decided to pursue medical treatment. Indeed, based on the human literature and the absence of surgical indications in imaging studies, a laparotomy did not seem required. A hepatoprotectant in the form of S-adenosylmethionine was prescribed. We also decided to treat the dog with broad-spectrum antibiotics even though there was no compelling supporting evidence of necessity. In the 2 reported cases of traumatic parenchymal hepatic emphysema in humans, bacterial cultures were negative (12). In veterinary cases, hepatic emphysema has been reported with hepatic, hepatobiliary intra-abdominal, or generalized infectious disease processes in which antibiotics are warranted (5,8,10,19).

A case of emphysematous hepatic abscess following vehicular trauma was reported in a dog. Necropsy showed a ruptured hepatic adenoma with necrosis and it was hypothesized that the gas resulted from gas-producing bacteria. However, aerobic culture did not show any microorganisms, and anaerobic culture was not done. It is possible that the adenoma was sterile (5). Unfortunately, aspiration of the gas-filled lesions to obtain samples for culture was not attempted in our case due to our limited suspicion of an infectious origin and the risks involved with the procedure. Because we chose not to obtain cytological samples of the lesions, an infectious component could not be excluded with certainty, influencing our decision to continue antibiotic administration. Unfortunately, the contribution of antibiotics to the improvement of this dog's condition remains unclear. In addition, as the need for antibiotics in cases of liver trauma associated with emphysema is not known, there is no recommendation as to the duration of antibiotic therapy, if such therapy is required.

In 1 of the 2 human cases of traumatic parenchymal hepatic emphysema, gas was still present after 18 d, though in a lesser amount. The time to complete resolution of hepatic emphysema was not mentioned in either case (12). In our case, hepatic emphysema was still present 2 wk after the trauma. The time for resorption and complete resolution of hepatic emphysema likely depends on the size of the lesion. In the study from Manfredi *et al* (6), all 10 cases with parenchymal hepatic emphysema had elevated liver enzymes. However, no follow-up biochemical analyses were reported (6). In our case, ALT continued to rise before showing a gradual return to normal, compatible with progressive improvement of the liver injuries. Unfortunately, since the dog died before follow-up assessment, it is unknown after how many days ALT returned to normal and the gas completely disappeared.

In the study by Manfredi *et al,* overall mortality was 40.5%. Cases with hepatic portal venous gas had better outcomes compared to those with parenchymal emphysema (n = 10, 90% mortality) (6). Conditions identified in terminal cases with parenchymal hepatic emphysema included chronic hepatitis and hepatic neoplasia. One dog with parenchymal emphysema secondary to trauma died following a massive traumatic hemothorax. In addition, in that study, a negative prognosis was correlated with elevated liver enzymes and liver neoplasia, both of which were associated with parenchymal emphysema. The prognosis for hepatic emphysema appears to correlate with the location of the gas. However, it is crucial to also consider the underlying cause of hepatic emphysema — not just its location. Although parenchymal emphysema generally indicates an unfavorable prognosis compared to hepatoportal emphysema, parenchymal hepatic emphysema resulting from trauma might carry a more favorable prognosis, even without surgical intervention, as demonstrated in the present case report.

In conclusion, this case report emphasizes the diagnosis of hepatic emphysema through the use of various imaging modalities. To date, in the case of trauma, the best imaging modality for identifying this type of lesion is not known, but complete abdominal ultrasound and even the use of VPOCUS appear capable of detecting such lesions. The various imaging modalities, along with close monitoring of the animal's clinical and biochemical findings progression, can assist in selecting the best therapeutic approach. In the present case, the dog was treated medically and made a complete recovery without requiring surgery. To our knowledge, this is the first case report of traumatic parenchymal hepatic emphysema to describe the clinical signs, imaging findings, and time course of lesions and serum biochemistry changes in a dog following trauma. CVJ

REFERENCES

1. Fahrner R, Rauchfuss F, Scheuerlein H, Settmacher U. Post-traumatic venous gas in the liver: A case report and review of the current literature. BMC Surg 2018;18:14.
2. Kinoshita H, Shinozaki M, Tanimura H, *et al*. Clinical features and management of hepatic portal venous gas: Four case reports and cumulative review of the literature. Arch Surg 2021;136:1410–1414.
3. Panicek DM, Paquet DJ, Clark KG, Urruti EJ, Brinsko RE. Hepatic parenchymal gas after blunt trauma. Radiology 1986; 159:343–344.
4. Shah PA, Cunningham SC, Morgan TA, Daly BD. Hepatic gas: Widening spectrum of causes detected at CT and US in the interventional era. Radiographics 2021;31:1403–1413.
5. Lord PF, Carb A, Halliwell WH. Emphysematous hepatic abscess associated with trauma, necrotic hepatic nodular hyperplasia and adenoma in a dog: A case history report. Vet Radiol 1982;23:46–49.
6. Manfredi S, Fabbi M, Bonazzi M. Ultrasonographic differentiation between portal venous and parenchymal gas may be important for the prognosis of canine and feline hepatic emphysema: 37 cases. Vet Radiol Ultrasound 2019;60:734–744.
7. Palko C, Durkan S. Medical management of feline emphysematous hepatitis: A case report. Clin Case Rep 2023;11:e7352.
8. Sato AF, Solano M. Radiographic diagnosis: Liver lobe entrapment and associated emphysematous hepatitis. Vet Radiol Ultrasound 1998;39:123–124.
9. Schwarz LA, Penninck DG, Leveille-Webster C. Hepatic abscesses in 13 dogs: A review of the ultrasonographic findings, clinical data, and therapeutic options. Vet Radiol Ultrasound 1998;39:357–365.
10. Westgren F, Hjorth T, Uhlhorn M, Etterlin PE, Ley CJ. Hepatic emphysema associated with ultrasound-guided liver biopsy in a dog. Act Vet Scand 2014;56:25.
11. Dill-Macky MJ. Benign hepatic portal venous gas following blunt abdominal trauma. Australas Radiol 1997;41:166–168.
12. Abramson SJ, Berdon WE, Kaufman RA, Ruzal-Shapiro C. Hepatic parenchymal and subcapsular gas after hepatic laceration caused by blunt abdominal trauma. Am J Roentgenol 1989;153:1031–1032.
13. Furuya Y, Yasuhara H, Ariki K. Hepatic portal venous gas caused by blunt abdominal trauma: Is it a true ominous sign of bowel necrosis? Report of a case. Surg Today 2002;32: 655–658.
14. Gregorič P, Višnar-Perovič A. Clinical importance of portal venous gas detected by abdominal sonography: A report of two cases. Radiol Oncol 2004;38:329–332.
15. Brown MA, Hauschildt JP, Casola G, Gosink BB, Hoyt DB. Intravascular gas as an incidental finding at US after blunt abdominal trauma. Radiology 1999;210:405–408.
16. Stuijvenberg M, Van der Meer NJ, Crolla RM. Pneumatosis intestinalis with gastric pneumatosis and hepatoportal venous gas in blunt abdominal trauma: A case report. Eur J Trauma Emerg Surg 2009;35:505.
17. Vercruysse GA, Adams SD, Feliciano DV. Computed tomographic evidence of hepatic portal venous gas after blunt abdominal trauma does not necessitate surgery. Am Surg 2008;74:335–337.
18. Chevallier P, Peten E, Souci J, Chau Y, Padovani B, Bruneton JN. Detection of portal venous gas on sonography, but not on CT. Eur Radiol 2002;12:1175–1178.
19. Cartwright JA, Breheny C, Major AC, Hill TL, Gow AG. Imaging diagnosis: A case of spontaneous hepatic portal vein gas in an 11-month-old West Highland white terrier. Vet Radiol Ultrasound 2016;57:54–57.

CASE REPORT
RAPPORT DE CAS

Treatment-Refractory Epilepsy Alimentary Therapy (TREAT): A canine case study

Grace Frankel, Melissa Findlay, Leeanne Bargen

ABSTRACT

Half of all epilepsy cases in both humans and canines are identified as idiopathic. Of these cases, 30 to 40% remain treatment-refractory to antiepileptic medications. Several human and dog studies have demonstrated low-carbohydrate diets and dietary medium-chain triglyceride (MCT) supplementation are effective for seizure reduction, with some patients achieving a seizure-free status. Recent evidence suggests the gut-brain axis has an important role in the pathology of neurological disease among both humans and dogs. Altered gut microbiota may have a major role in treatment-refractory epilepsy. This case report describes a dog with treatment-refractory epilepsy experiencing cluster seizures triggered by an altered gut microbiome despite therapeutic drug concentrations of multiple agents. Consideration of an underlying gastrointestinal disorder should be investigated in patients with treatment-refractory epilepsy, despite therapeutic concentrations of several antiepileptic medications. Dietary and gastrointestinal health-promoting interventions for epilepsy should also be considered before add-on pharmacotherapy or euthanasia. For difficult epilepsy cases, we suggest exploring the role of a limited-ingredient, low-carbohydrate diet, MCT supplementation, and/or pre/probiotics to augment pharmacotherapeutic strategies. This information may be critically valuable in designing high-quality, diet-based therapies for epileptic dogs.

Key clinical message:

Gastrointestinal workup, dietary changes to a low-carbohydrate diet, supplementation with MCTs, and addition of pre/probiotics could be considered to augment pharmacotherapeutic strategies in treatment-refractory epilepsy cases in dogs.

RÉSUMÉ

Thérapie alimentaire de l'épilepsie réfractaire au traitement (TREAT) : une étude de cas canin

La moitié des cas d'épilepsie chez les humains et les chiens sont identifiés comme idiopathiques. Parmi ces cas, 30 à 40 % restent réfractaires au traitement aux médicaments antiépileptiques. Plusieurs études sur les humains et les chiens ont démontré que les régimes pauvres en glucides et la supplémentation alimentaire en triglycérides à chaîne moyenne (TCM) sont efficaces pour réduire les crises, certains patients obtenant un statut sans crise. Des preuves récentes suggèrent que l'axe intestin-cerveau joue un rôle important dans la pathologie des maladies neurologiques chez les humains et les chiens. Une altération du microbiote intestinal peut jouer un rôle majeur dans l'épilepsie réfractaire au traitement. Ce rapport de cas décrit un chien souffrant d'épilepsie réfractaire au traitement présentant des crises en grappes déclenchées par un microbiome intestinal altéré malgré les concentrations thérapeutiques

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de plusieurs agents. La possibilité d'un trouble gastro-intestinal sous-jacent est à considérer chez les patients atteints d'épilepsie réfractaire au traitement, malgré les concentrations thérapeutiques de plusieurs médicaments antiépileptiques. Des interventions diététiques et gastro-intestinales favorisant la santé de l'épilepsie doivent également être envisagées avant une pharmacothérapie complémentaire ou l'euthanasie. Pour les cas d'épilepsie difficiles, nous suggérons d'explorer le rôle d'un régime alimentaire pauvre en glucides et à faible teneur en ingrédients, d'une supplémentation en TCM et/ou de pré/probiotiques pour renforcer les stratégies pharmacothérapeutiques. Ces informations peuvent être d'une valeur cruciale pour la conception de thérapies diététiques de haute qualité pour les chiens épileptiques.

Message clinique clé :

Un bilan gastro-intestinal, des changements alimentaires vers un régime pauvre en glucides, une supplémentation en TCM et l'ajout de pré/probiotiques pourraient être envisagés pour renforcer les stratégies pharmacothérapeutiques dans les cas d'épilepsie réfractaire au traitement chez les chiens.

(Traduit par Dr Serge Messier)

Can Vet J 2025;66:378–384

Approximately 1/2 of all epilepsy cases in both humans and canines are identified as idiopathic. Of these cases with unknown etiology, 30 to 40% remain treatment-refractory to antiepileptic medications (1,2). Maintenance pharmacotherapy choices for dogs and humans differ due to variations in gastrointestinal length, gastrointestinal transit time, medication absorption, intestinal pH, and metabolic half-life (3). Due to these differences, pharmacokinetics of medications are altered in dogs, resulting in limited antiepileptic choices. Combination antiepileptic therapies are associated with a high burden of adverse effects and drug interactions that can affect quality of life; therefore, additional management strategies are imperative for treatment-refractory cases.

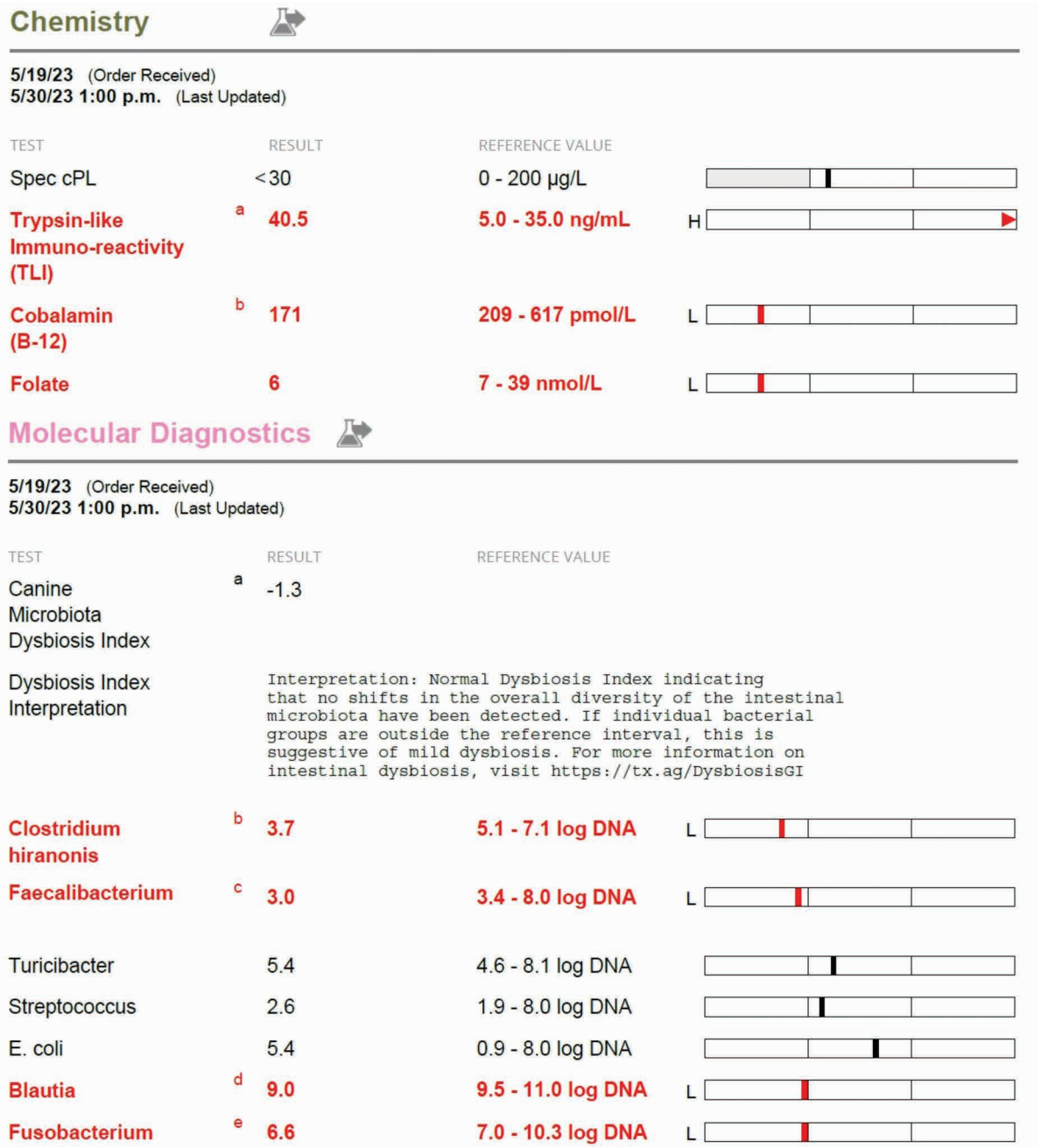
The ketogenic diet has been employed since the 1920s in human medicine as an adjunctive aid for seizure control in children. Due to risk of malnourishment, difficulty of diet adherence, high lipid profiles, and stunted growth, the modified medium-chain triglyceride (MCT) ketogenic diet was developed in the 1970s to allow for a more diverse dietary intake while still limiting carbohydrate content and promoting a ketogenic state. Several studies across humans, rodents, and dogs have demonstrated efficacy of dietary MCT supplementation in supporting seizure control, with dietary changes accounting for a $\geq$ 50% reduction in seizures and some patients achieving a seizure-free status (4).

Recent evidence suggests the gut-brain axis has an important role in the pathology of mental health and neurological disease in both humans and dogs (1,5,6). Altered gut microbiota, specifically an increased α-diversity indicating intestinal dysbiosis, may have a major role in treatment-refractory epilepsy. Altering gut microbiota *via* probiotics or fecal transplant to restore a "normal" gut may provide seizure control by promoting expression of healthy gut bacteria involved in neurotransmitter regulation and synthesis (7).

This case report describes a dog with treatment-refractory epilepsy experiencing cluster seizures triggered by an altered gut microbiome despite therapeutic drug concentrations of multiple agents. This case demonstrated that correcting intestinal malabsorption and altering gut microbiology through diet, pre/probiotics, and addition of MCTs can affect canine seizure control and has potential as a treatment strategy for refractory epilepsy cases.

CASE DESCRIPTION

In September 2020, a neutered male Australian shepherd dog was diagnosed at 3.2 y of age with idiopathic epilepsy. The dog experienced tonic-clonic cluster seizures initiating nocturnally, then continuing to occur every 4 to 6 h while he was falling asleep (video: https://youtube.com/shorts/cXy96a80NNM). The seizures typically lasted for 30 s to 3 min, with a postictal period of 2 to 20 min. The cluster events were not aborted by rectal diazepam, oral clorazepate, or atomized intranasal midazolam, and required hospitalization for administration of IV abortive medications (midazolam and levetiracetam). From September 2020 to April 2021, the dog was hospitalized 4 times for IV abortive antiepileptic therapy and experienced 2 episodes of stress colitis from hospitalization that resolved without intervention. He experienced a seizure-free interval of 15 mo (April 2021 to July 2022), receiving phenobarbital [30 mg,



Chemistry

5/19/23 (Order Received)
5/30/23 1:00 p.m. (Last Updated)

TEST		RESULT	REFERENCE VALUE	
Spec cPL		<30	0 - 200 µg/L	
Trypsin-like Immuno-reactivity (TLI)	a	**40.5**	**5.0 - 35.0 ng/mL**	H
Cobalamin (B-12)	b	**171**	**209 - 617 pmol/L**	L
Folate		**6**	**7 - 39 nmol/L**	L

Molecular Diagnostics

5/19/23 (Order Received)
5/30/23 1:00 p.m. (Last Updated)

TEST		RESULT	REFERENCE VALUE	
Canine Microbiota Dysbiosis Index	a	-1.3		
Dysbiosis Index Interpretation		Interpretation: Normal Dysbiosis Index indicating that no shifts in the overall diversity of the intestinal microbiota have been detected. If individual bacterial groups are outside the reference interval, this is suggestive of mild dysbiosis. For more information on intestinal dysbiosis, visit https://tx.ag/DysbiosisGI		
Clostridium hiranonis	b	**3.7**	**5.1 - 7.1 log DNA**	L
Faecalibacterium	c	**3.0**	**3.4 - 8.0 log DNA**	L
Turicibacter		5.4	4.6 - 8.1 log DNA	
Streptococcus		2.6	1.9 - 8.0 log DNA	
E. coli		5.4	0.9 - 8.0 log DNA	
Blautia	d	**9.0**	**9.5 - 11.0 log DNA**	L
Fusobacterium	e	**6.6**	**7.0 - 10.3 log DNA**	L

FIGURE 1. Results of a canine enteropathy panel of the feces of an epileptic dog before a dietary change.

q8h (serum concentration: 69 µmol/L, therapeutic range: 43 to 172 µmol/L)] and potassium bromide [520 mg/d (concentration: 12 mmol/L, suggested therapeutic range: 10 to 25 mmol/L with concomitant phenobarbital)].

In March 2022, the dog experienced colitis and tested positive for *Circovirus* and *Clostridium perfringens*. Between July 2022 and May 2023, he had 5 episodes of colitis, treated each time with antimicrobials (metronidazole), with subsequent hospital admission ~2 to 3 wk later for cluster seizures (July 2022, August 2022, September 2022, December 2022, and May 2023). At the December 2022 admission, the dog had his first episode of status epilepticus as a dose taper of phenobarbital was initiated to mitigate severe daytime sedation and ataxia from triple antiepileptic therapy after zonisamide was added to the treatment regimen in October 2022. He had been maintained on potassium bromide, phenobarbital, and zonisamide until the time of writing. The owner requested a gastrointestinal workup in May 2023 after observing a temporal relationship between colitis episodes and hospital admissions.

TABLE 1. Nutritional breakdown of medium-chain triglyceride (MCT)-supplemented, raw, low-carbohydrate canine diet.

Ingredient	Protein (g)	Energy from protein (kcal)	Fat (g)	Energy from fat (kcal)	Carbohydrate (g)	Energy from carbohydrate (kcal)	Fiber	Total energy (kcal)
Big Country Raw Beef Dinner[a] (380 g/d)	64.48	258	34.13	307	6.82	27	0.5%	592.4
ED Smith Canned Pumpkin (30 mL)	0.36	1.44	0	0	2.53	10	4 g	11.4
Psyllium husk (1 Tbsp)	0	0	0	0	13.33	53	11.7 g	53
MCT oil (8 mL daily, 9% of daily calories)	0	0	7.5	67.5	0	0	0	67.5
Herring oil (omega-3 supplement) (1 tsp)	0	0	4.77	43	0	0	0	43
Supplements[b] and pre/probiotics[c]	0	0	0	0	0	0	0	0
Totals	64.84	259 (34%)	46.40	418 (54%)	22.68	90 (12%)	14.7 g	767 kcal/d

[a] Ingredients: beef, beef heart, ground beef bone, beef liver, beef spleen, beef kidney, spinach, zucchini, broccoli, cranberries, blueberries, calcium phosphate, calcium carbonate, barley grass, wheat grass, kelp, zinc proteinate, manganese proteinate, copper proteinate, and vitamin E. Full nutritional analysis available from: https://bigcountryraw.ca.
[b] Supplements included vitamin B12 (1000 μg/d), folate (1 mg/d).
[c] Prebiotic/probiotic: 1 scoop daily of Fera Pet Organics (contained *L. acidophilus, L. casei, S. boulardii, L. plantarum, B. infantis, L. bularicus, B. bifidum, B. longum, B. animalis, L. brevis, L. rhamnosus, S. thermophilus,* acacia, inulin, and fructooligosaccharides).

A canine enteropathy panel confirmed bacterial dysbiosis with folate and vitamin B12 deficiency, indicating malabsorption (Figure 1). An abdominal ultrasonographic examination in June 2023 detected moderate diffuse hepatopathy, likely due to chronic administration of phenobarbital (no clinical signs or laboratory findings of liver impairment); possibly enlarged spleen (but normal in echogenicity and echotexture); and a questionable right adrenal nodule, a possible variant of normal (no clinical signs of adrenal disease, normal cortisol). The gastrointestinal tract was unremarkable. Inflammatory bowel disease, exocrine pancreatic insufficiency, and pancreatitis were ruled out by the interpreting veterinary radiologist and an internal medicine specialist.

Several veterinarian-recommended limited-ingredient prescription diets were trialed, as well as a home-cooked diet with consultation from a veterinary nutritionist, with no improvements in bowel health or seizure frequency. In May 2023, there was a complete diet change to a raw, limited-ingredient, low-carbohydrate (12%) diet with supplementation of folate, vitamin B12, omega-3, pre/probiotics, psyllium, and MCT oil (Table 1). A pulsed antibiotic course of tylosin (1/8 tsp, ~325 mg, 3×/wk for 2 mo) was also completed to reset gut microbiota. After the diet changes, in July 2023 the dog had a very brief hospital visit for IV levetiracetam treatment, but seizures were shorter in duration, frequency, and severity. In August 2023, he experienced a single seizure that was treated at home with 3 doses of oral levetiracetam. This was successful in aborting a cluster and avoided hospitalization. Phenobarbital and potassium bromide daily doses and serum concentrations before and after the diet change are shown in Table 2.

DISCUSSION

Ketogenic diet and medium-chain triglyceride supplementation

Although the ketogenic diet has been used for more than a century to control seizures in treatment-refractory epilepsy, the mechanism is still not completely understood. Glucose is the primary energy source of the brain. Epilepsy studies from humans, dogs, and rodents have examined glucose uptake by attaching a radioactive marker (fluoro-2-deoxyglucose) in which the rate of glycolysis can be visualized *via* positron emission tomography brain imaging (4). Glucose metabolism in epileptic brain areas is largely impaired with *hypo*metabolism and low glucose uptake, indicating an overall impairment in glucose utilization. In addition, there are shortages of glucose-derived intermediates and metabolites of the tricarboxylic acid (TCA) cycle, resulting in deficiency of the generation of adenosine triphosphate (ATP) required for appropriate neuronal membrane stabilization. A consequence of poor ATP generation is impaired shuttling of glutamate to glutamine by astrocytes, resulting in overproduction of the excitatory neurotransmitter glutamate (8).

When a seizure occurs, very high amounts of energy are required in the brain. With suboptimal glucose utilization, an energy deficit occurs, resulting in impaired membrane stabilization and thus ongoing seizures. To potentially aid in solving this energy deficit, the brain requires an alternate

TABLE 2. Phenobarbital and potassium bromide doses and drug concentrations before (unshaded) and after (shaded) the dog's diet changes.

Date	Phenobarbital daily dose (mg/d)	Phenobarbital concentration (μmol/L)	Potassium bromide daily dose (mg/d)	Potassium bromide concentration (mmol/L)	Comments
Sep 21/2020	120	104			Initial seizure Sep 12/2020
Jan 3/2021	150	104			
Jan 19/2021	150	109			
Mar 8/2021	180	111			
Mar 19/2021	180	146 (12:00 h)			Pharmacokinetic study
		132 (15:00 h)			Half-life calculated as 24 h (usual: 54 to 72 h)
		119 (18:45 h)			
Apr 16/2021			14 000 mg (loading dose)	12.1	Hospital admission, started potassium bromide
Apr 6/2022	90	69	520	12.0	12 mo seizure-free
Jul 28/2022			520	14.4	Emergency room visit
Aug 9/2022	135	93			Emergency room visit
Sep 16/2022	180	142.7	640	14.4	Hospital admission 3 d
Nov 12/2022			740	25.5	Experiencing ataxia and colitis
May 16 and 17/2023	105	130.4	620	14.4	Diet changed to raw with probiotics and medium-chain triglyceride supplementation after this hospital admission
Jun 9/2023	105	127.1			
Jul 28/2023	135	127.8	600	27.0	Was experiencing severe ataxia/somnolence; doses decreased
Feb 16/2024	105	117.6	320	23.2	6 mo seizure-free since diet changes; no colitis episodes

pathway to generate ATP that does not originate from glucose metabolism. In the ketogenic diet model, production of the C4 ketones acetoacetic acid and beta-hydroxybutyric acid can feed into the TCA cycle as an alternate pathway for ATP production. In a similar manner, supplementation of medium-chain fatty acids octnoate (C8) and decanoate (C10) can also feed into the TCA cycle, generating ATP. In a multicenter study that included 28 dogs with idiopathic epilepsy experiencing 3 or more seizures in the past 3 mo, supplementation with MCT oil (9% of daily calories) resulted in a statistically significant reduction in seizure frequency, from a median of 2.67 to 2.51 seizures/mo, with 2 dogs (7.1%) achieving a seizure-free status and 3 dogs (10.7%) showing a ≥ 50% decrease in seizures over a 6-month interval compared to olive oil-supplemented controls. Eleven dogs (39.3%) had no change or increased seizure activity. (9) As a follow-up to that study, researchers focused on dogs considered treatment-responsive to MCT oils (≥ 50% reduction in seizures) and discovered an increased γ-aminobutyric acid (GABA) concentration during the MCT-consumption phase accompanied by a significant shift of the GABA-glutamate balance (10). There is also emerging evidence that shifts in gut microbiota occur in animals fed with a low-carbohydrate diet, which may contribute to seizure protective effects (11).

Probiotic supplementation: Effects on neurotransmitters

Through metabolism and digestion, gut bacteria produce a wide range of neurotransmitters to communicate with the central nervous system. The *Bacillus* family produces dopamine and norepinephrine; *Enterococcus, Streptococcus,* and *Escherichia* families produce serotonin, with *Escherichia* also producing norepinephrine; bifidobacteria and lactobacilli produce GABA, and lactobacilli also produce acetylcholine (12). As a consequence, gut bacteria may have a major role in regulating neurotransmission, which has implications for disease states in which neurotransmitter imbalance leads to pathology. In epilepsy, serotonin and GABA can be helpful for seizure control as they are inhibitory neurotransmitters, whereas excitatory norepinephrine and a glutamate/glutamine imbalance can contribute to seizure activity (12). Dopamine dysregulation may also have a role in epilepsy as this neurotransmitter has effects on reward, sleep, and behavior (13). Studies in both humans and dogs with treatment-refractory epilepsy

Chemistry

2/17/24 (Order Received)
2/23/24 7:04 p.m. (Last Updated)

TEST		RESULT	REFERENCE VALUE		5/19/23
Spec cPL		<30	0 - 200 µg/L		< 30
Trypsin-like Immuno-reactivity (TLI)	a	32.2	5.0 - 35.0 ng/mL		40.5
Cobalamin (B-12)	b	**1,081**	**209 - 617 pmol/L**	H	**171**
Folate	c	**40**	**7 - 39 nmol/L**	H	**6**

Molecular Diagnostics

2/17/24 (Order Received)
2/23/24 7:04 p.m. (Last Updated)

TEST		RESULT	REFERENCE VALUE		5/19/23
Canine Microbiota Dysbiosis Index	a	2.7			-1.3
Dysbiosis Index Interpretation		Interpretation: The Dysbiosis Index (DI) is significantly increased, consistent with a major shift in the overall diversity of the intestinal microbiota. For more information on intestinal dysbiosis, visit https://tx.ag/DysbiosisGI			Interpret.
Clostridium hiranonis	b	**0.3**	**5.1 - 7.1 log DNA**	L	**3.7**
Faecalibacterium	c	4.2	3.4 - 8.0 log DNA		**3.0**
Turicibacter	d	7.2	4.6 - 8.1 log DNA		5.4
Streptococcus	e	4.1	1.9 - 8.0 log DNA		2.6
E. coli	f	7.5	0.9 - 8.0 log DNA		5.4
Blautia	g	10.2	9.5 - 11.0 log DNA		**9.0**
Fusobacterium	h	8.3	7.0 - 10.3 log DNA		**6.6**

FIGURE 2. Results of a canine enteropathy panel of the feces of an epileptic dog after a dietary change.

have shown a trend toward a higher alpha-diversity in the gut biome, indicating gut dysbiosis. Bacteroidetes was the dominant biome phylum for drug-*responsive* patients, whereas Firmicutes was the dominant phylum for drug-*resistant* patients (1,2). Drug-naïve dogs with epilepsy compared to healthy controls had reduced abundance of GABA-producing and short-chain fatty acid-producing bacteria as well as decreased bacteria associated with protective effects against brain disease (2). Probiotics may be a useful treatment strategy to shift the gut microbiome in favor of inhibitory neurotransmitter production to suppress seizures. In a prospective cohort trial of human patients with treatment-resistant epilepsy, after a 4-month trial of probiotics, 13/45 (28.9%) experienced a ≥ 50% seizure reduction. Quality-of-life scores on a scale of 10 (very well) to 50 (could not be worse) improved significantly, from a mean of 26.45 points before intervention to 19.23 points after intervention (14). Probiotic selection to favor strains of Bacteroidetes, *Lactobacillus,* and bifidobacteria may be helpful for seizure control, based on current literature. Future research utilizing fecal transplant from healthy donors may also be of significant benefit for treatment-refractory cases (15).

Serum drug concentrations overview

Before the diet changes, dose increases and additions to antiepileptic therapy were poorly correlated with therapeutic efficacy. Serum concentrations of antiepileptics were consistently within therapeutic ranges, with excellent adherence to pharmacotherapy. In a pharmacokinetic study with serial blood draws, the average half-life of phenobarbital ranged from 20 to 24 h in this dog, considerably shorter than the average half-life of phenobarbital (54 to 72 h), indicating a rapid-metabolizer status. All phenobarbital concentrations were trough values taken 30 min before the 15:30 dose, and q8h dosing was employed due to the short half-life (dosing at 07:30, 15:30, and 22:45 daily). After diet changes, potassium bromide had significantly higher absorption, facilitating a 47% dose reduction required to reduce side effects (ataxia, hind-leg weakness) and supratherapeutic concentrations. The effect on phenobarbital concentrations was less, but a small dose reduction was also facilitated (22%).

Case update

After the diet changes, adequate supplementation with folate and vitamin B12, addition of pre/probiotics and MCT oil, and a one-time pulse macrolide antibiotic therapy for microflora reset, the dog's recurrent colitis symptoms had resolved, with positive changes in stool consistency and frequency. On a repeat canine enteropathy panel (Figure 2), there were marked improvements in gut flora, except for a decrease in *Clostridium hiranonis*. Folate and vitamin B12 concentrations normalized with supplementation.

At the time of writing, the dog had been seizure-free since August 3, 2023. He had gained 4 kg of healthy weight over a 6-month interval. Dose reduction of potassium bromide (from 600 to 320 mg/d) and phenobarbital (from 120 to 105 mg/d) had been successful. These changes subsequently resolved ataxia, hind-leg weakness, and cognitive adverse effects. The dog was returning to competitive sport (agility, scent, rally obedience) after a 1-year hiatus due to side effects from epilepsy medications and repeated hospital admissions.

In conclusion, consideration of an underlying gastrointestinal disorder should be investigated in patients with treatment-refractory epilepsy despite therapeutic concentrations of several antiepileptic medications. Dietary and gastrointestinal health-promoting interventions for epilepsy should also be considered before add-on pharmacotherapy or euthanasia. Emerging evidence in both humans and dogs implies a link among gut dysbiosis, brain glucose metabolism/uptake, and treatment response to pharmacotherapy. We suggest exploring the role of a limited-ingredient, low-carbohydrate diet, MCT supplementation, and pre/probiotics to augment pharmacotherapeutic strategies in uncontrolled epilepsy cases. This information may be critically valuable in designing high-quality, diet-based therapies for epileptic dogs.

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REFERENCES

1. Peng A, Qiu X, Lai W, *et al.* Altered composition of the gut microbiome in patients with drug-resistant epilepsy. Epilepsy Res 2018;147:102–107.
2. García-Belenguer S, Grasa L, Valero O, Palacio J, Luño I, Rosado B. Gut microbiota in canine idiopathic epilepsy: Effects of disease and treatment. Animals 2021;11:3121.
3. Uriarte A, Maestro Saiz I. Canine *versus* human epilepsy: Are we up to date? J Small Anim Pract 2016;57:115–121.
4. Han FY, Conboy-Schmidt L, Rybachuk G, *et al.* Dietary medium chain triglycerides for management of epilepsy: New data from human, dog, and rodent studies. Epilepsia 2021;62:1790–1806.
5. Chatzikonstantinou S, Gioula G, Kimiskidis VK, McKenna J, Mavroudis I, Kazis D. The gut microbiome in drug-resistant epilepsy. Epilepsia Open 2021;6:28–37.
6. Afzaal M, Saeed F, Shah YA, *et al.* Human gut microbiota in health and disease: Unveiling the relationship. Front Microbiol 2022;13:999001.
7. Ding M, Lang Y, Shu H, Shao J, Cui L. Microbiota–gut–brain axis and epilepsy: A review on mechanisms and potential therapeutics. Front Immunol 2021;12:742449.
8. McDonald T, Puchowicz M, Borges K. Impairments in oxidative glucose metabolism in epilepsy and metabolic treatments thereof. Front Cell Neurosci 2018;12:274.
9. Berk BA, Law TH, Packer RMA, *et al.* A multicenter randomized controlled trial of medium-chain triglyceride dietary supplementation on epilepsy in dogs. J Vet Intern Med 2020;34:1248–1259.
10. Berk BA, Ottka C, Law TH, *et al.* Metabolic fingerprinting of dogs with idiopathic epilepsy receiving a ketogenic medium-chain triglyceride (MCT) oil. Front Vet Sci 2022;9:935430.
11. Olson CA, Vuong HE, Yano JM, Liang QY, Nusbaum DJ, Hsiao EY. The gut microbiota mediates the anti-seizure effects of the ketogenic diet. Cell 2018;173:1728–1741.
12. Yue Q, Cal M, Xiao B, Zhan Q, Zeng C. The microbiota-gut-brain-axis and epilepsy. Cell Molec Neurobiol 2022;42:439–453.
13. Dicks L. Gut bacteria and neurotransmitters. Microorganisms 2022;10:1838.
14. Gomez-Eguilaz M, Ramon-Trapero RL, Perez-Martinez L, Blanco JR. The beneficial effects of probiotics as a supplementary treatment in drug-resistant epilepsy: A pilot study. Benef Microbes 2018;9:875–881.
15. Mejia-Granados DM, Villasana-Salazar B, Lozano-Garcia L, Cavalheiro EA, Striano P. Gut-microbiota-directed strategies to treat epilepsy: Clinical and experimental evidence. Europ J Epilepsy 2021;90:80–92.

CASE REPORT
RAPPORT DE CAS

Suspected spontaneous femoral artery rupture in an otherwise healthy American pit bull

Andrew O. Carter, Kelsey Turley

ABSTRACT

A 1-year-old intact male American pit bull experienced a ruptured right femoral artery without trauma or coagulopathy. The dog was presented with right pelvic limb lameness and progressive subcutaneous swelling and bruising. Radiographs of the region identified soft-tissue swelling in the right thigh musculature. Bloodwork monitoring consisted of packed cell volume and total solids, which continued to decrease during hospitalization. A CT scan showed a non-contrast-enhancing possible mass effect in the region. Multiple blood and plasma transfusions were instituted while the dog was in hospital and in preparation for amputation of the limb due to concern for a neoplastic process. During surgery, upon removal of a blood clot, profuse hemorrhage from a branch of the femoral artery was noted. The artery was ligated and amputation completed without complication. After surgery, the packed cell volume returned to normal and no additional bruising was noted. Histopathologic examination of the amputated limb showed extensive subacute hemorrhage and hematoma formation. No evidence of neoplasia or inflammatory foci was noted. At a 2-week recheck, all bruising had resolved and the dog was walking well.

Key clinical message:

This case report describes the first diagnosis and treatment of a suspected spontaneous femoral artery rupture in veterinary medicine.

RÉSUMÉ

Rupture spontanée de l'artère fémorale chez un pitbull américain autrement en bonne santé

Un pitbull américain mâle intact âgé d'un an a subi une rupture de l'artère fémorale droite sans traumatisme ou coagulopathie. Le chien a été présenté avec une boiterie du membre pelvien droit et une enflure sous-cutanée progressive et des ecchymoses. Les radiographies de la région ont identifié un gonflement des tissus mous dans la musculature de la cuisse droite. Le suivi des analyses sanguines comprenait l'hématocrite et les solides totaux, qui ont continué à diminuer pendant l'hospitalisation. Une tomodensitométrie a montré un possible effet de masse non contrasté dans la région. De multiples transfusions de sang et de plasma ont été instituées pendant que le chien était hospitalisé et en préparation de l'amputation du membre en raison d'une inquiétude concernant un processus néoplasique. Pendant l'opération, après le retrait d'un caillot sanguin, une hémorragie abondante d'une branche de l'artère fémorale a été notée. L'artère a été ligaturée et l'amputation a été réalisée sans complication. Après l'opération, l'hématocrite est revenu à la normale et aucune ecchymose supplémentaire n'a été notée. L'examen histopathologique du membre amputé a révélé une hémorragie subaiguë étendue et la formation d'un hématome. Aucune preuve de néoplasie ou de foyers inflammatoires n'a été notée. Lors d'une réévaluation après 2 semaines, toutes les ecchymoses avaient disparu et le chien marchait bien.

Message clinique clé :

Ce rapport de cas décrit le premier diagnostic et le premier traitement d'une rupture spontanée présumée de l'artère fémorale en médecine vétérinaire.

(Traduit par Dr Serge Messier)

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Spontaneous rupture of the femoral artery has been reported rarely in human medicine (1,2) and has not been reported in veterinary literature. The purpose of this case report is to describe the presentation and successful treatment of a 1-year-old intact male dog that was suspected of having a spontaneous femoral artery rupture.

CASE DESCRIPTION

A 1-year-old intact male, American pit bull dog was brought to the emergency service of a specialty veterinary hospital with acute onset of right pelvic limb lameness, progressive swelling of the right pelvic limb, and recent lethargy. The dog was reported to have been acting normally throughout the evening until it was taken for a walk. During the walk, the dog was allowed to run off-leash but was never out of the owners' sight. Toward the end of the walk, the dog developed a right hind-limb limp. After returning home, the owners noted there was bruising along the inside of the dog's right hind leg and the dog had become lethargic. At ~7 pm that evening, the dog was brought to the emergency department. A thorough history revealed that the dog had a superficial laceration many months earlier that required veterinary treatment and suturing, which healed completely without complication or excessive hemorrhage. The owners could not recall the location of this previous laceration but were confident it was not in the right pelvic limb. There was no additional medical history of note.

Examination that evening revealed that the dog had difficulty walking and had notable lameness in the right pelvic limb. He had a prolonged capillary refill time and a large area of bruising on the medial aspect of the right thigh, extending from the right stifle cranially to the caudal abdomen. An area of firm, subcutaneous swelling was noted to be associated with the bruising along the cranial portion of the right medial thigh. Initial blood pressure, obtained using Doppler, was 56 mmHg. The dog received an initial, 15 mL/kg bolus of IV fluids and then was maintained at a rate of 1.6 mL/kg per hour. Systolic blood pressure immediately improved to 130 mmHg. The area of bruising was shaved and thoroughly investigated; no puncture wounds or other external signs of trauma were seen. Right pelvic limb radiographs were obtained and showed marked, nonspecific, soft-tissue and subcutaneous swelling of the right inguinal region and proximal right thigh. No bony abnormalities were noted.

At the time of admission, packed cell volume (PCV) was 45% and total solids (TS) was 5.8 g/dL. Prothrombin time and partial thromboplastin time were both normal, at 15 s [reference (ref): 11 to 17 s] and 75 s (ref: 72 to 102 s), respectively. Complete blood (cell) count showed a hematocrit of 44.6% (ref: 37.3 to 61.7%), white blood cell count of 6.62 K/μL (ref: 5.05 to 16.76 K/μL), and platelet count of 189 000/μL (ref: 148 000 to 484 000/μL). A buccal mucosal bleeding time was < 4 min (considered normal). Blood chemistry showed mild hypokalemia (3.3 mmol/L; ref: 3.5 to 5.8 mmol/L) and hypochloremia (106 mmol/L; ref: 109 to 122 mmol/L). Point-of-care ultrasonography did not reveal any abnormalities or free fluid in any body cavity but showed a diffusely widened soft-tissue layer with hyperechoic regions among fascial layers, consistent with subcutaneous fluid in the region of the swelling/bruising.

The dog was hospitalized for continued monitoring and further workup. Overnight treatments included IV fluids at 1.5 mL/kg per hour, methadone (0.1 mg/kg, IV, q6h as needed for pain), Yunnan Baiyao (2 capsules q12h), and aminocaproic acid (33 mg/kg, IV, q6h). Rechecks of the PCV and TS were scheduled q4h. At the time of admittance to the intensive care unit, PCV and TS were 34% and 4.2 g/dL, respectively.

Due to ongoing hemorrhage and concern for development of coagulopathy, a transfusion of 3.7 mL/kg of fresh frozen plasma was administered over 3 h. A soft padded pressure bandage was placed on the right hind limb and over the caudal abdomen. Blood typing was also undertaken using the Quick Test DEA 1 (Alvedia, Lyon, France). The dog was DEA 1.1-negative. Following the fresh frozen plasma transfusion, PCV and TS were 29% and 5 g/dL. Overnight and into the following morning, the dog was bright and interactive during examination. Systolic blood pressure ranged between 94 and 130 mmHg, heart rate was between 120 and 140 bpm, and capillary refill time was between 1 and 2 s.

Assessment by the intensive care team later that morning (Day 2 of hospitalization) revealed continued progression of the subcutaneous swelling and bruising (Figure 1). The dog was in lateral recumbency and minimally responsive to interaction. He was vocalizing inappropriately. Mucus membranes were pale and we were unable to measure blood pressure. The dog's heart rate was 144 bpm. The PCV and TS were 25% and 4.8 g/dL, respectively. Hypertonic saline (3 mL/kg) was given over 15 min while blood for transfusion was obtained. A transfusion of stored whole blood (8.5 mL/kg) was administered as a bolus. At the conclusion of the transfusion, the dog was markedly brighter; however, the PCV and TS were 19% and

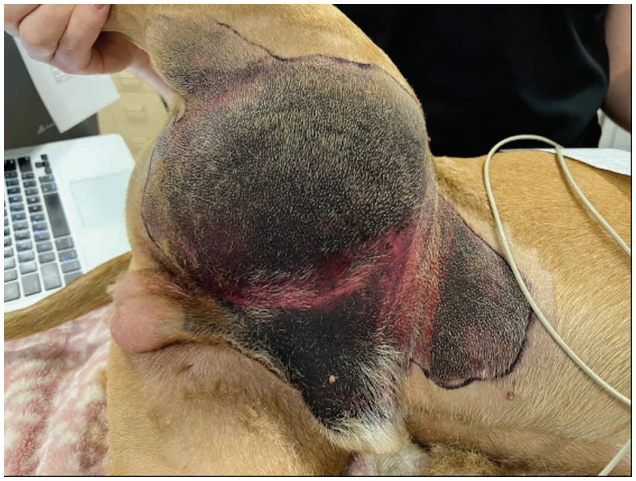

FIGURE 1. Photograph of the dog, showing progression of subcutaneous swelling and border of bruising 6 h into the period of hospitalization.

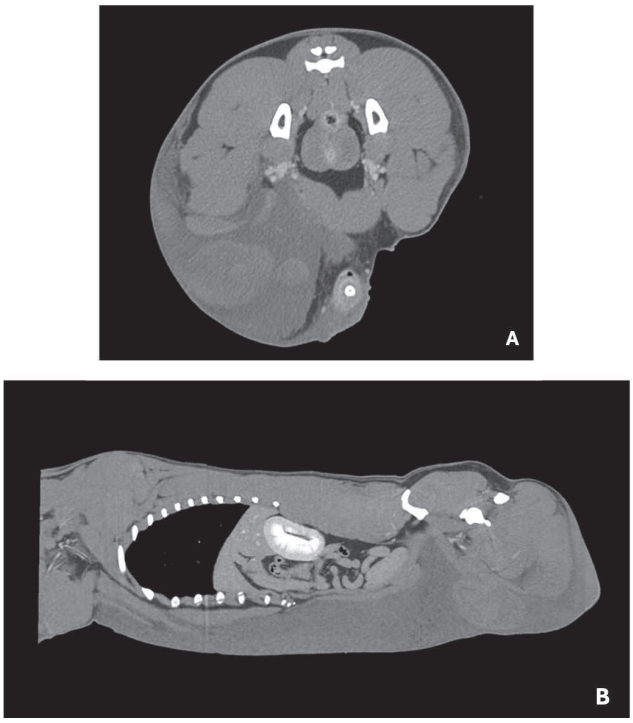

FIGURE 2. Computed tomographic images showing axial (A) and sagittal (B) views of subcutaneous swelling extending from the right pelvic limb to the axilla in this dog. Radiodensity of mass effect was 62 HU and of the swelling extending into the axilla was between 30 and 40 HU.

4 g/dL, respectively. The dog began receiving treatment with diphenhydramine (2 mg/kg, IM, q8h) and pantoprazole (1 mg/kg, IV, q12h) because of potential mast cell tumor degranulation. The methadone was discontinued, and a fentanyl constant-rate infusion was started due to persistent pain. We discussed differential diagnoses with the owners, who opted to pursue a CT scan before surgery. As a CT was unavailable until the following day, the plan was to proceed to surgery without preoperative CT if the dog deteriorated further. Over the next 24 h, the dog received an additional 8.1 mL/kg of stored whole blood and 8.6 mL/kg of packed red blood cells.

On the 3rd day of hospitalization, the dog underwent CT scanning of the thorax, abdomen, and pelvis (Figure 2). A mild pneumomediastinum was noted; however, the remainder of the thoracic scan was within normal limits. The mild pneumomediastinum was deemed incidental by the radiologist. A heterogenous, soft-tissue-attenuating mass effect with minimal contrast enhancement was noted within the medial part of the proximal right thigh and right inguinal region. Differential diagnoses included hemangiosarcoma, mast cell tumor, another neoplastic process, or a possible clot secondary to hemorrhage. As minimal contrast enhancement was present within the mass-like structure (confirmed by HU measurement of 62 HU pre- and post-contrast), previous hemorrhage and hematoma formation was the primary differential diagnosis. Marked subcutaneous hemorrhage was noted in the regions of the right thigh, right inguinal region, and right abdominal and thoracic body wall (CT density measuring between 30 and 40 HU). The remainder of the abdomen and pelvis were normal. There was no evidence of skeletal trauma or lymphadenopathy. Ultrasound-guided fine-needle aspirate was obtained, and results of an in-house cytologic evaluation were consistent with hemorrhage.

Same-day surgical exploration and hind-limb amputation was recommended due to continued, life-threatening hemorrhage. The presurgical PCV and TS were 16% and 4.4 g/dL, respectively. The dog received a fresh whole-blood transfusion (7.8 mL/kg) beginning before surgery. The transfusion finished at the beginning of anesthesia. During anesthesia, the dog had recurring accelerated idioventricular rhythm but no arrhythmias requiring medical intervention. Bloodwork completed before surgery showed a creatinine value of 1.7 mg/dL, an increase of 0.2 mg/dL from values on admission. The rest of the clinical pathology values remained unremarkable. Throughout surgery, the dog received cefazolin (22 mg/kg, IV, q90min).

During surgery, a standard circumferential incision was made around the right proximal thigh. The subcutaneous tissues were noted to be diffusely edematous. Electrocautery was used to transect muscle bellies as encountered on dissection and for hemostasis. A large blood clot was present in the craniomedial aspect of the limb. Upon removal of the

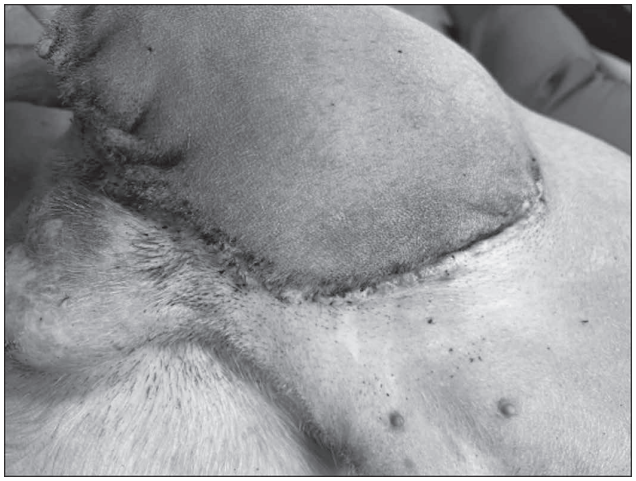

FIGURE 3. Photograph showing the incision at 14 d postoperatively, at the time of suture removal. Note the mild trauma to the scrotum adjacent to the location of the amputated limb; however, the remainder of the bruising had resolved.

blood clot, profuse hemorrhage from a branch of the femoral artery was noted. The artery was clamped with right-angle forceps and ligated with 2 encircling ligatures using 2-0 silk sutures. After ligation, no additional active hemorrhage was noted. A more proximal portion of the femoral artery within the inguinal triangle was located, ligated, and transected. When major nerves were encountered during dissection, bupivacaine liposome injectable suspension (Nocita; Elanco, Greenfield, Indiana, USA) was instilled before transection. Once all soft-tissue attachments to the body were transected, the femur was disarticulated from the pelvis. The entire limb was submitted for histopathologic review. Routine closure of the muscle and soft tissues was completed. Following surgery, the PCV and TS were 17.5% and 4.4 g/dL, respectively. Recovery from anesthesia was uneventful. The dog was maintained on fentanyl (3 μg/kg per hour) and lidocaine (25 μg/kg per minute) after surgery. A fentanyl patch was placed to minimize time in hospital after surgery. A single ketamine dose (0.5 mg/kg, IV) was given and treatment with gabapentin (9 mg/kg, PO) was started on the evening after surgery.

Five hours after surgery, PCV and TS were 26% and 5.6 g/dL, respectively, and creatinine was 0.7 mg/dL. The dog began receiving ampicillin/sulbactam (30 mg/kg, IV, q8h) due to concern for tissue health during surgery. Thirty-six hours post-amputation, the PCV and TS continued to remain stable at 26% and 5.6 g/dL. The dog was discharged ~48 h after surgery with the fentanyl patch in place, gabapentin (9.23 mg/kg, q8h), carprofen (2.3 mg/kg, q8h), trazodone (4.6 mg/kg, q8h), and amoxicillin/clavulanic acid (17.3 mg/kg, q12h). At discharge, the dog was eating well and walking without assistance.

At home, the dog continued to recover from the right hind-limb amputation. No additional signs of bleeding occurred. At time of incision recheck and suture removal, the incision was appropriately healed with no notable bruising present on the body. Mild irritation was observed on the portion of the scrotum adjacent to the amputated limb (Figure 3). Continued monitoring of the area was recommended. At the time of writing, no additional complications or recurrence had been reported.

Histopathologic analysis of the subcutis and musculature of the right thigh revealed locally extensive, subacute hemorrhage and hematoma formation with venous thrombi and interstitial edema. No evidence of neoplasia or cystic structures was identified. In addition, no infectious or inflammatory foci were present. Analysis of the popliteal lymph node was consistent with mild, chronic lymphoid hyperplasia with sinus erythrocytosis. No comments were made by the pathologist that the vessel itself appeared abnormal.

DISCUSSION

The dog in this report was diagnosed with suspected spontaneous femoral artery rupture based on history (no trauma), normal prothrombin time/partial thromboplastin time, imaging (radiographs, CT), and unremarkable histopathologic evaluation of the amputated limb. Spontaneous arterial rupture has not been reported in veterinary medicine. In human literature, spontaneous artery rupture without known trauma is extremely rare and typically associated with pseudoaneurysm formation. Case reports exist for both younger and older age demographics (2,3).

Treatment of arterial rupture in humans can include open or endovascular surgical repair of the vessel or ligation of the affected vessel (1). In animals, treatment of trauma to major vascular structures is always surgical and often necessitates ligation. In cases involving the femoral artery, ligation or vascular repair can be considered; however, amputation is often the procedure of choice, especially when there is concern for neoplasia. In this case, vessel repair was not attempted due to the location of the rupture and in consideration of the differential diagnoses. Vessel repair could be considered in cases where neoplasia is ruled out.

Although no underlying weakness of the vessel wall was noted in this case, this could predispose a vessel to

rupture. These abnormalities include aneurysm and arteriovenous fistula and are rarely reported in veterinary literature. Femoral artery aneurysms have been reported as 0.13% of all peripheral arterial aneurysms in humans (3). Incidentally discovered aneurysms in dogs have a reported incidence of 0.49% in the portal vasculature and 0.21% in the right auricle (4,5). Aneurysms have also been identified secondary to systemic fungal infections (6,7). These vascular abnormalities are difficult to identify before rupture and are usually only diagnosed upon diagnostic imaging or histopathologic evaluation. In the present case, no evidence of an underlying vessel wall abnormality was present or identified *via* CT imaging, intraoperative visualization, or histopathologic evaluation. An underlying connective tissue disorder was less likely due to the conjunction of the histopathologic report showing no connective tissue abnormalities, the intraoperative findings, and good healing after surgery. A single case report in veterinary medicine described pseudoaneurysm associated with the femoral artery following penetrating trauma multiple weeks earlier (8). In the present case, the previous injury required treatment to resolve and did not occur on the limb in question.

Neoplasms such as intramuscular hemangiosarcoma or mast cell tumor, though uncommon, were considered as differential diagnoses in this case due to continued hemorrhage and a potential mass effect in the proximal thigh. Before CT imaging and cytology samples, treatments were initiated for potential mast cell tumor degranulation. Neoplasia was eventually ruled out based on intraoperative findings, in-house cytology, and histopathology. Imaging showed CT density, both pre- and post-contrast, consistent with past hemorrhage and hematoma formation (9). The lack of contrast enhancement made the differential diagnosis of a neoplastic mass less likely. This information could be used in the future to help differentiate neoplastic masses from hemorrhage and hematoma formation. In this case, it did not affect the owners' decision to pursue amputation; however, it could offer additional information for treatment options moving forward.

In conclusion, this case report describes the successful treatment of progressive subcutaneous hemorrhage secondary to suspected spontaneous femoral artery rupture. Although rare, spontaneous femoral artery rupture should be considered as a differential diagnosis for progressive, acute, subcutaneous hind-limb hemorrhage in a dog. CVJ

REFERENCES

1. Siani A, Flaishman I, Siani LM, *et al.* Spontaneous rupture of the superficial femoral artery treated *via* an endovascular approach. Tex Heart Inst J 2008;35:66–68.
2. Darigny S, Astarci P, Elens M. A rare case of spontaneous superficial femoral artery pseudoaneurysm in a young patient: Case report and review of literature. J Surg Case Rep 2021; 2021:rjab327.
3. Karasov IA, Umarov AH, Airapetyan AA, *et al.* Rupture of a deep femoral artery aneurysm with pulsating hematoma: A case report. Future Cardiol 2022;18:765–769.
4. Bertolini G, Caldin M. Computed tomography findings in portal vein aneurysm of dogs. Vet J 2012;193:475–480.
5. Costa A, Cordella A, Caldin M, Bertolini G. Right auricle aneurysm in dogs can be detected with multidetector-row CT and presents with varying morphology. Vet Radiol Ultrasound 2023;64:28–35.
6. Murata Y, Chambers JK, Uchida K, *et al.* Mycotic aneurysm caused by *Graphium* species in a dog. J Vet Med Sci 2015; 77:1285–1288.
7. Gershenson RT, Melidone R, Sutherland-Smith J, Rogers CL. Abdominal aortic aneurysm associated with systemic fungal infection in a German shepherd dog. J Am Anim Hosp Assoc 2011;47:45–49.
8. Tikekar A, Milner HR. Diagnosis and treatment of a suspected pseudoaneurysm of the femoral artery in a working police dog. N Z Vet J 2015;63:121–124.
9. Arnold SA, Platt SR, Gendron KP, West FD. Imaging ischemic and hemorrhagic disease of the brain in dogs. Front Vet Sci 2020;7:279.

CASE REPORT
RAPPORT DE CAS

Amanda J. Butler, Briar Spinney, Laura Perry, Andrea Bourque, William B. Stoughton

Nutritional secondary hyperparathyroidism and subsequent fibrous osteodystrophy in a 3-year-old dromedary camel

ABSTRACT

A 3-year-old female dromedary camel was referred as an urgent case for evaluation of chronic weight loss, facial deformity, and hind-limb lameness. On initial examination, the camel was emaciated, with bilateral masses protruding from the maxillary and mandibular bones and extending into the oral cavity; the lameness could not be assessed due to recumbency. Clinical pathology and fecal flotation findings were consistent with secondary nutritional hyperparathyroidism, hypovitaminosis D, marked anemia, hypoproteinemia, and parasitism. The camel was euthanized based on the presumptive diagnosis of fibrous osteodystrophy, which was confirmed on postmortem examination. Preventative strategies were recommended for future care of dromedaries and included camel husbandry with adequate ultraviolet light exposure, adequate nutrition, appropriate anthelmintic control programs, and vitamin D supplementation.

Key clinical message:

The cause of fibrous osteodystrophy in camels can be multifactorial and include secondary nutritional hyperparathyroidism, hypovitaminosis D due to inadequate exposure to ultraviolet light or intake, and parasitism. Specific nutrient requirements, sun exposure, and anthelmintic protocols are essential for camels living in North America.

RÉSUMÉ

Hyperparathyroïdie nutritionnelle secondaire et ostéodystrophie fibreuse subséquente chez un dromadaire de 3 ans

Une chamelle dromadaire de 3 ans a été référée en urgence pour évaluation par suite d'une perte de poids chronique, d'une déformation faciale et d'une boiterie des membres postérieurs. Lors de l'examen initial, la chamelle était émaciée, avec des masses bilatérales faisant protrusion des os maxillaires et mandibulaires et s'étendant dans la cavité buccale; la boiterie n'a pas pu être évaluée en raison du décubitus. La pathologie clinique et les résultats de la flottaison fécale étaient compatibles avec une hyperparathyroïdie nutritionnelle secondaire, une hypovitaminose D, une anémie marquée, une hypoprotéinémie et un parasitisme. La chamelle a été euthanasiée sur la base du diagnostic présomptif d'ostéodystrophie fibreuse, qui a été confirmé lors de l'examen post-mortem. Des stratégies préventives ont été recommandées pour les soins futurs des dromadaires et comprenaient l'élevage de chameaux avec une exposition adéquate aux rayons ultraviolets, une nutrition adéquate, des programmes de contrôle anthelminthique appropriés et une supplémentation en vitamine D.

Message clinique clé :

La cause de l'ostéodystrophie fibreuse chez les chameaux peut être multifactorielle et inclure une hyperparathyroïdie nutritionnelle secondaire, une hypovitaminose D due à une exposition ou à un apport inadéquat aux rayons ultraviolets et au parasitisme. Des besoins nutritionnels spécifiques, une exposition au soleil et des protocoles anthelminthiques sont essentiels pour les chameaux vivant en Amérique du Nord.

(Traduit par Dr Serge Messier)

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Parathyroid hormone (PTH) aids in maintaining blood calcium, phosphorus, and vitamin D homeostasis (1,2). The parathyroid gland is stimulated to secrete PTH when blood calcium and 1,25-dihydroxycholecalciferol (active vitamin D) levels are low, or when phosphorus levels are high (1). Parathyroid hormone promotes expression on osteoblasts and osteocytes of nuclear factor-kappa B ligand (RANKL), which binds to nuclear factor-kappa (RANK) receptors on osteoprogenitor cells (3). This binding modulates the differentiation of osteoclasts, which results in an overall increased osteoclast activity and breakdown of bone (3). The latter results in loss of bone mass while releasing calcium and phosphorus into circulation. The lost bone is replaced with fibrous tissue to maintain structural integrity; however, this stroma is substantially weaker than bone (4,5). In addition to its effect on bones, PTH works in the kidneys to increase calcium resorption and phosphorus excretion in the renal tubules and collecting ducts. Parathyroid hormone also stimulates the conversion of 25-dihydroxycholecalciferol to the active form 1,25 — dihydroxycholecalciferol in the kidneys (1). Active vitamin D increases calcium and phosphorus absorption in the small intestines (1,2). The combination of these mechanisms leads to an overall increase in blood calcium and decrease in phosphorus.

Inappropriate release of PTH from the parathyroid gland can be classified as either a primary or a secondary condition. Primary hyperparathyroidism results from a functional secretory tumor of the parathyroid gland leading to increased PTH secretion. Primary hyperparathyroidism results in moderate-to-marked hypercalcemia with normal-to-low serum phosphorus levels, in association with inappropriately high PTH concentrations (1,2).

Secondary hyperparathyroidism can occur with both nutritional imbalances and renal disease (6–8). Nutritional hyperparathyroidism can occur secondary to feeding diets low in calcium or high in phosphorus, or with chronic ingestion of oxalate containing plants, which causes decreased calcium absorption in the small intestines (1). Severe parasitism and other malabsorptive conditions can also decrease calcium and vitamin D absorption in the small intestines, leading to hypovitaminosis D and secondary hyperparathyroidism (9–11). Renal disease can result in hyperparathyroidism due to decreased calcium reabsorption and phosphorus excretion by the kidneys, as well as impaired conversion to the active form of vitamin D resulting in impaired gastrointestinal calcium absorption (12).

Fibrous osteodystrophy (FO) results from prolonged osteoclastic activity and resorption of cortical and cancellous bone, which is then replaced by mature fibrous connective tissue. All bones can be affected with FO; however, areas of bone subjected to excessive mechanical stress, such as the bones of the skull, are most commonly and severely affected (1,6–8). Fibrous osteodystrophy can be a sequala to either primary or secondary hyperparathyroidism or hypercalcemia of malignancy (6–8). Secondary nutritional hyperparathyroidism is the most frequently reported cause of FO in camels (13,14). Other causes of FO in animals include renal secondary hyperparathyroidism, hypovitaminosis D, and malabsorption leading to impairment of calcium uptake in the small intestines or neoplasia (9,11,12). In dogs, FO is often referred to as “rubber jaw” and has been reported to be associated with dietary calcium and phosphorus imbalance (15). Similarly, in horses, FO is commonly referred to as “big head” or “bran disease” and is caused by feeding diets high in phosphorus and low in calcium, such as bran (7).

CASE DESCRIPTION

A 3-year-old female dromedary camel was presented as an urgent case to a referral veterinary hospital. The camel had a 6-month history of progressive hind-end weakness and muscle wasting. She had a 3-month history of facial deformities consisting of firm, bilateral maxillary swellings that had progressively worsened. Hind-limb radiographs obtained by the referring veterinarian (rDVM) 2 mo earlier showed evidence of bilateral bony proliferations on the dorsal aspects of both metatarsal bones and bilateral osteoarthritis of the distal intertarsal and tarsal intertarsal joints. Previous treatments had included meloxicam (Metacam; Boehringer Ingelheim, Burlington, Ontario) and firocoxib (Previcox; Boehringer Ingelheim). Dosages, frequency, and duration were unknown, but these medications did not improve the camel’s lameness.

During the summer, the camel was housed with goats and had access to an outdoor shelter. In the winter, she was housed indoors with minimal or no access to ultraviolet (UV) light. She was dewormed with ivermectin (Stromectol; Merck Animal Health, New Jersey, USA) at 6 mo of age and given fenbendazole (Panacur; Merck Animal Health) 2 mo before presentation, after fecal flotation detected trichostrongyle-type eggs. The camel had been fed a concentrated feed specifically formulated for camels (Dodson & Horrell, Nantwich, UK); however, the owner switched her to an equine sweet feed 6 mo before presentation. In addition,

the camel received 1 flake of hay per day, apples, carrots, and a calcium phosphate and glucosamine supplement. The camel had had a good appetite until a few months before presentation, but her facial masses now inhibited normal mastication, and the owner said she was dropping a substantial amount of feed.

A serum biochemistry panel requested by the rDVM showed a moderate total hypocalcemia and hyperphosphatemia (actual values unknown). These blood-work findings, in addition to the history and clinical signs, were suggestive of secondary hyperparathyroidism and subsequent FO.

On presentation, the camel was alert, in sternal recumbency, and refusing to stand. Physical examination revealed emaciation with a body condition score of 1/9 and severe muscle atrophy. The camel vocalized continuously during palpation of her limbs; at that time it was unclear if this was due to musculoskeletal pain, stress of the procedure, or being in an unfamiliar environment. Large, firm, maxillary and mandibular masses were present bilaterally, worse on the left, which extended into the oral cavity. Due to the masses, the camel was dysphagic and could not prehend or masticate feed when offered, and her mouth was continually open. There was minimal airflow through both nares, which suggested extension of the masses into the nasal cavities. The camel's respiratory effort was increased but the respiratory rate was normal. Her temperature was normal at 38.5°C. Heart rate and lung sounds could not be evaluated due to the camel's loud vocalizations during handling. Perineal fecal staining was apparent; the owner stated this was normal when this camel was transported and was related to stress.

Blood was collected from the right jugular vein for a CBC, biochemistry panel, PTH chemiluminescent immunometric assay, serum ionized calcium test, and 25-hydroxyvitamin D immunoassay. The CBC revealed a moderate normocytic, normochromic anemia (hemoglobin concentration: 6.7 g/dL, reference: 8 to 16 g/L; hematocrit: 0.16 L/L, reference: 0.24 to 0.35 L/L), a mild leukopenia characterized by a moderate lymphopenia ($2.22 \times 10^6/mm^3$, min reference: $5.33 \times 10^6/mm^3$), and a moderate hypoproteinemia (47 g/L, reference: 63 to 88 g/L) (13,14). Serum biochemistry panel revealed a marked hypocalcemia (1.31 mmol/L, mean reference: 2.53 mmol/L), a low ionized calcium (0.79 mmol/L, range: 1.0 to 1.33 mmol/L), mild hyperphosphatemia (2.8 mmol/L, range: 1.08 to 2.75 mmol/L), a low creatinine (66 μmol/L, range: 145 to 203 μmol/L), a moderate increase in alkaline phosphatase (871 μ/L, range: 58 to 132 μ/L), and a moderate hypoproteinemia (47 g/L, range: 57 to 66 g/L) characterized by a marked hypoalbuminemia (15 g/L, mean reference: 34 g/L) with a mild hyperglobulinemia (32 g/L, mean reference: 25 g/L) (16,17). A fecal floatation was also done and was positive for trichostrongyle-type eggs.

Differential diagnoses for the anemia in this camel were parasitism *(Haemonchus contortus),* anemia of chronic disease, elevated PTH interfering with erythropoiesis, or compartment 3 gastric ulcers; whereas differential diagnoses for hypoproteinemia included prolonged starvation due to dysphagia, parasitism *(Haemonchus contortus, Ostertagia ostertagi,* or *Trichostrongylus axei),* protein-losing enteropathy, or malabsorption. The low creatinine was attributed to the marked decrease in muscle mass. The increase in alkaline phosphatase was suspected to be due to increases in the bone isoform (B-ALP) *versus* increases in liver alkaline phosphatase (L-ALP) seen with cholestasis. Hyperphosphatemia and low ionized calcium were secondary to inadequate calcium intake in relation to high dietary phosphorus. Parathyroid hormone concentrations were elevated (193.9 pmol/L, mean reference: 11.45 ± 4.84 pmol/L) and 25-hydroxyvitamin D concentrations were low (62 ng/mL, mean reference: 390 ± 45 ng/mL) (18,19). Values used as references for PTH and 25-hydroxyvitamin D concentrations were not from camels living in North America and may not be representative of animals in this region (18,19).

Based on the historical and clinical findings, the working diagnosis was nutritional secondary hyperparathyroidism with subsequent FO. Due to the severity of the facial deformity, dysphagia, emaciation, weakness, and arthritic changes noted by the rDVM, and a grave long-term prognosis, the camel was euthanized. To facilitate euthanasia, the camel initially received an intramuscular sedative bolus consisting of 100 mg ketamine (Narketan; Vétoquinol, Lavaltrie, Quebec), 100 mg xylazine (Xylamax; Vétoquinol), and 15 mg butorphanol tartrate (Torbugesic; Zoetis, Kirkland, Quebec), followed by intravenous administration of 31 200 mg pentobarbital sodium (Euthansol; Merck Animal Health, Kirkland, Quebec). The camel was then submitted for computed tomography and necropsy.

Computed tomography revealed obliteration of the normal maxillary and mandibular architecture (Figures 1, 2). Large, mixed, echogenic, soft-tissue masses were present and replaced the normal bone of the maxillae and mandibles. The sinus cavities were also markedly reduced due to the infiltrative nature of the mass (Figures 1, 2).

Gross necropsy findings revealed diffuse muscle and mucous membrane pallor consistent with anemia. The

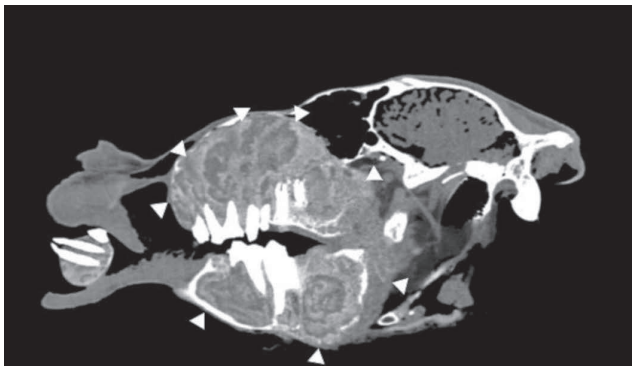

FIGURE 1. Computed tomographic image of a sagittal view of the camel's head, revealing mixed echogenic soft-tissue masses (arrowheads) obliterating the normal bony architecture of the maxilla and mandible.

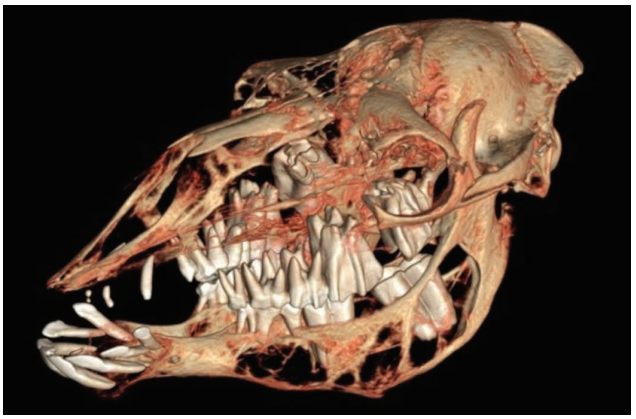

FIGURE 2. Computed tomographic 3D reconstruction image of the camel's head, depicting marked loss of the cortical and medullary bone of maxillae, mandibles, and calvarium. The fibrous tissue is not depicted in this reconstruction, so the maxillary teeth appear to be unattached. The pink areas are regions where only a thin layer of cortical bone remains.

long bones were easily sectioned and serous atrophy of fat was detected within the medullary cavities, indicating the severity of emaciation. There was generalized loss of the medullary bone, and cortices appeared thin. Several small (3 to 4-millimeters in greatest diameter) erosions and indentations were noted on the articular cartilage of right distal tibia, the right lateral 4th tarsal bone, and the articular surfaces in both elbows, consistent with osteoarthritis. In addition, the surfaces of both calcaneal tuberosities were markedly eroded and contained areas of hemorrhage. Moderate ascites, hydrothorax, hydropericardium, and moderate mesenteric edema were present and were attributed to decreased oncotic pressure due to marked hypoalbuminemia. Numerous intraluminal nematodes were identified in compartment 3 of the forestomach and were later identified as *Haemonchus contortus*.

Large, very firm, coalescing masses of pale tan-colored tissue protruded from both maxillary and mandibular bones and extended into the oral cavity (Figure 3). This abnormal tissue replaced most of the bone in these areas, filling and obliterating the nasal passages and sinuses (Figure 3). Examination of histological sections of the masses showed teeth embedded in the dense, fibrous, connective tissue that had replaced bone and marrow spaces. Further histopathological examination of the maxillary and mandibular masses revealed marked loss of cortical and medullary bone and replacement with large masses of poorly cellular, mature, organized fibrous stroma, consistent with FO. The calvarium was markedly thinned and could easily be fractured with little pressure. Histology of long bones confirmed a generalized, severe osteopenia. The parathyroid glands revealed bilaterally symmetrical, moderate, diffuse, chief cell hyperplasia, further confirming the diagnosis of hyperparathyroidism.

Histology of the 3rd compartment of the forestomach showed moderate-to-severe, locally extensive mucosal hyperplasia with mild eosinophilic and lymphofollicular infiltrates suggestive of *Ostertagia ostertagi*. The intestines showed multifocal moderate eosinophilic and lymphoplasmacytic enteritis, which could have caused malabsorption and contributed to the hypoproteinemia. The kidneys appeared normal, both grossly and histologically. The lungs had severe pulmonary congestion and edema associated with euthanasia. The heart had mild, multifocal, acute myocardial degeneration and hemorrhage, which was considered an acute terminal change. No significant microscopic abnormalities were noted in sections of brain (cerebrum, thalamus, brain stem, hippocampus), pituitary gland, thyroid gland, kidney, spleen, or pancreas.

Historical, clinical, and postmortem findings aided in a diagnosis of secondary hyperparathyroidism with subsequent FO; however, no definitive cause could be identified. Poor nutrition with a diet low in calcium and high in phosphorus was suspected. Inadequate UV light exposure resulting in hypovitaminosis D and a high parasite burden leading to malabsorption were considered risk factors in this case. Recommendations focused on dietary and environmental management, appropriate anthelmintic treatment protocols, and vitamin D supplementation.

DISCUSSION

Multiple management factors, including inappropriate nutrition, inadequate UV light exposure, and parasitism, contributed to the development of FO in this camel.

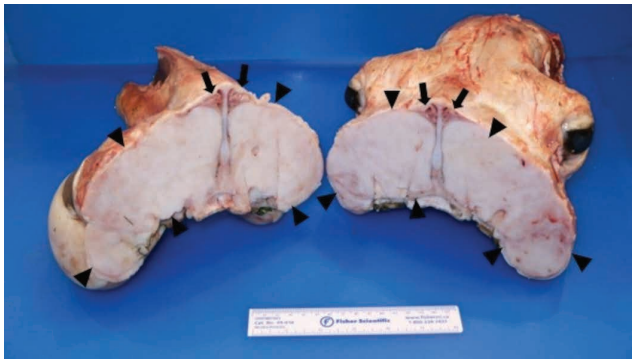

FIGURE 3. Photographic image from the camel's necropsy, showing the fibrous tissue masses (arrowheads) replacing the normal architecture of the maxillary bones and sinuses (arrows) and partially obstructing the nasal passages.

Diets containing low calcium concentrations or abnormal calcium to phosphorous (Ca:P) ratios can lead to secondary nutritional hyperparathyroidism and FO (1). Oxalate-containing plants can be present in lush forage and have been reported to cause this condition in horses (7). Oxalates bind calcium, making it unavailable for absorption (1). A diet low in calcium and protein has been attributed to the development of FO in dromedary camels (14,20). In this case, a complete nutritional analysis, including vitamin E and selenium, was recommended to ensure that all nutrients and minerals were at acceptable levels (21). Vitamin E and selenium concentrations in the diets of animals in Atlantic Canada must be carefully monitored, as the soil in that area is frequently deficient in these nutrients (22).

Camelid maintenance requirements for dietary calcium and phosphorus are 30 mg/kg per day [equivalent to 0.25% of dry matter (DM)] and 20.4 mg/kg per day (equivalent to 0.17% of DM), respectively (23). Growing camelids up to 15 mo of age have higher calcium and phosphorus requirements, at 145 mg/kg per day (0.53 to 0.73% of DM) and 75 mg/kg per day (0.27 to 0.38% of DM), respectively. Forage should have a Ca:P ratio of 1.5:1 or higher (23). Any forage with a Ca:P ratio of 1:1 or lower should not be fed to camelids (23). Alfalfa has a higher Ca:P ratio of 4 to 8:1 and can be a source of calcium for camelids, but can result in obesity (23). Supplementation of calcium may be required in young growing camelids and is dependent on the type of forage fed and results of nutritional analysis.

Hypovitaminosis D results from either decreased UV light exposure or inadequate dietary intake of vitamin D, which results in decreased calcium absorption (1,2). Vitamin D deficiency can cause rickets or osteomalacia, which results in abnormal bone growth or turnover (1,2). These 2 conditions can be exacerbated by nutritional secondary hyperparathyroidism or malabsorptive conditions (4,11). Camels are adapted to arid climates where UV light exposure duration can be greater than 16 h/d; therefore, limited sun exposure could quickly result in calcium dysregulation (24). Studies showed that camels have significantly higher vitamin D levels when compared to ruminants, and variations in plasma vitamin D depend on the season, drought conditions, and sun exposure (19,24,25). In addition, hyponatremia can decrease the amount of calcium and vitamin D absorbed by the small intestines, which was not observed in this case (26). Vitamin D deficiency alone does not normally lead to FO, but combined with secondary hyperparathyroidism and malabsorption, these factors can contribute to pathology. The North American climate poses a challenge for dromedary camels to obtain sufficient UV light during the winter when there is diminished natural sunlight and animals require indoor housing. Recommendations for camels living in Canada include access to winter shelter modified to incorporate natural lighting, with the potential addition of artificial UV light. Vitamin D levels should be evaluated yearly, and vitamin D supplementation should be given to camels living in the northern hemisphere. No specific recommendations are available for daily vitamin D requirements in dromedary camels, but the suggested daily intake for llamas and alpacas living in North America is 30 IU/d (23).

Malabsorption could have contributed to the metabolic derangements identified in the case described herein (9–11). The moderate eosinophilic and lymphoplasmacytic enteritis, accompanied by a large area of mucosal thickening and hyperplasia in compartment 3, are associated with malabsorption. Although the diet may have provided appropriate nutrition for a growing dromedary, the chronic enteritis and parasitism inhibited adequate absorption of essential nutrients (9–11). Secondary hyperparathyroidism and hypocalcemia have been documented in canines with chronic enteropathy — particularly with a protein-losing enteropathy due to decreased intestinal absorption of 25-hydroxyvitamin D and calcium, which could have been a contributing factor in this case (11). If medical management was a feasible option in this case, it would have been focused on reducing the parasite load. Examination of abomasal contents revealed *Haemonchus contortus*. Other changes noted on postmortem examination, specifically extensive compartment 3 mucosal hyperplasia and eosinophilic enteritis, were suggestive of both *Ostertagia ostertagi*

and *trichrostrongylus axei,* respectively. Blood work and necropsy findings of anemia, hypoproteinemia, and tricavitary effusions could all be explained by endoparasitism.

Lameness was the first abnormal clinical sign noted in this camel. Generalized osteopenia, articular erosions, and osteoarthritis, as noted in this case, have been reported in Bactrian and dromedary camels diagnosed with rickets, osteomalacia, or FO (13,14,20,26). The extent of erosion appreciated in this camel's tarsi explained the degree of lameness noted by the rDVM. With FO, osteoclasis at the epiphyseal trabeculae of the long bones results in a lack of support for articular cartilage, causing its collapse and subsequent erosion (27). Therefore, metabolic disturbance should be considered a differential diagnosis for any immature animal with a progressing lameness, particularly any exotic species living in the northern hemisphere.

This case highlighted the potential for metabolic derangements to create rapidly progressive musculoskeletal disease in young, growing dromedary camels living outside their native habitat. Major risk factors identified were gastrointestinal parasitism, inappropriate diet, and inadequate sun exposure. Without a single inciting cause, recommendations were based on a dromedary camel's physiologic needs while also considering the unique challenges related to living in Canada. Implementation of husbandry changes, such as adequate exposure to UV light or vitamin D supplementation, along with adequate nutrition and parasite control could decrease the risk of metabolic bone disease for dromedary camels in the future. CVJ

REFERENCES

1. Toribio R. Endocrine and metabolic diseases. In: Smith BP, Van Metre DC, Pusterla N, eds. Large Animal Internal Medicine. 6th ed. Philadelphia, Pennsylvania: Mosby, 2019: 1352–1420.
2. Constable PD, Hinchcliff KW, Done SH, Grünberg W. Veterinary Medicine. 11th ed. Philadelphia, Pennsylvania: WB Saunders, 2017:1371–1539.
3. Silva BC, Bilezikian JP. Parathyroid hormone: Anabolic and catabolic actions on the skeleton. Curr Opin Pharmacol 2015; 22:41–50.
4. Levine BS, Walling MW, Coburn JW. Intestinal absorption of calcium: Its assessment, normal physiology, and alterations in various disease states. In: Bronner F, Coburn JW, eds. Disorders of Mineral Metabolism. Cambridge, Massachusetts: Academic Press, 1982:103–188.
5. Bikle DD. Vitamin D insufficiency/deficiency in gastrointestinal disorders. J Bone Miner Res 2007;22:V50–V54.
6. Varshney JP, Kumar G, Singh SK. Evaluation of promising biochemical markers of nutritional osteodystrophy in goats. Small Rumin Res 2018;169:86–89.
7. Ospina JC, Doncel B, Verjan N. Maxillofacial fibrous osteodystrophy in equine: Case Report. Braz J Vet Pathol 2014;7: 100–105.
8. Bas A, López I, Estepa JC, Rodríguez M, Aguilera-Tejero E. Nutritional secondary hyperparathyroidism in rabbits. Domest Anim Endocrinol 2005;28:380–390.
9. Ebrahim Z. Effect of gastrointestinal parasites infestation on some hematological and biochemical parameters in sheep. Alex J Vet Sci 2018;59:44.
10. Fekete SG, Kellems RO. Interrelationship of feeding with immunity and parasitic infection: A review. Vet Med (Praha) 2007;52:131–143.
11. Jones C, Jablonski SA, Petroff BK, Langlois DK. Relationship between serum magnesium, calcium, and parathyroid concentrations in dogs with abnormally low serum 25-hydroxyvitamin D concentration and chronic or protein-losing enteropathy. J Vet Intern Med 2023;37:101–109.
12. El-Kishawi AMW, El-Nahas AM. Renal osteodystrophy: Review of the disease and its treatment. Saudi J Kidney Dis Transpl 2006;17:373–382.
13. Lynch MJ, Slocombe RF, Harrigan KE, Laing CJ. Fibrous osteodystrophy in dromedary camels *(Camelus dromedarius)*. J Zoo Wildl Med 1999;30:577–583.
14. Hines ES, Stevenson VB, Patton ME, *et al*. Fibrous osteodystrophy in a dromedary camel. J Vet Diagn Invest 2021;33:144–148.
15. Uhl EW. The pathology of vitamin D deficiency in domesticated animals: An evolutionary and comparative overview. Int J Paleopathol 2018;23:100–109.
16. Bogin E. Clinical pathology of camelides: Present and future. Rev Med Vet (Toulouse) 2000;151:563–568.
17. Teare JA. ISIS Reference Ranges for Physiological Values in Captive Wildlife. Apple Valley, Minnesota, USA: International Species Information System, 2002.
18. Zongping L. Studies on the haematology and trace element status of adult Bactrian camels *(Camelus bactrianus)* in China. Vet Res Comm 2003;27:397–405.
19. El Khasmi M, Faye B. Blood, milk, and meat vitamin D in the dromedary camel. Iran J Appl Anim Sci 2019;9:585–595.
20. Caligiuri R, Kollias GV, Spencer C. Metabolic bone disease in dromedary camels *(Camelus dromedarius)*. J Zoo Wildl Med 1989;20:482–487.
21. Hashi AM, Kamoun M. Feed requirements of the camel. In: Tisserand JL, ed. Elevage et Alimentation du Dromadaire [Breeding and Feeding the Camel]. Zaragoza, Spain: CIHEAM Zaragoza, 1995:71–80.
22. Winter KA, Gupta UC. Selenium content of forages grown in Nova Scotia, New Brunswick, and Newfoundland. Can J Anim Sci 1979;59:107–111.
23. Van Saun RJ. Feeding and nutrition. In: Niehaus AJ, ed. Medicine and Surgery of Camelids. Hoboken, New Jersey: Wiley-Blackwell, 2022:55–107.
24. Mohamed HE. Vitamin D status in camels *(Camelus dromedarius)*. Res J Biol Sci 2008;3:446–447.
25. El Khasmi M, Faye B. Parathyroid hormone-related peptide and vitamin D in phosphocalcic metabolism for dromedary camel. Iran J Appl Anim Sci 2011;1:203–211.
26. Zongping L. Studies on rickets and osteomalacia in Bactrian camels *(Camelus bactrianus)*. Vet J 2005;169:444–453.
27. Craig LE, Dittmer KE, Thompson K. Bones and joints. In: Maxie MG, ed. Jubb, Kennedy & Palmer's Pathology of Domestic Animals. 6th ed. Vol 1. Philadelphia, Pennsylvania: WB Saunders, 2016:74–80.

CASE REPORT
RAPPORT DE CAS

Suyeon Kim, Keunhwan Jang, Seung-Bum Cho, Songju Oh, Jungwoo Han, Ha-Jung Kim

Feline atopic syndrome: An insight into its effects on the central nervous system through vestibular disease

ABSTRACT

A 2-year-old male Abyssinian cat was presented with a left head tilt, lethargy, compulsive walking with a left-circling tendency, medial strabismus, a delayed pupillary light reflex, abdominal papules, and severe pruritus that began at 4 mo of age. Imaging revealed mineral opacity foci in the left ear, and magnetic resonance imaging confirmed heterogeneous signal changes, suggesting a diagnosis of otitis interna. No abnormal findings were observed on cerebrospinal fluid tests. Treatment with prednisolone, antibiotics, a hypoallergenic diet, and probiotics led to the resolution of skin issues and neurological improvement. Despite a persistent head tilt after more than 5 mo, there was no recurrence of severe neurological symptoms during the tapering off of prednisolone treatment.

Key clinical message:

This is the first case report illustrating the potential impact of feline atopic syndrome on the central nervous system. It emphasizes the importance of viewing feline atopic syndrome as more than just a skin disorder.

RÉSUMÉ

Syndrome atopique félin : un aperçu de ses effets sur le système nerveux central par le biais d'une maladie vestibulaire

Un chat abyssin mâle de 2 ans a été présenté avec une inclinaison de la tête vers la gauche, de la léthargie, une marche compulsive avec une tendance à tourner vers la gauche, un strabisme médial, un réflexe pupillaire à la lumière retardé, des papules abdominales et un prurit sévère qui a commencé à l'âge de 4 mois. L'imagerie a révélé des foyers d'opacité minérale dans l'oreille gauche et l'imagerie par résonance magnétique a confirmé des changements de signal hétérogènes, suggérant un diagnostic d'otite interne. Aucune anomalie n'a été observée lors des tests effectués sur du liquide céphalorachidien. Le traitement avec de la prednisolone, des antibiotiques, un régime hypoallergénique et des probiotiques a conduit à la résolution des problèmes cutanés et à une amélioration neurologique. Malgré une inclinaison persistante de la tête après plus de 5 mois, il n'y a pas eu de récidive de symptômes neurologiques graves pendant la diminution progressive du traitement par prednisolone.

Message clinique clé :

Il s'agit du premier rapport de cas illustrant l'impact potentiel du syndrome atopique félin sur le système nerveux central. Il souligne l'importance de considérer le syndrome atopique félin comme bien plus qu'une simple maladie cutanée.

(Traduit par Dr Serge Messier)

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Otitis media/interna (OMI) is recognized as a prevalent cause of peripheral vestibular disease in cats (1,2) and has the potential to alter the fluid composition of the inner ear (3). It leads to the impairment of sensory structures in the vestibular system, affecting balance (4).

Animals with prolonged ear issues may exhibit progressive symptoms of central vestibular disease (CVD) (5). Among cats showing central vestibular signs, 93% display a subdued mental state, 90% exhibit cranial nerve deficits other than the facial and vestibulocochlear nerves, and 85% demonstrate ataxia (2).

Intracranial complications arising from OMI in cats may not be readily recognized due to the low prevalence of the disease, and 53% of affected cats do not exhibit evidence of external or middle ear disease upon otoscopic examination (6).

In addition, although 16 to 20% of cats with feline atopic syndrome are reported to have otitis externa (7), cats, unlike dogs, very rarely develop otitis media as a result of otitis externa (8). Cats may also have otitis media without otitis externa (9).

This report presents a rare case demonstrating that feline atopic syndrome is a potential cause of otitis interna. It is also the first case to show that atopic syndrome in cats may affect the central nervous system.

CASE DESCRIPTION

A 4-month-old male Abyssinian cat was brought to a veterinary hospital due to circling and falling behavior. A complete history revealed that the cat had received the second dose of feline viral rhinotracheitis, calicivirus, panleukopenia (FVRCP) vaccine and the first dose of leukemia vaccine. The cat had also received a dewormer to prevent heartworm and internal and external parasites. The initial diagnosis involved otitis externa and media based on skull radiography and otoscopy examinations and considering the presence of neurological symptoms. Otoscopic examination revealed a large amount of brown cerumen in the ear canal, along with erythema, swelling, and an opaque tympanic membrane. No abnormalities were observed on skull radiography.

Temporary improvement of neurological signs occurred when the external ear canals were cleaned. However, the cat subsequently developed a left-sided head tilt, right-jerk horizontal nystagmus, compulsive walking, and lethargy. One year later, systemic papules appeared and pruritus developed. Compulsive walking and an obtunded mentation simultaneously developed. Although prednisolone, amoxicillin-clavulanic acid, and marbofloxacin were administered for 8 mo, the systemic papules, pruritus, and neurological signs exhibited a waxing and waning pattern.

At 2 y of age, the cat was referred to the veterinary medical teaching hospital for further investigation. The cat was presented with a left head tilt, lethargy, diarrhea, and diffuse papules on the abdominal skin. Upon physical examination, the cat exhibited mild lethargy, and multiple papules and crusts were noted in the abdominal and inguinal regions (Figure 1). The cat's visual analog scale score was 9.5 out of 10, indicating severe pruritus (10–12).

Cytologic examination of the abdominal papules revealed a low number of cocci. There were no respiratory signs, and the cat's vital signs were within normal limits. Upon microscopic evaluation of a fecal swab to identify the organism responsible for the diarrhea, dysbiosis was observed. The cat displayed a left head tilt (Figure 2), compulsive walking with a left-circling tendency, medial strabismus, and a delayed pupillary light reflex in both eyes. Otoscopic examination to investigate if otitis was causing the head tilt did not reveal any evidence of external or middle ear disease. Cytologic evaluations of swabs of the ear canal did not reveal any specific findings. A chemistry panel indicated elevated levels of aspartate aminotransferase (53 U/L; reference range: 0 to 48 U/L) and creatine kinase (402 U/L; reference range: 0 to 314 U/L). No abnormalities or notable results were detected on the complete blood (cell) count, electrolyte analysis, blood gas analysis, or coagulation panel. On skull radiography, multiple opaque foci were identified in the left auricle and external ear canal. This finding suggested the presence of mineralization in the ear canal wall, which can be observed in cases of otitis externa (13). Similar changes can occur when otitis externa becomes chronic in cats (9). For this reason, the possibility of otitis externa could not be excluded, even in the absence of clear abnormalities in the external ear canal. Therefore, the differential diagnoses included food allergy, feline atopic syndrome, OMI resulting from chronic otitis externa, a nasopharyngeal polyp/mass, and central nervous system disease.

Magnetic resonance imaging (MRI) with clear contrast enhancement revealed a heterogeneous signal change in the left inner ear (Figure 3), characterized by T2-weighted hypointensity, T2-fluid-attenuated inversion recovery (FLAIR) hypointensity, and T1-weighted isointensity. In addition, mild contrast enhancement of the right facial and vestibulocochlear nerves (Figure 3 A) and mild thickening

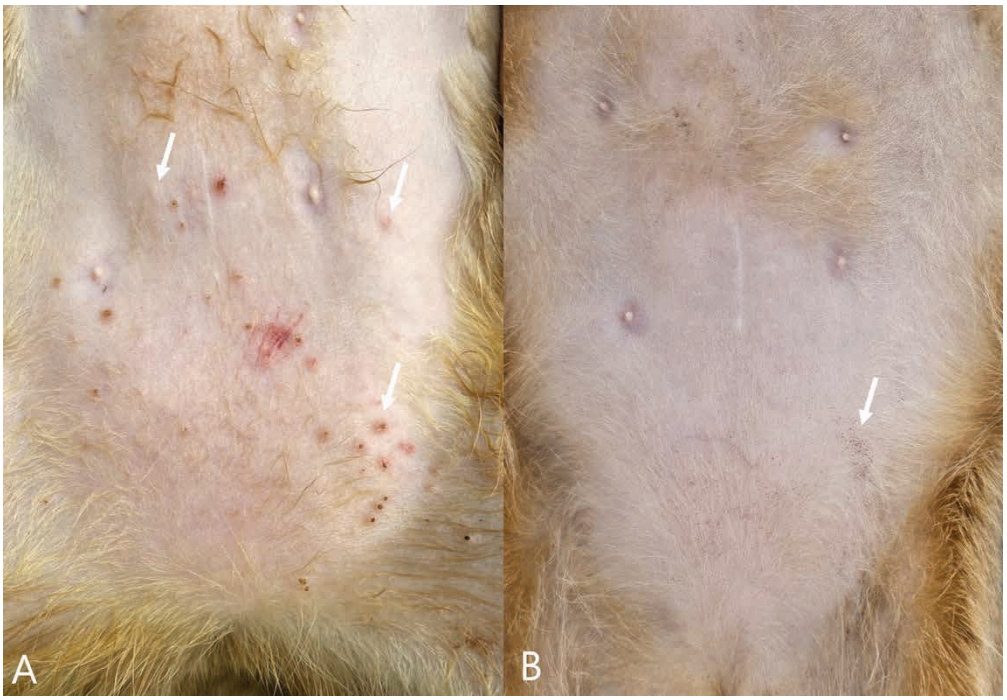

FIGURE 1. A — At the first visit, there were papules and crusts (arrows) in the cat's abdominal and inguinal regions. B — After 2 mo of treatment, the papules and crusts disappeared, leaving a few comedones (arrow) remaining.

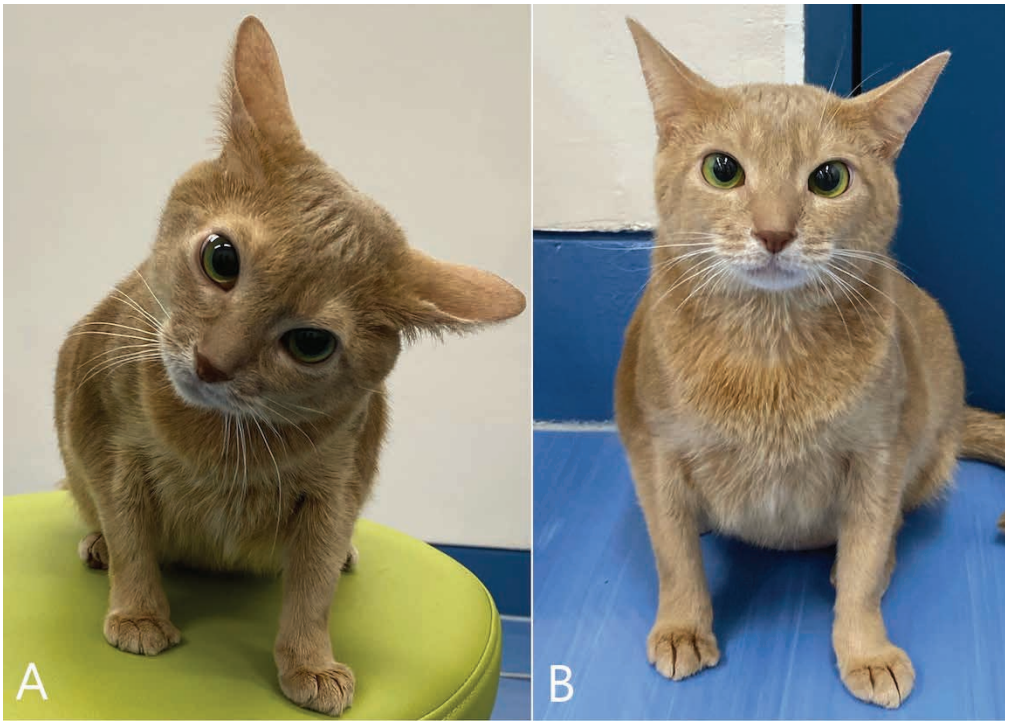

FIGURE 2. A — The cat exhibited a left head tilt; however, there was no evidence of external or middle ear disease as the cause of the head tilt. B — Although a slight left head tilt persisted after treatment, there was a clear improvement compared with before treatment.

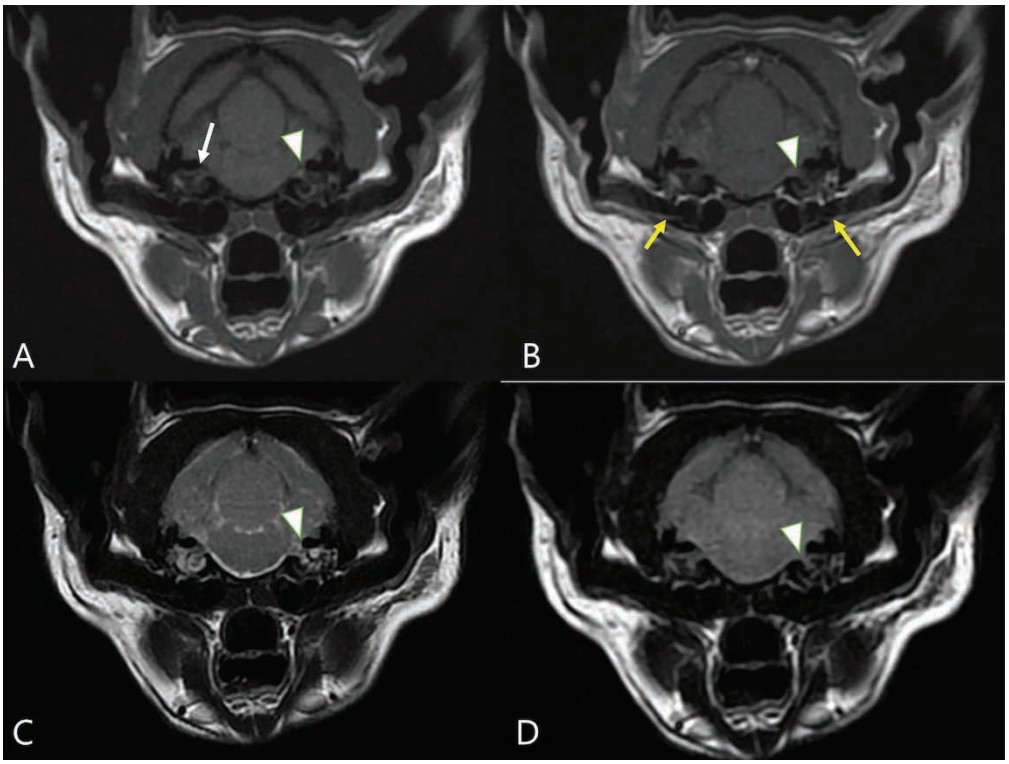

FIGURE 3. A to D — Transverse magnetic resonance images showing a heterogeneous signal change in the cat's left inner ear (arrowhead) with T1-weighted isointensity (A) exhibiting clear contrast enhancement (B), T2-weighted hypointensity (C), and T2-fluid-attenuated inversion recovery (FLAIR) hypointensity (D). In addition, mild contrast enhancement of the right facial and vestibulocochlear nerve (white arrow in A) and mild thickening and enhancement of the walls of the bilateral external acoustic meatus (yellow arrows in B) were observed.

and enhancement of the walls of the bilateral external acoustic meatus were observed (Figure 3 B). Results of feline cerebrospinal fluid (CSF) infectious disease polymerase chain reaction panel and microbial culture tests were normal.

Combining the above results, a diagnosis of peripheral and central vestibular disease stemming from otitis interna was established. Food allergies and feline atopic syndrome remained as differential diagnoses. Treatment included the administration of an anti-inflammatory agent (prednisolone: Solondo tablet; Yuhan, Seoul, Korea), 1 mg/kg, PO q12h and antibiotics (cefixime: Cefixime capsule; Withus Pharm, Seoul, Korea, 12.5 mg/kg, PO, q12h; and marbofloxacin: Marbocyl tablet; Vetoquinol Korea, Gyeonggi, Korea, 5.5 mg/kg, PO, q24h). Dietary restriction was implemented by feeding hypoallergenic cat food (Hill's Prescription Diet z/d Dry Cat Food; Hill's Pet Nutrition, Overland Park, Kansas, USA). Probiotics (*Lactobacillus sakei* proBio65, LACTOVET freeze-dried powder; ProBionic, Jeollabuk-do, Korea), 2×10^9 CFU, q24h, were prescribed to manage dysbiosis.

After 2 mo of treatment, the papules and crusts disappeared (Figure 1) and the diarrhea resolved. The visual analog scale score indicating pruritus improved to 0 out of 10. In addition, cytologic assessment revealed the absence of cocci on the skin, and dysbiosis was not observed on a repeated rectal swab. The cat displayed improved mentation. The medial strabismus had resolved and compulsive walking with a left-circling tendency had ceased. Although the left head tilt and delayed pupillary light reflex in both eyes persisted, a clear improvement was evident compared with the cat's condition before treatment (Figure 2).

Despite the feeding of hypoallergenic cat food and probiotics for $>$ 5 mo, new papules appeared on the abdomen. Eosinophils were detected on cytology and no infectious agents were identified. Thus, a tentative diagnosis of feline atopic syndrome was made based on the cutaneous symptoms and the response to anti-inflammatory therapy. At

the time of writing, the cat was undergoing investigations to determine environmental causes, and treatment plans were being scheduled accordingly.

Despite the stepwise decrease in prednisolone over 5 mo from the start of treatment, there was no reappearance of the cat's obtunded mentation, compulsive walking with a left-circling tendency, or medial strabismus. However, the left head tilt remained.

DISCUSSION

This study reports a case with a tentative diagnosis of feline atopic syndrome affecting both the peripheral and central nervous systems. Diagnosis was supported by clinical signs, neurological examination, MRI findings, and response to treatment. To our knowledge, this is the first reported case demonstrating the potential effect of feline atopic syndrome on the central nervous system.

Reports illustrating the connection between atopic dermatitis and the central nervous system are rare. In humans, the association between atopic dermatitis and abnormal brain structures (including decreased size of caudate nucleus, cortex, and gyrus) has been reported (14), but the precise process remains unclear. In dogs with syringomyelia, including OMI, high levels of interleukin (IL)-31 were observed in CSF samples, but there appeared to be no correlation with itching or pain (15).

In this case, there were no chronic respiratory symptoms, tumors, foreign bodies, trauma, or history of a tick infestation that could have caused OMI. Ultimately, considering that atopy might lead to increased activity of neutrophils in otitis media, likely due to the heightened reaction of preactivated inflammatory cells to bacterial presence as shown in humans (16), the inflammatory state may also be caused by immune dysfunction in feline atopic syndrome (17). Furthermore, the inner ear communicates directly with the middle ear in cats (18), which suggests that feline atopic syndrome may be a cause of otitis interna. In addition, although progression from otitis externa to otitis media is rare in cats (8), considering that control of otitis externa in cats with allergic skin disease requires addressing the underlying allergy (9), we suggest the otitis externa that occurred at 4 mo of age in this cat persisted as an underlying condition with uncontrolled feline atopic syndrome and progressed to otitis interna over a period of more than a year.

Interestingly in this case, there were not only symptoms suggestive of peripheral vestibular disease, such as the left head tilt and compulsive inclination to circle to the left, but also symptoms raising suspicion of CVD, including the animal's obtunded mentation, delayed pupillary light reflex, and medial strabismus in both eyes.

There is 1 published report indicating that the spread of infection from the inner ear to the central nervous system is signified by the presence of pleocytosis in the CSF, as observed in 10 of 11 cases of canine OMI (19). In humans, it was reported that the cochlear aqueduct serves as a direct path for infections and inflammation between the CSF and the inner ear (20). The neurological outcomes of acute meningitis caused by OMI are influenced by the presence of tumor necrosis factor, prostaglandins, and IL-1 (21).

Unfortunately, due to insufficient CSF volume, we were unable to conduct cytology and fluid analysis, which precluded CSF culture. In addition, MRI findings did not reveal any lesions in the brain stem or diencephalon, and thus we could not confirm intracranial inflammation. However, considering the report that 15% of cats displaying clinical signs of CVD do not have detectable lesions on MRI despite having a focal clinical localization (2), reports of decreased production of IL-1 and tumor necrosis factor associated with the use of dexamethasone in human bacterial meningitis (21), and the clinical improvement observed in the current case with anti-inflammatories (such as the cat's improved mentation, cessation of compulsive left circling, and disappearance of medial strabismus), we suggest that the clinical signs described herein likely involved not only the peripheral vestibular system but also the central vestibular system.

Although inner-ear lavage and bacterial culture were not attempted due to a lack of owner consent, the cat was treated concurrently with anti-inflammatory drugs and antibiotics before referral. There was no improvement in neurological symptoms after $>$ 8 mo of antibiotic and prednisolone treatment before admission to the referral hospital. Considering that neurological symptoms improved after the management of feline atopic syndrome, and that no bacteria were cultured from the CSF, we postulate that feline atopic syndrome contributed to the development of vestibular disease affecting the central nervous system.

Despite mild contrast enhancement of the right facial and vestibulocochlear nerves on MRI, the clinical signs observed were left head tilt and a left-circling tendency. The lesion in the right facial and vestibulocochlear nerves might have been less severe than the lesion in the left inner ear, considering that head tilt can occur when one side is more severely affected in cases of bilateral vestibular disease (22).

Despite treatment, the persistent left head tilt was believed to stem from permanent damage to the vestibulocochlear nerve (23). The limited regenerative capacity

of vestibular and cochlear sensory cells in adult mammals (24), as evidenced in this cat, can contribute to a lasting impairment of balance function (4).

In conclusion, we report a rare case indicating that feline atopic syndrome (and/or otitis externa due to feline atopic syndrome) could be potential causes of otitis interna. It is also the first reported case demonstrating that atopic syndrome in cats may affect the central nervous system.

Comprehensive diagnostic evaluations for vestibular syndrome should be conducted in these cases. The link between feline atopic syndrome and central nervous system involvement emphasizes the importance of viewing feline atopic syndrome as more than just a skin disorder.

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REFERENCES

1. Grapes NJ, Taylor-Brown FE, Volk HA, De Decker S. Clinical reasoning in feline vestibular syndrome: Which presenting features are the most important? J Feline Med Surg 2021;23:669–678.
2. Negrin A, Cherubini GB, Lamb C, Benigni L, Adams V, Platt S. Clinical signs, magnetic resonance imaging findings and outcome in 77 cats with vestibular disease: A retrospective study. J Feline Med Surg 2010;12:291–299.
3. Penido Nde O, Chandrasekhar SS, Borin A, Maranhão AS, Gurgel Testa JR. Complications of otitis media: A potentially lethal problem still present. Braz J Otorhinolaryngol 2016;82:253–262.
4. Paulin J, Morshed M, Armién AG. Otitis interna induced by *Cryptococcus neoformans* var. *grubii* in a cat. Vet Pathol 2013;50:260–263.
5. Sturges BK, Dickinson PJ, Kortz GD, *et al*. Clinical signs, magnetic resonance imaging features, and outcome after surgical and medical treatment of otogenic intracranial infection in 11 cats and 4 dogs. J Vet Intern Med 2006;20:648–656.
6. Moore SA, Bentley RT, Carrera-Justiz S, Foss KD, da Costa RC, Cook LB. Clinical features and short-term outcome of presumptive intracranial complications associated with otitis media/interna: A multi-center retrospective study of 19 cats (2009–2017). J Feline Med Surg 2019;21:148–155.
7. Santoro D, Pucheu-Haston CM, Prost C, Mueller RS, Jackson H. Clinical signs and diagnosis of feline atopic syndrome: Detailed guidelines for a correct diagnosis. Vet Dermatol 2021;32:26.
8. Swales N, Foster A, Barnard N. Retrospective study of the presentation, diagnosis and management of 16 cats with otitis media not due to nasopharyngeal polyp. J Feline Med Surg 2018;20:1082–1086.
9. Brame B, Cain C. Chronic otitis in cats: Clinical management of primary, predisposing and perpetuating factors. J Feline Med Surg 2021;23:433–446.
10. Colombo S, Sartori R, Schievano C, Borio S. Development and validation of an owner-assessed Visual Analog Scale for feline pruritus severity scoring (VAScat). Vet Dermatol 2022;33:407–413.
11. Brame BE, Canning P, Morris DO, Drobatz KJ, Rook K, Cain CL. Interobserver reliability of Feline Dermatitis Extent and Severity Index (FEDESI) and Scoring Feline Allergic Dermatitis (SCORFAD) and the relationship between lesion scores and pruritus. Vet Dermatol 2021;32:492.
12. Hill PB, Lau P, Rybnicek J. Development of an owner-assessed scale to measure the severity of pruritus in dogs. Vet Dermatol 2007;18:301–308.
13. Thrall DE. Textbook of Veterinary Diagnostic Radiology. 7th ed. Philadelphia, Pennsylvania: Elsevier, 2018.
14. Chen Y, Cui L, Li H, Gao A. Abnormal brain structure in atopic dermatitis: Evidence from Mendelian randomization study. Skin Res Technol 2023;29:e13515.
15. Lemke L, Carlson R, Flegel T, *et al*. Interleukin-31 in serum and cerebrospinal fluid of dogs with syringomyelia. BMC Vet Res 2023;19:244.
16. Hurst DS, Venge P. Evidence of eosinophil, neutrophil, and mast-cell mediators in the effusion of OME patients with and without atopy. Allergy 2000;55:435–441.
17. Gedon NKY, Mueller RS. Atopic dermatitis in cats and dogs: A difficult disease for animals and owners. Clin Transl Allergy 2018;8:41.
18. Sula MM, Njaa BL, Payton ME. Histologic characterization of the cat middle ear: In sickness and in health. Vet Pathol 2014;51:951–967.
19. Orlandi R, Gutierrez-Quintana R, Carletti B, *et al*. Clinical signs, MRI findings and outcome in dogs with peripheral vestibular disease: A retrospective study. BMC Vet Res 2020;16:159.
20. Di Stadio A, De Luca P, Koohi N, *et al*. Neuroinflammatory disorders of the brain and inner ear: A systematic review of auditory function in patients with migraine, multiple sclerosis, and neurodegeneration to support the idea of an innovative 'window of discovery'. Front Neurol 2023;14:1204132.
21. Roos KL. The use of adjunctive therapy to alter the pathophysiology of bacterial meningitis. Clin Neuropharmacol 1995;18:138–147.
22. Ettinger SJ, Feldman EC, Cote E. Textbook of Veterinary Internal Medicine. 8th ed. St. Louis, Missouri: Elsevier, 2017.
23. Nelson RW, Couto CG. Small Animal Internal Medicine. 6th ed. St. Louis, Missouri: Elsevier Health Sciences, 2020:1109–1116.
24. Groves AK. The challenge of hair cell regeneration. Exp Biol Med 2010;235:434–446.

CASE REPORT
RAPPORT DE CAS

Pulmonary vein stenosis secondary to a mediastinal mass in a cat

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ABSTRACT

This report describes the case of a 5-year-old spayed female munchkin cat that was presented with respiratory distress. Thoracic radiography revealed pleural effusion with a diffuse interstitial lung pattern. Echocardiography identified a mass compressing the left atrium, accompanied by a turbulent jet-like flow from the pulmonary veins, with a velocity of 1.6 m/s. Computed tomography revealed a diffuse, homogenous mediastinal mass compressing the dorsal border of the left atrium and surrounding the ascending aorta and the pulmonary arteries and veins. The distal caudal pulmonary veins were dilated, indicating pulmonary vein stenosis secondary to the mediastinal mass. The caudodorsal ostium was stenotic due to the mediastinal mass compression. In addition, bilateral adrenomegaly and multifocal masses were evident in the kidneys, stomach, and cecum. Lymphoma was diagnosed in the cecal mass using cytology and PCR for antigen receptor rearrangements (PARR). This case describes clinically significant acquired pulmonary vein stenosis secondary to a mediastinal mass, which was likely responsible for the respiratory distress and pleural effusion.

Key clinical message:

To our knowledge, this is the first report of acquired pulmonary vein stenosis secondary to a mediastinal mass in a cat. Computed tomography could be helpful in confirming pulmonary vein stenosis and identifying its etiology.

RÉSUMÉ

Sténose de la veine pulmonaire secondaire à une masse médiastinale chez un chat

Ce rapport décrit le cas d'une chatte munchkin stérilisée âgée de 5 ans présentée avec une détresse respiratoire. La radiographie thoracique a révélé un épanchement pleural avec un motif pulmonaire interstitiel diffus. L'échocardiographie a identifié une masse comprimant l'oreillette gauche, accompagnée d'un flux turbulent de type jet provenant des veines pulmonaires, avec une vitesse de 1,6 m/s. La tomodensitométrie a révélé une masse médiastinale diffuse et homogène comprimant le bord dorsal de l'oreillette gauche et entourant l'aorte ascendante et les artères et veines pulmonaires. Les veines pulmonaires caudales distales étaient dilatées, indiquant une sténose de la veine pulmonaire secondaire à la masse médiastinale. L'ostium caudo-dorsal était sténosé en raison de la compression de la masse médiastinale. De plus, une adrénomégalie bilatérale et des masses multifocales étaient visibles dans les reins, l'estomac et le cæcum. Un lymphome a été diagnostiqué dans la masse cæcale à l'aide de la cytologie et de la PCR pour les réarrangements des récepteurs d'antigènes (PARR). Ce cas décrit une

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sténose acquise de la veine pulmonaire cliniquement significative secondaire à une masse médiastinale, qui était probablement responsable de la détresse respiratoire et de l'épanchement pleural.

Message clinique clé :

À notre connaissance, il s'agit du premier rapport de sténose acquise de la veine pulmonaire secondaire à une masse médiastinale chez un chat. La tomodensitométrie pourrait être utile pour confirmer la sténose de la veine pulmonaire et identifier son étiologie.

(Traduit par Dr Serge Messier)

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Pulmonary vein stenosis is characterized by narrowing of the pulmonary veins. It is relatively rare in human children and is often associated with various congenital heart diseases (1–3). The disease is rarer in adults and is associated with tumors or fibrosing mediastinitis (1). Only 1 veterinary study has reported congenital pulmonary vein stenosis with left-sided heart failure in a 5-month-old cat (4). No study has examined acquired pulmonary vein stenosis secondary to tumors in cats. Herein, we present a case of acquired pulmonary vein stenosis secondary to a mediastinal mass in a cat.

CASE DESCRIPTION

A 5-year-old spayed female munchkin cat weighing 2.12 kg was admitted to a local animal hospital with respiratory distress. The owner reported no history of trauma or recent surgery. Moreover, there had been no recent changes in the cat's environment.

Physical examination indicated normothermia (37.5°C) with an increased respiratory rate (66 rpm). The systolic blood pressure was unremarkable (140 mmHg). Feline serum amyloid A test revealed an equivocal result (5 μg/mL; reference range: normal = < 5 μg/mL, equivocal = 5 to 10 μg/mL, abnormal = > 10 μg/mL). An NT-proBNP test (SNAP Feline proBNP; IDEXX, Westbrook, Maine, USA) revealed 2 sample spots, indicating an abnormal result. The cat's feline immunodeficiency virus and feline leukemia virus statuses were unknown. Thoracic radiography (HF-525Plus Vet; Eco-ray, Nam-myeon, Korea) (55 kVP, 200 mA, 5 mAs) showed bilateral pleural effusions with a diffuse lung interstitial pattern (Figure 1). The heart was difficult to evaluate because of the pleural effusion. Thoracocentesis revealed a clear-colored effusion with a total solid level of 5.2 g/dL and TNCC of 2.11 × 10^3/μL, indicating a modified transudate. There was no evidence of inflammatory or neoplastic cells.

Point-of-care echocardiography (Aplio i700; Canon, Tokyo, Japan) revealed jet-like turbulent flows in the right and left atria, with a suspected adjacent heart tumor. Tricuspid and pulmonary valve regurgitation were not observed. Abdominal ultrasonography revealed left kidney renomegaly with masses at the gastric and ileocecocolic junctions. The left kidney was 47.8 mm in length (reference range: 30.0 to 43.0 mm) (5). The gastric mass was isoechoic with an oval shape, whereas the ileocecocolic junction mass was hypoechoic with an oval to rectangular shape. The sizes (W × H) of the gastric and ileocecocolic junction masses were 14.0 × 6.2 mm and 14.6 × 9.6 mm, respectively. The cat was administered furosemide (1 mg/kg, q8h, IV), dalteparin (100 μg/kg, q12h, SC), and spironolactone (1.5 mg/kg, q12h, PO) preemptively, due to concerns of congenital or acquired cardiac disease. The cat was then referred to Konkuk Veterinary Medical Center (Seoul, Korea) for further evaluation.

A thorough echocardiogram (V8; Samsung Medison, Seoul, Korea) was undertaken. An echogenic mass compressing the left atrium and a jet-like turbulent flow from the pulmonary veins into the left atrium were identified (Figure 2 A, B). The mass measured 13.9 × 18.6 mm (W × H) in size. A similar turbulent flow was observed in the right atrium (Figure 3 A). The velocities of the turbulent flows in the right and left atria were 1.42 and 1.63 m/s, respectively (Figure 3 A, B). The size of the left atrium (8.2 mm) and the left atrial to aortic root ratio (1.2) were within normal ranges. The thicknesses of the interventricular septum and left ventricular free walls were 4.6 and 3.7 mm, respectively (reference ranges: interventricular septum = 3.0 to 6.0 mm, left ventricular free wall = 2.5 to 6.0 mm) (6–8). The right ventricular outflow tract flow (peak velocity: 0.9 m/s) and diameter of the main pulmonary artery (MPA/Ao; 1.1) were unremarkable. No mitral, tricuspid, or pulmonary regurgitations were observed.

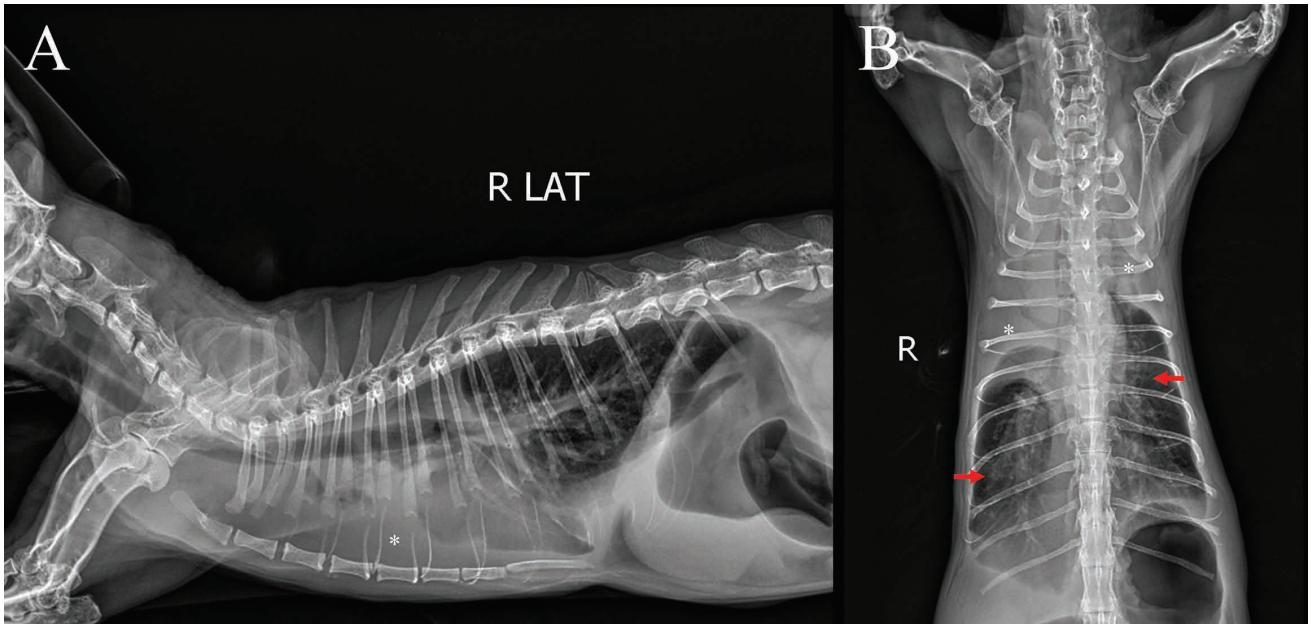

FIGURE 1. A and B — Right lateral and ventrodorsal thoracic radiographic images. Bilateral pleural effusion in the cranial thoracic cavity (asterisks) and diffuse lung interstitial patterns (red arrows) are noted.

Moreover, the diastolic function was unremarkable. In the echocardiogram, it was difficult to determine whether the tumor was an extrinsic mass compressing the heart or a mass originating from the heart. Therefore, computed tomography (CT) was used to evaluate the origin of the mass.

A CT scan was obtained using a 160-slice CT scanner (Aquilion Lightning; Canon). Scanning parameters were as follows: 120 kVP, 150 mA, matrix size: 512 × 512, rotation time: 0.75 s, and slice thickness: 1.0 mm. Anesthesia was induced with alfaxalone (5 mg/kg, IV) and maintained with 2% isoflurane gas. The cat was positioned in sternal recumbency under general anesthesia, and a nonionic contrast medium (Iohexol, 350 mg/mL; Omnipaque; GE Healthcare, Chicago, Illinois, USA) was administered using a power injector (CT9000 ADV; Mallinckrodt, Dublin, Ireland). Postcontrast images were acquired 70 s after contrast administration.

The CT scan revealed a diffuse homogeneous mediastinal mass compressing the dorsal border of the left atrium and surrounding the descending aorta, pulmonary arteries, and veins (Figure 4 A). The mediastinal mass measured 2.2 × 1.7 × 1.9 cm (L × W × H) in size. The distal caudal pulmonary veins were dilated, indicating pulmonary vein stenosis secondary to the mediastinal mass (Figure 4 B). The pulmonary vein ostia of the right cranial (RO), left cranial (LO), and caudodorsal (CDO) veins measured 5.5, 2.5, and 3.5 mm, respectively (Figure 4 C to E). The CDO was deemed stenotic owing to the compression of the mediastinal mass. Compression of the mass into the caudal vena cava was observed and considered the cause of the turbulent jet-like flow in the right atrium. The mass showed mild homogeneous enhancement (pre: 43 HU, post: 58 HU). Multifocal ground-glass opacities and patchy consolidation in the bilateral cranial lung fields were observed, along with pleural effusion and sternal lymphadenopathy. The lymph node measured 8.0 × 5.2 × 8.7 mm (L × W × H) in size. Homogeneous bilateral renal, gastric, and cecal masses were also detected.

The lengths of the left and right kidneys were 4.6 and 3.8 cm, respectively. The mass in the left kidney measured 4.2 × 3.5 × 2.5 cm (L × W × H) in size, whereas that in the right kidney measured 1.6 × 2.1 × 1.3 cm. The cecal and gastric masses measured 2.1 × 2.0 × 0.8 cm and 1.4 × 2.5 × 0.6 cm (L × W × H) in size, respectively. Bilateral adrenomegaly (left: 8.5 mm, right: 6.4 mm) was also observed (reference range: 3.0 to 4.8 mm) (9). Based on the clinical presentation, lymphoma was the leading differential diagnosis for the mediastinal and abdominal masses. The differential diagnoses for the lung included pulmonary metastases, interstitial edema, atelectasis, and consolidation.

The mediastinal mass was not aspirated due to concerns about damaging adjacent cardiac structures and pulmonary

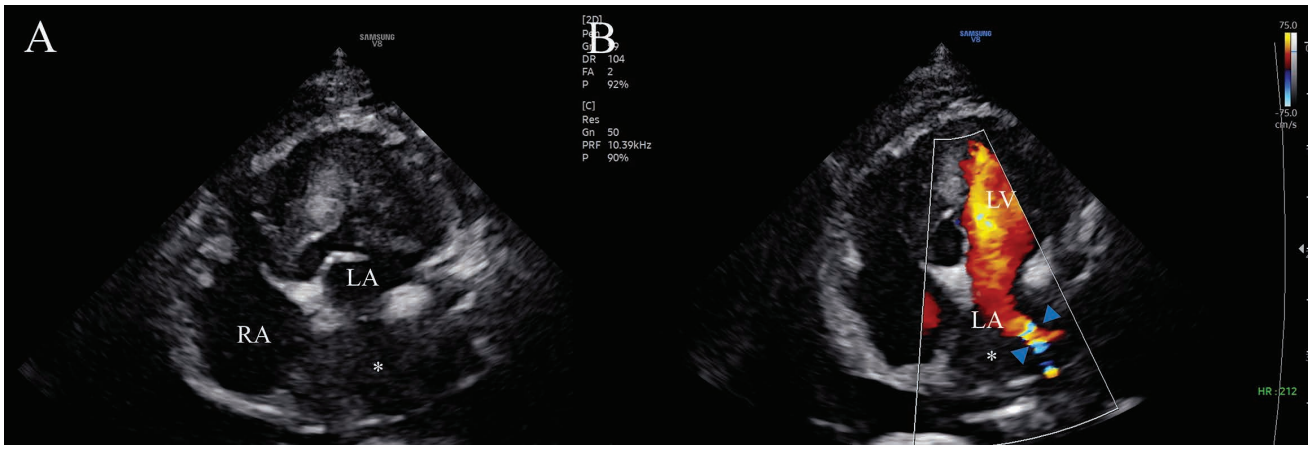

FIGURE 2. A and B — Echocardiographic images of the modified left apical 4-chamber view. An echogenic mass (asterisk) compressing the left atrium is noted. The turbulent jet-like flow into the left atrium (LA) (blue arrowheads) can be seen.
LV — Left ventricle; RA — Right atrium.

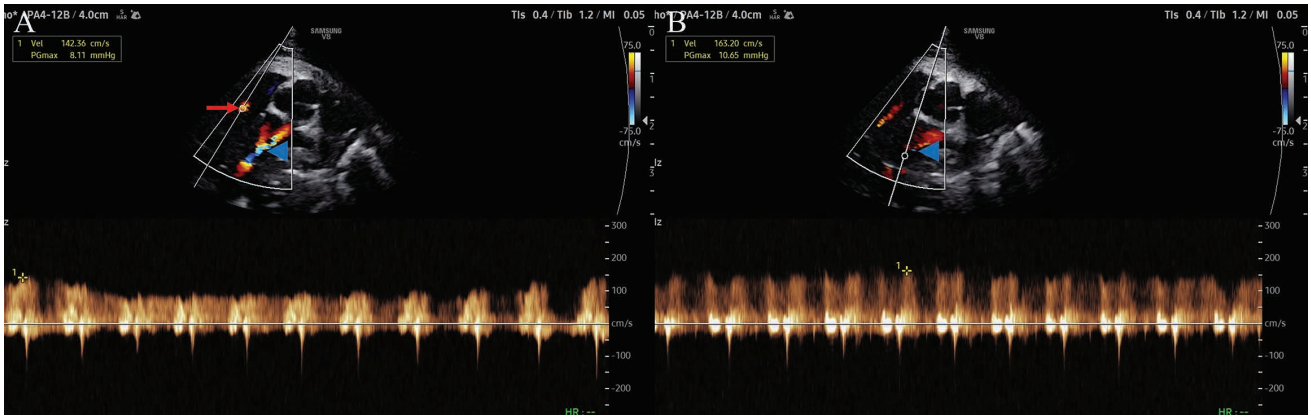

FIGURE 3. A and B — Continuous-wave Doppler images of the modified left atrial to aortic root (LA/Ao) view. The turbulent jet-like flow into the right atrium (red arrow) can be seen. The peak velocities of the turbulent flows in the right atrium (red arrow) and left atrium (blue arrowhead) were 1.42 and 1.63 m/s, respectively.

vessels. In addition, the gastric and renal masses were not aspirated because the liver tissue had to be penetrated to reach the gastric mass, and the bilateral renal masses were not as conspicuous with ultrasound as they were with CT imaging. Therefore, fine-needle aspiration of the cecal mass was completed. Cytologic examination revealed moderate-to-large-sized lymphocytes with high cellularity, mild anisocytosis, coarse chromatin patterns, marked nucleoli, and mitotic figures (Figure 5). Polymerase chain reaction for antigen receptor rearrangements indicated monoclonality in the immunoglobulin kappa deleting element. Gastric, renal, adrenal, and mediastinal masses, as well as sternal lymphadenopathy, were suspected to be indicative of lymphoma. The owner declined further treatment and the cat died 1 wk after the diagnosis. No necropsy was done.

DISCUSSION

To our knowledge, this is the first report of acquired pulmonary vein stenosis secondary to a mediastinal mass in veterinary medicine. In humans, patients with acquired pulmonary vein stenosis present with dyspnea, hemoptysis, and pleural effusion (10–12). The cat in this report had respiratory distress and pleural effusion. In the absence of other cardiac structural abnormalities, the pulmonary vein stenosis was likely responsible for the clinical signs and pleural effusion because of the high turbulent pulmonary vein inflow velocity. Pulmonary vein stenosis may be

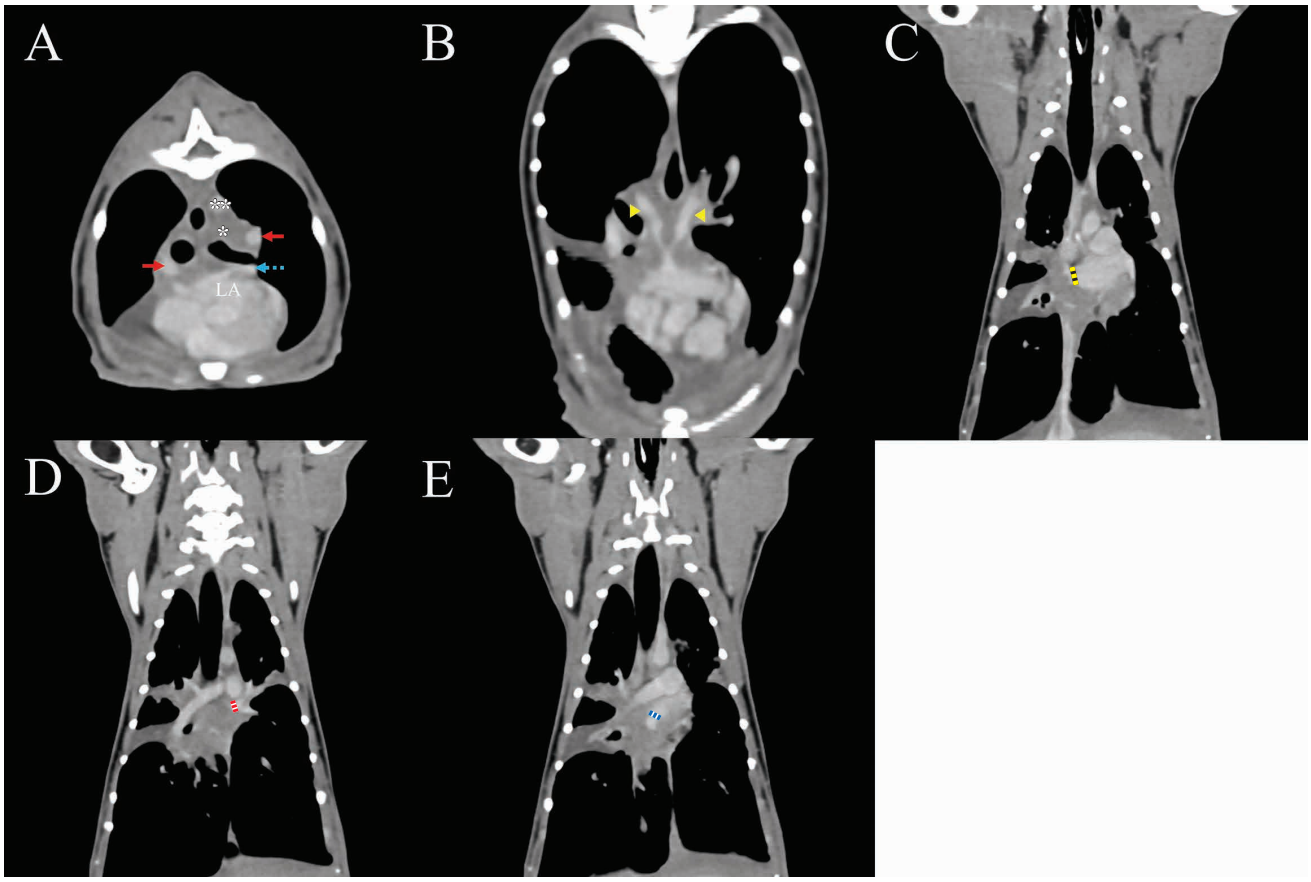

FIGURE 4. Postcontrast computed tomographic images in the transverse (A), oblique dorsal (B), and dorsal (C, D, E) planes. A diffuse, homogenous mediastinal mass (asterisk) compresses the dorsal border of the left atrium (LA) and surrounds the descending aorta (double asterisk), pulmonary arteries (red arrows), and pulmonary veins (blue dashed arrow) (A). The distal caudal pulmonary veins (yellow arrowheads) are dilated (B). The pulmonary vein ostia of the right cranial (yellow dashed line) (C), left cranial (red dashed line) (D), and caudodorsal (blue dashed line) (E) veins measured 5.5, 2.6, and 3.5 mm, respectively. The caudodorsal ostium is considered stenotic secondary to the mediastinal mass. All images are viewed in the soft-tissue window (window level = 60 HU; window width = 400 HU; slice thickness = 1.00 mm).

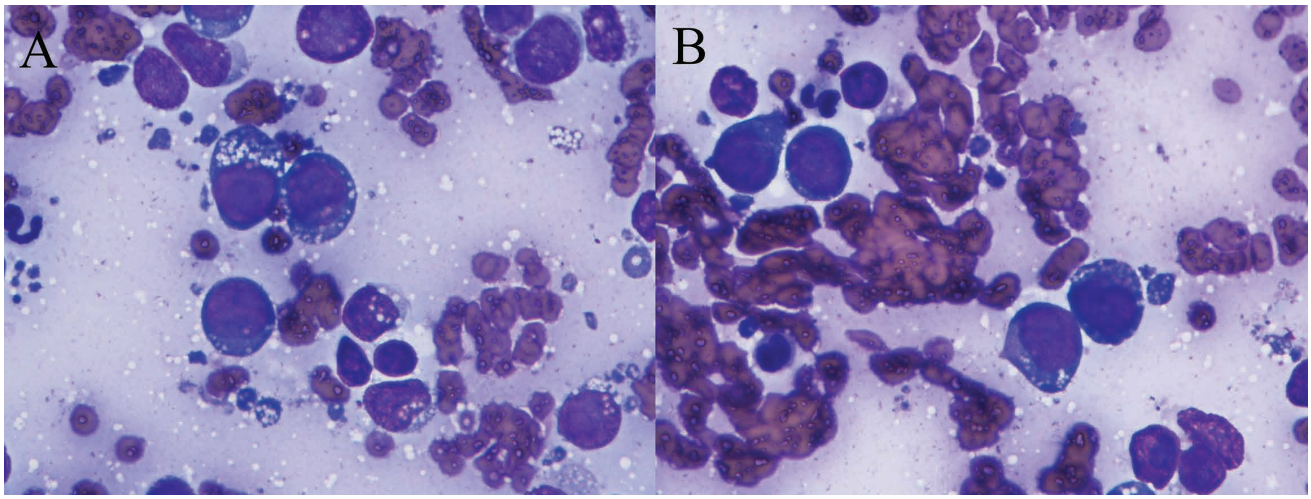

FIGURE 5. A and B — Cytologic examiantion of the aspirated cecal mass. Moderate-to-large-sized lymphocytes with high cellularity, mild anisocytosis, coarse chromatin patterns, marked nucleoli, and mitotic figures are seen. Diff-Quik staining, 1000× magnification (A, B).

clinically important in cases with high turbulent pulmonary vein velocity and functionally significant obstruction. The etiologies of acquired pulmonary vein stenosis in human adults include neoplastic and nonneoplastic masses and radiofrequency ablation for atrial fibrillation (2). Reported neoplastic etiologies include primary lung cancer, lymphoma, and mediastinal metastasis, whereas nonneoplastic etiologies include fibrosing mediastinitis and sarcoidosis (2,13–17). In a previous study in dogs, pulmonary vein stenosis was diagnosed following radiofrequency energy application within the pulmonary veins (18). The dogs exhibited pulmonary vein resistance, an increase in pulmonary capillary wedge pressure, and a decrease in cardiac output after radiofrequency energy application (18). A previous case report described congenital pulmonary vein stenosis in a 5-month-old cat (4). That cat exhibited an abnormal tubular structure in the left atrial base with a turbulent pulmonary vein velocity of 2.6 m/s. In contrast, the cat in the present report exhibited acquired pulmonary vein stenosis without such tubular structure in the left atrium. This cat also exhibited less severe pulmonary vein stenosis than the previously reported cat (4).

In humans, a turbulent pulmonary vein flow with a velocity of 1.1 to 1.5 m/s is diagnostic of pulmonary vein stenosis (3). If the velocity is $>$ 1.6 m/s, the stenosis may be a functionally significant obstruction (1). Cross-sectional imaging evaluates the absence or narrowing of pulmonary veins (2). Regional lung edema with increased lung opacity, lung fibrosis, and decreased lung perfusion can be observed (2). The cat described herein exhibited a pulmonary vein velocity of 1.63 m/s, consistent with functionally significant pulmonary vein stenosis. A previous study reported the RO, LO, and CDO diameters of the pulmonary veins of healthy cats using electrocardiography-gated cardiac CT (19). The ostium diameter varied during diastole and systole. The mean RO, LO, and CDO were 4.73 to 5.97, 1.97 to 2.52, and 7.13 to 8.03 mm, respectively (19). Although cardiac-gated CT was not used in our case and the cat's body weight was lower than the mean value reported in the abovementioned study, the CDO diameters were compared to confirm stenosis. The RO and LO diameters were similar; however, the CDO diameter was substantially narrower than that in healthy cats. In addition, dilation of the distal caudal pulmonary veins draining into the CDO indicated functional pulmonary vein stenosis.

The differential diagnoses for pulmonary vein stenosis include superior mitral valve stenosis (SMS) and *cor triatriatum sinister* (CTS). In SMS, the obstructive membrane is apical to the left atrial appendages with dilation of the basal chamber, where all pulmonary veins empty (20,21). In CTS, the obstructive membrane is basal to the left atrial appendage, which results in a non-dilated apical left atrium and a dilated basal left atrium (21). No obstructive membranes were observed in the present case; therefore, SMS and CTS were excluded.

Pulmonary hypertension can be caused by pulmonary vein stenosis, as noted in previous reports (1,4,22–25). However, no evidence of pulmonary hypertension was observed in the present case. Although subjective right atrial dilatation was observed, the cat did not exhibit tricuspid or pulmonary regurgitation. The absence of pulmonary hypertension could have been related to the severity of pulmonary vein stenosis because it was less severe in the previously reported cat (4). The pulmonary vein branches draining into the LO and RO may have compensated for the decreased return from the CDO. If the LO and RO were stenotic, pulmonary hypertension could have been evident.

Common mediastinal tumors in cats include thymomas, lymphomas, and ectopic thyroid tumors (26). A previous study demonstrated that 24% of mediastinal lymphomas had other tumor involvements (27). As the cecal mass was confirmed to be lymphoma, the mediastinal and other organ masses were presumed to be indicative of lymphoma. Chemotherapy protocols, such as CHOP or Wisconsin–Madison, could have helped reduce the size of the presuemd mediastinal lymphoma, thereby alleviating pulmonary vein stenosis (28). A simple corticosteroid palliation could have been done as well; however, treatment was not pursued because the owner declined.

This case report describes pulmonary vein stenosis secondary to a mediastinal mass in a cat. As in humans, acquired pulmonary vein stenosis in cats can occur secondary to neoplastic infiltration or extrinsic compression. When a turbulent jet-like flow from the pulmonary veins is observed in the left atrium, CT can be helpful in confirming pulmonary vein stenosis and identifying its etiology. CVJ

REFERENCES

1. Latson LA, Prieto LR. Congenital and acquired pulmonary vein stenosis. Circulation 2007;115:103–108.
2. Galizia M, Renapurkar R, Prieto L, *et al.* Radiologic review of acquired pulmonary vein stenosis in adults. Cardiovasc Diagn Ther 2018;8:387–398.
3. Pazos-López P, García-Rodríguez C, Guitián-González A, *et al.* Pulmonary vein stenosis: Etiology, diagnosis and management. World J Cardiol 2016;8:81–88.
4. Kriström K, Karlstam E, Nielsen T, Lagerqvist A, Dirven M. A case of congenital pulmonary vein stenosis with secondary

post-capillary pulmonary hypertension and left sided congestive heart failure in a cat. Vet Sci 2022;10:23.
5. Debruyn K, Haers H, Combes A, *et al*. Ultrasonography of the feline kidney: Technique, anatomy and changes associated with disease. J Feline Med Surg 2012;14:794–803.
6. Sisson DD, Knight DH, Helinski C, *et al*. Plasma taurine concentrations and M-mode echocardiographic measures in healthy cats and in cats with dilated cardiomyopathy. J Vet Intern Med 1991;5:232–238.
7. Jacobs G, Knight DH. M-mode echocardiographic measurements in nonanesthetized healthy cats: Effects of body weight, heart rate, and other variables. Am J Vet Res 1985;46: 1705–1711.
8. Abbott JA, MacLean HN. Two-dimensional echocardiographic assessment of the feline left atrium. J Vet Intern Med 2006;20:111–119.
9. Combes A, Pey P, Paepe D, *et al*. Ultrasonographic appearance of adrenal glands in healthy and sick cats. J Feline Med Surg 2013;15:445–457.
10. Yang S, Wang J, Li J, Huang K, Yang Y. Refractory pleural effusion as a rare complication of pulmonary vascular stenosis induced by fibrosing mediastinitis: A case report and literature review. J Int Med Res 2021;49:3000605211010073.
11. Braun S, Platzek I, Zöphel K, *et al*. Haemoptysis due to pulmonary venous stenosis. Eur Respir Rev 2014;23:170–179.
12. Fender EA, Widmer RJ, Hodge DO, *et al*. Severe pulmonary vein stenosis resulting from ablation for atrial fibrillation: Presentation, management, and clinical outcomes. Circulation 2016;134:1812–1821.
13. Stinson JM, Goodwin RA. Pulmonary vein obstruction by bronchogenic carcinoma. South Med J 1976;69:1482–1483.
14. Can MM. Severe pulmonary vein stenosis due to invasion of metastatic lung cancer. Anadolu Kardiyol Derg 2013;13:E17.
15. Webb WR. Lymphoma and lymphoproliferative disease. In: Webb WR, Higgins CB, eds. Thoracic Imaging: Pulmonary and Cardiovascular Radiology. 2nd ed. Philadelphia, Pennsylvania: Lippincott Williams & Wilkins, 2011:131.
16. Rossi SE, McAdams HP, Rosado-de-Christenson ML, Franks TJ, Galvin JR. Fibrosing mediastinitis. Radiographics 2001;21: 737–757.
17. Hamzeh I, Rashid A, Shaib F, Dawn B. Pulmonary vein stenosis due to a compressive malignant tumor detected by transesophageal echocardiography. Circulation 2011;123:349–350.
18. Taylor GW, Kay GN, Zheng X, Bishop S, Ideker RE. Pathological effects of extensive radiofrequency energy applications in the pulmonary veins in dogs. Circulation 2000;101:1736–1742.
19. Kim J, Kim DH, Kim K, Oh D, Choi J, Yoon J. Evaluation of the pulmonary vein ostia during the cardiac cycle using electrocardiography-gated cardiac computed tomography in cats. Front Vet Sci 2022;9:1013963.
20. Allen HD, Driscoll DJ, Shaddy RE, Feltes TF. Moss & Adams' Heart Disease in Infants, Children, and Adolescents: Including the Fetus and Young Adult. Philadelphia, Pennsylvania: Lippincott Williams & Wilkins, 2013.
21. Bharucha T, Spicer DE, Mohun TJ, Black D, Henry GW, Anderson RH. Cor triatriatum or divided atriums: Which approach provides the better understanding. Cardiol Young 2015;25:193–207.
22. Backes CH, Nealon E, Armstrong AK, *et al*. Pulmonary vein stenosis in infants: A systematic review, meta-analysis, and meta-regression. J Pediatr 2018;198:36–45.
23. Drossner DM, Kim DW, Maher KO, Mahle WT. Pulmonary vein stenosis: Prematurity and associated conditions. Pediatrics 2008;122:e656–e661.
24. Seale AN, Daubeney PEF. Pulmonary vein stenosis: Novel strategies for a challenging and resistant condition. J Thorac Cardiovasc Surg 2016;151:618–620.
25. Breinholt JP, Hawkins JA, Minich LA, *et al*. Pulmonary vein stenosis with normal connection: Associated cardiac abnormalities and variable outcome. Ann Thorac Surg 1999;68:164–168.
26. Larson MM. Mediastinal disease. In: Holland M, Hudson J, eds. Feline Diagnostic Imaging. 1st ed. New Jersey: John Wiley & Sons, 2020:293–303.
27. Fabrizio F, Calam AE, Dobson JM, *et al*. Feline mediastinal lymphoma: A retrospective study of signalment, retroviral status, response to chemotherapy and prognostic indicators. J Feline Med Surg 2014;16:637–644.
28. Collette SA, Allstadt SD, Chon EM, *et al*. Treatment of feline intermediate- to high-grade lymphoma with a modified university of Wisconsin-Madison protocol: 119 cases (2004–2012). Vet Comp Oncol 2016;14:s136–s146.

ARTICLE

Renata H. Pinho, Alexandra R. Robinson, Jessica Pang, Daniel S.J. Pang

Veterinary students do not need an elephantine memory: Effectiveness of an anesthetic pre-induction checklist

ABSTRACT

Background

Checklists are widely recognized as safety measures in both aviation and human medicine, effectively preventing omissions caused by memory failures.

Objective

To assess whether a pre-induction safety checklist completed by veterinary students during a spay/neuter laboratory minimized the number of incomplete pre-induction tasks.

Participants and procedure

Third-year veterinary students (N = 53) managed the anesthesia of dogs and cats admitted for spay/neuter surgery under supervision. The use of a pre-induction checklist was mandatory to ensure appropriate preparation before anesthesia induction. Differences in checklist completeness between the 1st and 2nd wk of the spay/neuter laboratory were compared using Fisher's exact test.

Results

Over 2 wk, 83 anesthesia procedures were completed. Use of the pre-induction checklist identified at least 1 omitted pre-induction task in 67.5% (56/83) of anesthesia procedures. The number of incomplete pre-induction tasks identified through use of the checklist decreased significantly from the 1st (82.9%, 34/41) to the 2nd (52.4%, 22/42) wk (odds ratio: 4.4, 95% CI: 1.7 to 11; $P = 0.0046$). The most frequently missed item was premeasuring the endotracheal tube insertion depth (42.2%, 35/83), followed by failure to leak-test the endotracheal tube cuffs and not having gauze available (15.7%, 13/83 for each). Finally, the checklist identified closed adjustable pressure-limiting valves in 4.8% (4/83) of cases.

Conclusion and clinical relevance

The pre-induction checklist was effective in ensuring that veterinary students completed relevant pre-induction tasks during a spay/neuter laboratory. Results suggested the pre-induction checklist was a valuable tool that improved patient safety and prevented life-threatening equipment errors such as closed adjustable pressure-limiting valves.

RÉSUMÉ

Les étudiants vétérinaires n'ont pas besoin d'une mémoire éléphantesque : efficacité d'une liste de contrôle de pré-induction anesthésique

Contexte

Les listes de contrôle sont largement reconnues comme des mesures de sécurité en aéronautique et en médecine humaine, empêchant efficacement les omissions causées par des défaillances de mémoire.

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Unpublished supplementary material (Figure S1) is available online from: Supplementary Materials.

Objectif

Évaluer si une liste de contrôle de sécurité de pré-induction complétée par des étudiants vétérinaires lors d'un laboratoire de stérilisation/castration minimise le nombre de tâches de pré-induction incomplètes.

Participants et procédure

Des étudiants vétérinaires de troisième année (N = 53) ont géré l'anesthésie de chiens et de chats admis pour une chirurgie de stérilisation/castration sous supervision. L'utilisation d'une liste de contrôle de pré-induction était obligatoire pour assurer une préparation appropriée avant l'induction de l'anesthésie. Les différences dans l'exhaustivité de la liste de contrôle entre la 1ère et la 2ème semaine du laboratoire de stérilisation/castration ont été comparées à l'aide du test exact de Fisher.

Résultats

Sur 2 semaines, 83 procédures d'anesthésie ont été réalisées. L'utilisation de la liste de contrôle de pré-induction a permis d'identifier au moins une tâche de pré-induction omise dans 67,5 % (56/83) des procédures d'anesthésie. Le nombre de tâches de pré-induction incomplètes identifiées grâce à l'utilisation de la liste de contrôle a diminué de manière significative de la 1ère (82,9 %, 34/41) à la 2ème (52,4 %, 22/42) semaine (rapport de cotes : 4,4, IC à 95 % : 1,7 à 11; P = 0,0046). L'élément le plus fréquemment oublié était la pré-mesure de la profondeur d'insertion du tube endotrachéal (42,2 %, 35/83), suivi par l'absence de test d'étanchéité des ballonnets du tube endotrachéal et le manque de gaze disponible (15,7 %, 13/83 pour chaque). Enfin, la liste de contrôle a identifié des valves de limitation de pression réglables fermées dans 4,8 % (4/83) des cas.

Conclusion et pertinence clinique

La liste de contrôle de pré-induction s'est avérée efficace pour garantir que les étudiants vétérinaires ont effectué les tâches de pré-induction pertinentes lors d'un laboratoire de stérilisation. Les résultats ont suggéré que la liste de contrôle de pré-induction était un outil précieux qui a amélioré la sécurité des patients et a évité les erreurs d'équipement potentiellement mortelles telles que la fermeture des valves de limitation de pression réglables.

(Traduit par Dr Serge Messier)

Can Vet J 2025;66:409–416

INTRODUCTION

Checklists are simple, inexpensive, and effective tools that can assist in reducing mental workload and memory failures, especially in situations associated with multiple steps (1). They have also been shown to improve communication and teamwork within the workplace (2). Checklist designs vary depending on their intended use. For example, "read-do" checklists are designed to provide users with guidance to complete a task when there may be less familiarity with task requirements, whereas "do-confirm" checklists are designed to confirm that tasks with which users are familiar have been completed (3).

Incorporating checklists into healthcare was inspired by evidence of their value in aviation. There is a heavy reliance on checklists in the aviation industry to ensure safe takeoff and landing practices and to manage critical events (4). Patient-safety strategies in human medicine now routinely include the use of checklists at critical points in the care pathway (5,6). For example, infections associated with central venous catheters in humans are expensive to treat and are associated with a high incidence of morbidity and mortality (6). Checklist use in human intensive care units was shown to improve healthcare providers' compliance completing basic tasks, such as hand washing and aseptic technique, before insertion of central venous catheters. The effect of checklist implementation was a reduction in catheter infection, from a median of 2.7 per 1000 catheter days to 0 within 3 mo ($P \leq 0.002$), and this improvement was sustained over several years (6).

In 2008, the World Health Organization (WHO) published guidelines that recommended the use of a surgical checklist to improve safety practices in human operating rooms (ORs) (7). Based on those guidelines, a 19-item "do-confirm" surgical safety checklist was designed to be completed in 3 phases: sign in, time out, and sign out.

"Sign in" occurs before anesthetic induction and consists of, *e.g.,* confirmation of patient identity, surgical procedure, and site; and confirming preparation of anesthetic equipment. "Time out" occurs before incision and "sign out" occurs before the patient is transported out of the OR (5). The WHO surgical safety checklist was tested in a global quality improvement study with the aims of improving patient care and reducing surgical complications and mortality (5). Significant reductions in postoperative complications (11 to 7%; $P < 0.001$) and mortality (1.5 to 0.8%; $P = 0.003$) were achieved, leading to widespread adoption of the checklist internationally (5).

In veterinary medicine, there is evidence that the use of a checklist reduces surgical site infection rates (8–10), ensures antimicrobial administration at the correct times (11), and improves the dissemination of important postoperative care information (12). In veterinary anesthesia, the incorporation of 2 checkboxes in the anesthesia record for "OR check" and "intubation confirmation" reduced the proportion of incidents of closed adjustable pressure-limiting (APL) valves and esophageal intubation by 75% (13).

Preparation before anesthesia includes multiple steps. These include setting up and testing the anesthetic machine and breathing circuit, ensuring appropriate intubation supplies are available [*e.g.,* endotracheal tubes (ETTs), laryngoscope, and material to secure the tube in position] and preparation of necessary medications. Mistakes or omissions made at any stage of preparation have the potential to compromise patient safety. Furthermore, the anesthetist is part of a multidisciplinary team, and fostering good communication and teamwork are crucial for avoiding mistakes and omissions in the perianesthetic period.

In human anesthesia, use of a checklist during anesthetic machine preparation was associated with a reduction in perioperative morbidity and mortality (14). For human anesthesia residents, use of an anesthesia checklist improved patient safety by increasing the completeness of safety tasks carried out before anesthetic induction (15).

Despite anecdotal widespread use of checklists in veterinary teaching hospitals, the benefits of pre-induction checklists during veterinary student training have not been reported. Using a "do-confirm" checklist designed to be applied immediately before induction of general anesthesia, the aim of this study was to assess the number of incomplete pre-induction tasks identified while using the pre-induction checklist during a live-animal spay/neuter teaching laboratory.

MATERIALS AND METHODS

The study was approved by the University of Calgary (Calgary, Alberta) Human Ethics Review Board (REB23-1535) and was undertaken during a 3rd-year veterinary student live-animal spay/neuter laboratory. A deferred consent process was used: Before the study began, students were informed that data from anesthetic records would be collected for research and collected data would not be used as part of any performance assessment. Students were told that study details would be disclosed at the end of the spay/neuter laboratory, to avoid awareness of study details affecting behavior (16). In addition, students were told that all collected data would be anonymized before analysis. At the end of the spay/neuter laboratory, the primary researcher disclosed all study information and written consent was requested from each student. Data from students who did not provide informed consent were excluded from analysis. Students who provided consent received a gift card.

The study was conducted over the course of 6 d — 3 d during each of 2 consecutive weeks. Third-year veterinary students (degree duration is 4 y) shared responsibility for the anesthetic and surgical management of cats and dogs admitted for sterilization. Supervision was provided by veterinary anesthetists ($n = 3$) and surgeons ($n = 3$). Students were organized into teams of 3 to 4, with 1 to 2 students responsible for anesthesia and 1 to 2 students responsible for surgery, and students rotated between roles over the course of the laboratory. Each team of student anesthetists was accompanied by a registered animal health technologist dedicated to providing anesthesia support. Over the course of the 2 wk, each student served as primary anesthetist for at least 2 anesthetic procedures.

Anesthesia students had primary responsibility for preparing their animal (clinical examination, anesthetic protocol preparation, premedication, intravenous catheter placement) and their anesthesia station (anesthesia machine check, supplies for intravenous catheter and orotracheal intubation).

Study design

The University of Calgary's Faculty of Veterinary Medicine (UCVM) Pre-induction Checklist was created for local use and is an adaptation of the Association of Veterinary Anaesthetists and WHO surgery checklists (17) (Table 1; Figure S1, available online from: Supplementary Materials).

Students received a lecture on anesthetic errors and safety from a Board-certified anesthesiologist ~10 wk

TABLE 1. University of Calgary's (Calgary, Alberta) Faculty of Veterinary Medicine (UCVM) Pre-induction Checklist items with expectations and relevance for each.

Checklist item	Task expected/material prepared and available	Relevance of the item
Confirm verbally with all team members: patient ID, owner consent, procedure and site	Verbal communication between anesthesia team members	Ensures correct patient will be anesthetized, avoiding risk of wrong-patient errors (18) Also ensures correct procedure and location (19)
IV access patent	IV catheter flushed with saline and patency confirmed	Reduces risk of extravascular anesthetic drug administration
Medications prepared	All medications to be used during anesthesia prepared and available for use	Ensures planned medications are prepared and available
Volumes confirmed	Volumes of prepared medications checked against written anesthetic plan	Reduces risk of dosing errors (20)
Labelled	All syringes labelled with content	Reduces risk of medication errors
ETTs	3 different sizes of ETTs	Reduces risk of delaying intubation due to missing ETTs
Cuffs tested	All ETT cuffs tested (inflated) and subsequently deflated	Ensures ability to seal the airway
Tube tie	A tube tie of appropriate length to hold the ETT in place	Reduces risk of ETT displacement leading to airway trauma, accidental extubation, endobronchial intubation
Laryngoscope	A laryngoscope with blade and light tested	Facilitates and decreases time of endotracheal intubation, and reduces risk of esophageal intubation (21)
Lube	A blob of lubrifying gel to be placed on the distal end of the ETT	Facilitates intubation and an airtight seal
Gauze	Gauze to assist with exteriorizing the tongue during intubation	Facilitates intubation
ETT insertion depth premeasured	Length of ETT to be inserted identified by measuring distance from incisors to point of shoulder (acromion)	Reduces risk of endobronchial intubation (21)
Anesthetic machine checked today	Full anesthetic machine check completed once daily	Ensures proper functioning of anesthetic machine (14,22)
Breathing system leak-tested for this case	Abbreviated preanesthetic machine check	Ensures proper function of breathing system
APL valve open	Self-explanatory	Reduces risk of adverse events due to obstruction of gas evacuation (13,17)
Oxygen pipeline supply connected	Self-explanatory	Ensures appropriate provision of oxygen
Adequate oxygen in E cylinder	Volume of E cylinder (secondary oxygen supply) confirmed by visual inspection of pressure gauge; minimum acceptable volume: ~50%	Ensures an appropriate supply of backup oxygen should the primary (pipeline) supply fail
Distribution of tasks between team members	Tasks to be distributed: injection of induction agent, intubation, placing monitoring equipment, preparing and connecting crystalloid, recording information on anesthetic record	Confirms each member is aware of and prepared for their assigned task, thus ensuring prompt patient care
Patient/procedural risks identified and communicated	Patient comorbidities (*e.g.*, cardiac disease) and procedural risks (*e.g.*, hemorrhage) communicated	Ensures all team members are aware of concerns and potential complications
Emergency interventions available	Team members aware of location of emergency supplies (crash cart)	Ensures access to emergency supplies without delay

APL — Adjustable pressure-limiting; ETT — Endotracheal tube; IV — Intravenous.

TABLE 2. Incidences of incomplete pre-induction checklist items during 3rd-year veterinary student spay/neuter laboratory.

Checklist item	Incidence of incomplete checklist items (1st wk)	Incidence of incomplete checklist items (2nd wk)	Incidence of incomplete checklist items (total)
ETT depth premeasured	46.3% (19/41)	38.1% (16/42)	42.2% (35/83)
ETT cuffs tested	26.8% (11/41)	4.8% (2/42)	15.7% (13/83)
Gauze	19.5% (8/41)	11.9% (5/42)	15.7% (13/83)
ETT tie	17.1% (7/41)	9.5% (4/42)	13.3% (11/83)
Emergency interventions	24.4% (10/41)	2.4% (1/42)	13.3% (11/83)
Medication volumes confirmed	4.9% (2/41)	4.8% (2/42)	4.8% (4/83)
ETT (3 sizes)	7.3% (3/41)	2.4% (1/42)	4.8% (4/83)
Lube	9.8% (4/41)	0% (0/42)	4.8% (4/83)
APL valve	4.9% (2/41)	4.8% (2/42)	4.8% (4/83)
Adequate oxygen	2.4% (1/41)	7.1% (3/42)	4.8% (4/83)
Distribution of tasks	7.3% (3/41)	2.4% (1/42)	4.8% (4/83)
Laryngoscope	7.3% (3/41)	0% (0/42)	3.6% (3/83)
Confirm verbally	4.9% (2/41)	0% (0/42)	2.4% (2/83)
Medications labelled	2.4% (1/41)	2.4% (1/42)	2.4% (2/83)
IV access patent	0% (0/41)	2.4% (1/42)	1.2% (1/83)
Anesthetic machine tested	2.4% (1/41)	0% (0/42)	1.2% (1/83)
Circuit leak-tested	0% (0/41)	2.4% (1/42)	1.2% (1/83)
Risks identified/communicated	2.4% (1/41)	0% (0/42)	1.2% (1/83)
Medications prepared	0% (0/41)	0% (0/42)	0% (0/83)
Oxygen connected	0% (0/41)	0% (0/42)	0% (0/83)

APL — Adjustable pressure-limiting; ETT — Endotracheal tube; IV — Intravenous.

before the spay/neuter laboratory, as part of their anesthesia course. The lecture included instruction on the use of checklists in the perianesthetic period. The anesthesia course also included an anesthetic equipment laboratory in which training was provided on all items included on the pre-induction checklist. During the laboratory, students were shown how to complete a preanesthetic machine check using printed instructions. Students were instructed that there was no expectation that the instructions should be memorized and that the instructions would be provided with each anesthesia machine at every subsequent laboratory. Instruction in preparing supplies for intubation was also provided and a copy of the UCVM Pre-induction Checklist was distributed to each student at the end of the laboratory. Approximately 3 wk before the spay/neuter laboratory, students used the pre-induction checklist during a live-animal dentistry and anesthesia laboratory.

In the spay/neuter laboratory, students responsible for anesthesia worked in teams of 2 to 3 students alongside a dedicated animal health technician. Students were instructed to use the pre-induction checklist, as a "do-confirm" checklist, immediately before inducing general anesthesia. This involved reading each checklist item out loud and confirming with the team that the task was complete. Items were checked off once confirmed as complete. Independent of the student-technician team, checklist use was observed by 1 of 3 supervising anesthetists and any items omitted during application of the checklist were recorded as incomplete items for later analysis. Students were required to correct any items identified as incomplete before continuing to the next item on the checklist, so that all items were completed before anesthesia was induced.

Data analysis

The number of anesthesia procedures with at least 1 incomplete item and the total number of incomplete items were analyzed descriptively to generate percentages of the total number of checklists and items, respectively.

Differences in item completeness between the 1st and 2nd wk of the spay/neuter laboratory were compared using Fisher's exact test (GraphPad Prism software version 10.2.0 for macOS; GraphPad Software, La Jolla, California, USA). P-values < 0.05 were considered statistically significant. All data supporting the results are available in a data repository (Pang D, 2024: https://doi.org/10.7910/DVN/O6CNKD).

RESULTS

During the 6 d of the spay/neuter laboratory, 83 anesthesia procedures were completed (week 1, $n = 41$; week 2, $n = 42$) by the 3rd-year veterinary students (N = 53). All students provided written, informed consent at the end of the laboratory, allowing analysis of all 83 checklists (1 per procedure), resulting in 1660 checklist items (83 checklists $\times$ 20 items per checklist).

Over the 6 d, 67.5% (56/83) of anesthesia procedures had at least 1 incomplete item during application of the checklist. Comparing the 3 operating days of the 1st wk to those of the 2nd wk, the number of anesthesia procedures with at least 1 omitted item decreased significantly between the 1st (82.9%, 34/41) and 2nd (52.4%, 22/42) wk (odds ratio: 4.4, 95% CI: 1.7 to 11; $P = 0.0046$). Similarly, the number of incomplete individual items decreased between the 1st (9.6%, 79/820) and 2nd (4.8%, 40/840) wk (odds ratio: 2.1, 95% CI: 1.5 to 3.1; $P = 0.0001$).

Premeasuring the ETT insertion depth was the most frequently incomplete item, followed by leak-testing the ETT cuffs and having gauze (for tongue traction), an ETT tie, and emergency interventions available. Only medication preparation and pipeline oxygen connection were complete in all 83 procedures (Table 2).

DISCUSSION

Use of the pre-induction checklist reduced the number of incomplete pre-induction tasks in a 3rd-year veterinary student spay/neuter laboratory over the 6-day study period. These omitted tasks may not have been identified without the checklist and, consequently, may have been left incomplete before induction of anesthesia. Some essential tasks, such as preparing sedative and anesthetic medications and connecting pipeline oxygen supplies, were consistently completed. The use of the checklist potentially prevented adverse events; *e.g.,* by identifying the 4 APL valves left closed before induction. In addition, task completion improved over time.

The 4 most omitted items on the pre-induction checklist were all related to orotracheal intubation. The act of orotracheal intubation in dogs and cats must be completed correctly to achieve the intended goal of securing an airway. When done incorrectly, several complications, including unintended endobronchial intubation and an airway leak, can occur (21). These complications increase the risk of hypoxemia and local environmental pollution and aspiration pneumonia, respectively. Inadvertent endobronchial intubation is easily avoided by premeasuring the insertion depth of the ETT and ensuring it is properly secured once placed. Ensuring a leak-free seal around the ETT cuff requires testing of cuffs before intubation and confirming the absence of a leak after intubation. The latter was not included in the checklist as it is completed after induction. Leaks from and around the ETT cuff during anesthesia is the most reported complication of endotracheal intubation in dogs (21). With ETT reuse, commonly practiced in veterinary medicine, cuff material deteriorates over repeated cycles of use and cleaning, leading to leaks occurring at the cuff, pilot tube, or inflation valve. Therefore, it is considered best practice to test cuffs for leaks before use. In addition, cuff testing checks for ETT manufacturing defects.

In addition to complications related to the ETT and intubation, a serious and relatively frequent safety incident in veterinary anesthesia is an unintended closed APL valve (or pop-off valve) (13,17,23). The purpose of this valve is to allow excess gas to be scavenged from the anesthetic breathing system. Consequently, if the valve is left closed during routine use, pressure rapidly builds up in the system and in the patient's airways, ultimately resulting in cardiovascular collapse and death.

In an investigation into postmortem lesions associated with perianesthetic mortality in animals, anesthetic complications were identified as the cause of death in 3 out of 221 cases. Two of those cases were associated with closed APL valve accidents (24). In human anesthesia, a study of perianesthetic morbidity and mortality identified a decreased risk of coma or death during or 24 h after anesthesia when equipment underwent a check-out procedure that included a checklist (14). Similarly, in veterinary anesthesia, standardizing the anesthetic machine check-out procedure reduced the incidence of closed APL valves from 1 to 0.2% (13). Using the pre-induction checklist in the present study identified an unintended closed APL valve in ~5% of cases, demonstrating a benefit of checklist use for patient safety.

To be effective, checklists must be properly executed; this includes using them at the right time (before induction of anesthesia, for the checklist used here) and completing all listed tasks. Failure to apply checklists appropriately risks compromising their effectiveness. For example, transient hypoxemia occurred in a cat when a pre-induction checklist was not fully completed (17).

Some checklist items (*e.g.,* APL valve open, emergency interventions available) have a more explicit relationship with safety than others (*e.g.,* gauze, tube tie, and laryngoscope available to assist intubation). However, the inclusion on the checklist of items not directly related to patient safety still serves an important purpose by avoiding prolonging intubation by ensuring all necessary supplies are readily available. This fulfills an important goal of checklists: to avoid reliance on memory. This has further value in a scenario in which trainees or new staff members are present or when items are easily forgotten or their locations are unknown. A prolonged time to intubation may increase patient risk, such as with a longer period of apnea

(a common complication at induction) or increased risk of aspiration of gastric contents in some animal populations (*e.g.,* gastric and small-intestinal foreign bodies, pregnancy).

The use of the pre-induction checklist facilitated the work of the supervising anesthetists. The observed improvement in task completion during the 2nd wk of the spay/neuter laboratory can be explained by increased familiarity with, or perhaps memorization of, the checklist items by the students. Regardless of the reason for increased familiarity, a checklist item was omitted in more than 50% of cases in the 2nd wk, which demonstrates the ongoing importance of the checklist as a cognitive aid.

This study had several limitations. It did not assess the performance of students who did not use a checklist. Because the checklist used has been integrated into the curriculum and was part of our program's standard of care for delivering anesthesia, it was decided when designing the study that not using the checklist would represent a compromise in both patient care and potentially student education within the context of a busy teaching laboratory. Similarly, as checklists were used for all anesthetic procedures, the study was not designed to assess anesthetic complications or patient outcomes related to checklist-item completeness. However, as some items were closely related to risk of harm (*e.g.,* closed APL valve), the results suggest that, by using the checklist, harm was likely avoided. Due to the limitations of the study design (*i.e.,* all students used the checklist), it is possible that a closed APL valve may have been identified without the use of a checklist. However, the opportunities to do so before dangerous overinflation of the rebreathing bag occurred were limited.

Some known benefits of checklist use (*e.g.,* communication and teamwork) were not evaluated in this study. Therefore, additional benefits may have been provided. Finally, the population assessed in the present study comprised veterinary students who had received training and practice in checklist use before participating in the laboratory. The observed results could vary in a different population, such as untrained students or veterinary technicians.

In conclusion, using a pre-induction checklist effectively promoted the completeness of relevant pre-induction tasks that, if omitted, had the potential to jeopardize patient safety. Using a pre-induction checklist can avoid unintended harm around the time of anesthetic induction during live-animal spay/neuter laboratories with veterinary students and can reduce the reliance on memory to complete all necessary tasks before inducing anesthesia. The latter benefit simplifies the process of anesthetic induction for students.

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REFERENCES

1. Winters BD, Gurses AP, Lehmann H, Sexton JB, Rampersad C, Pronovost PJ. Clinical review — Checklists: Translating evidence into practice. Crit Care 2009;13:210.
2. Russ S, Rout S, Sevdalis N, Moorthy K, Darzi A, Vincent C. Do safety checklists improve teamwork and communication in the operating room? A systematic review. Ann Surg 2013;258: 856–871.
3. Alfred M, Barg-Walkow LH, Keebler JR, Chaparro A. Checking all the boxes: A checklist for when and how to use checklists effectively. BMJ Qual Saf 2024;33:673—681.
4. Jelacic S, Bowdle A, Nair BG, Nair AA, Edwards M, Boorman DJ. Lessons from aviation safety: Pilot monitoring, the sterile flight deck rule, and aviation-style computerised checklists in the operating room. Br J Anaesth 2023;131: 796–801.
5. Haynes AB, Weiser TG, Berry WR, *et al.* A surgical safety checklist to reduce morbidity and mortality in a global population. N Engl J Med 2009;360:491–499.
6. Pronovost P, Needham D, Berenholtz S, *et al.* An intervention to decrease catheter-related bloodstream infections in the ICU. N Engl J Med 2006;355:2725–2732.
7. World Alliance for Patient Safety. WHO Guidelines for Safe Surgery. Geneva, Switzerland: World Health Organization, 2008.
8. Bergström A, Dimopoulou M, Eldh M. Reduction of surgical complications in dogs and cats by the use of a surgical safety checklist: Surgical checklist to reduce postoperative complications. Vet Surg 2016;45:571–576.
9. Launcelott ZA, Lustgarten J, Sung J, Samuels S, Davis S, Davis GJ. Effects of a surgical checklist on decreasing incisional infections following foreign body removal from the gastrointestinal tract in dogs. Can Vet J 2019;60:67–72.
10. Thieman Mankin KM, Jeffery ND, Kerwin SC. The impact of a surgical checklist on surgical outcomes in an academic institution. Vet Surg 2021;50:848–857.
11. Menoud G, Axiak Flammer S, Spadavecchia C, Raillard M. Development and implementation of a perianesthetic safety checklist in a veterinary university small animal teaching hospital. Front Vet Sci 2018;5:60.
12. Ruch-Gallie R, Weir H, Kogan LR. Impact of checklist use on wellness and post-elective surgery appointments in a veterinary teaching hospital. J Vet Med Educ 2017;44:364–368.
13. Hofmeister EH, Quandt J, Braun C, Shepard M. Development, implementation and impact of simple patient safety interventions in a university teaching hospital. Vet Anaesth Analg 2014; 41:243–248.
14. Arbous MS, Meursing AEE, Van Kleef JW, *et al.* Impact of anesthesia management characteristics on severe morbidity and mortality. Anesthesiology 2005;102:257–268.
15. Beck S, Reich C, Krause D, *et al.* For beginners in anaesthesia, self-training with an audiovisual checklist improves safety during anaesthesia induction: A randomised, controlled two-centre study. Eur J Anaesthesiol 2018;35:527–533.
16. McCambridge J, Witton J, Elbourne DR. Systematic review of the Hawthorne effect: New concepts are needed to study research participation effects. J Clin Epidemiol 2014;67: 267–277.
17. Pang J, Yates E, Pang DSJ. A closed 'pop-off' valve and patient safety incident: A human factors approach to understanding error. Vet Rec Case Rep 2021;9:e189.

18. Chassin MR, Becher EC. The wrong patient. Ann Intern Med 2002;136:826.
19. Edmonds C, Liguori G, Stanton M. Two cases of a wrong-site peripheral nerve block and a process to prevent this complication. Reg Anesth Pain Med 2005;30:99–103.
20. Pinho RH, Tscheng D, Cheema JS, Pang DSJ. Incidence and type of voluntary reported perianesthetic medication errors in community veterinary clinics in Calgary, Canada. Am J Vet Res 2024;85:ajvr.24.04.0119.
21. Klonner ME, Springer S, Braun C. Complications secondary to endotracheal intubation in dogs and cats: A questionnaire-based survey among veterinary anaesthesiologists. Vet Anaesth Analg 2023;50:220–229.
22. Marchiori JJM, Prebble MJ, Pang DSJ. A prospective survey of veterinary anesthesia equipment in Alberta, Canada, using a standardized checkout procedure. Can Vet J Rev 2023;64:159–166.
23. Ludders J, McMillan M. Errors in Veterinary Anesthesia. Ames, Iowa: Wiley Blackwell, 2017.
24. DeLay J. Perianesthetic mortality in domestic animals: A retrospective study of postmortem lesions and review of autopsy procedures. Vet Pathol 2016;53:1078–1086.

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Nous sommes toujours à la recherche de photos pour la couverture de *La RVC*. Nous encourageons même la famille et les amis des vétérinaires à nous faire parvenir des photos d'animaux. Même si les caméras numériques facilitent la soumission de photos, il est nécessaire de respecter certains critères pour que nous puissions les utiliser pour les couvertures. Voici quelques paramètres à respecter :

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ARTICLE

J. Scott Weese

Canine contact networks: A survey-based study of dog contacts and activities

ABSTRACT

Background

Contact networks influence infectious disease risk and transmission.

Objective

To evaluate dog contact numbers and exposure to higher-risk activities (*e.g.,* day care) in dogs from Canada and the United States.

Animals and procedure

Dog owners were recruited to participate in a survey about their dogs' contacts with other dogs and participation in selected activities.

Results

Data were obtained for 1058 dogs: 829 from a commercial survey group and 329 from a social media recruitment group. Median numbers of dog contacts in the preceding 24 h, 7 d, and 14 d were 1, 3, and 4, respectively, in the social media group, and 2 for each time period in the commercial survey group. In the commercial survey group, dogs had a median of 1 type of queried contact (*e.g.,* day care) (range: 0 to 10). In the social media group, 16% dogs had no reported contacts with any other dogs in the preceding 24 h, whereas 5.2% had no contacts in the preceding week and 3.0% had no contacts over the preceding 2 wk.

Conclusion and clinical relevance

Dog contact activities are variable and influenced by many factors. Understanding this is important for disease modeling, development of infection-control interventions, and assessment of risk.

RÉSUMÉ

Réseaux de contacts canins : une étude basée sur une enquête sur les contacts et les activités des chiens

Contexte

Les réseaux de contacts influencent le risque et la transmission des maladies infectieuses.

Objectif

Évaluer le nombre de contacts canins et l'exposition à des activités à risque élevé (p. ex., garderie) chez les chiens du Canada et des États-Unis.

Animaux et procédure

Les propriétaires de chiens ont été recrutés pour participer à une enquête sur les contacts de leurs chiens avec d'autres chiens et leur participation à des activités sélectionnées.

Résultats

Les données ont été obtenues pour 1058 chiens : 829 d'un groupe d'enquête commerciale et 329 d'un groupe de recrutement sur les réseaux sociaux. Le nombre médian de contacts avec des chiens au cours

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des 24 heures, 7 jours et 14 jours précédents était respectivement de 1, 3 et 4 dans le groupe des réseaux sociaux et de 2 pour chaque période dans le groupe d'enquête commerciale. Dans le groupe d'enquête commerciale, les chiens avaient une médiane de 1 type de contact interrogé (p. ex., garderie) (plage : 0 à 10). Dans le groupe des réseaux sociaux, 16 % des chiens n'avaient signalé aucun contact avec d'autres chiens au cours des 24 heures précédentes, tandis que 5,2 % n'avaient eu aucun contact au cours de la semaine précédente et 3,0 % n'avaient eu aucun contact au cours des 2 semaines précédentes.

Conclusion et pertinence clinique

Les activités de contact des chiens sont variables et influencées par de nombreux facteurs. Il est important de comprendre cela pour la modélisation des maladies, le développement d'interventions de contrôle des infections et l'évaluation des risques.

(Traduit par Dr Serge Messier)

Can Vet J 2025;66:417–424

INTRODUCTION

Contact networks represent a quantitative assessment of interactions between pairs of individuals, including position, proximity, and nature of the contact (*e.g.,* frequency or duration) (1). These networks are important to understand for evaluation of infectious disease risk and transmission. They represent the number and types of contacts that an individual has with other individuals. Defining contact networks can be challenging (2), but an understanding of contacts is required for accurate disease modelling, intervention development, and risk communication.

Contact networks have received limited attention in veterinary medicine, despite the importance of infectious diseases. Most of the study of dogs has focused on free-ranging dogs (3–8), usually in low- and middle-income countries and with a focus on rabies virus exposure. Study of household pets has been very limited. A study of 214 dog-owning households in the United Kingdom investigated walking behaviors, with a focus on areas where dogs were walked, evaluating frequency and types of exposures (9). Yet little information is available about the number of dog-dog contacts that occur in daily life and the frequency of exposure to situations that are higher-risk for pathogen exposure, such as boarding, day care, and other activities that may increase the risk of new dog-dog contacts.

Given the paucity of data and the continued problems with endemic and emerging infectious diseases in dogs, the objective of this study was to evaluate dog contact numbers and activities among dogs from Canada and the United States.

MATERIALS AND METHODS

An online survey of dog owners was conducted. Individuals who owned a dog, were ≥ 18 y of age, and could complete the survey in English were eligible for inclusion. There were 2 recruitment methods. One group was a convenience sample recruited through social media outreach *via* X (formerly Twitter), Facebook, and http://www.wormsandgermsblog.com. Another group of dog owners was reached through a commercial survey panel (Centiment, Denver, Colorado, USA), using the same inclusion criteria. Responses for that group were limited to Canada and the United States. The commercial survey group received an incentive from the host company for completing the survey. There was no incentive for the social media group. Social media group responses were collected between March 25 and April 10, 2024. The commercial survey group was surveyed between April 10 and April 17, 2024.

The survey queried the owner's location (country, state/province) and number of dogs in the household. For each dog, the numbers of direct physical contacts with other dogs (including household contacts) in the preceding 24 h, 7 d, and 14 d, were queried. "Physical contact" was defined in the survey as follows: "This is when your dog has had direct nose-to-nose or other direct dog-to-dog contact. There is no minimum timeframe (*e.g.,* quick direct nose-to-nose contact with a dog on a walk counts the same as spending all day in a day care with another dog)." Respondents were asked to estimate the number of contacts with "different dogs," so repeated contact with the same dog was only counted as 1 contact. A history (in the past 14 d) of boarding, participating in a group puppy or training class, participating in an organized event (*e.g.,* dog show, agility), visiting a veterinary clinic, participating in pack walking, visiting a friend or neighbor that owned a dog, visiting a family member (living at a different location) that owned a dog, visiting an off-leash park, attending a day care, or visiting a grooming facility were specifically queried. If there were more than 3 dogs in the household, information was

TABLE 1. Number of owner-reported dog contacts in the preceding 24 h, 7 d, and 14 d for dogs; social media recruitment group (n = 329 dogs) and commercial survey group (n = 829 dogs).

Group	Timeframe	Median	Range	IQR (25th to 75th percentiles)
Social media recruitment group	Preceding 24 h	1	0 to 40	2 (1 to 3)
	Preceding 7 d	3	0 to 100	5 (1 to 6)
	Preceding 14 d	4	0 to 150	7 (2 to 9)
Commercial survey group	Preceding 24 h	2	0 to 30	2 (1 to 3)
	Preceding 7 d	2	0 to 33	3 (1 to 4)
	Preceding 14 d	2	0 to 40	3 (1 to 4)

IQR — Interquartile range.

only requested for 3. In addition, dog age was obtained for the commercial survey group.

Results were described. Comparisons of dog-level data were made using Wilcoxon and X^2 tests. The Steel-Dwass test was used to compare median contacts by age category. Least-squares fitting was used to assess contact activities that were significantly associated with greater numbers of overall dog contacts. Stepwise forward logistic regression was used to evaluate variables associated with a dog being in the top quartile of 14-day contacts, using a liberal univariable P-value of 0.20 to enter variables into the final model and a P-value cutoff of < 0.1 to retain in the final model. Biologically plausible 2-way interactions were tested. Odds ratios and 95% confidence intervals were reported. For all analyses, $P < 0.05$ was considered statistically significant.

This study was approved by the University of Guelph Research Ethics Board (Guelph, Ontario).

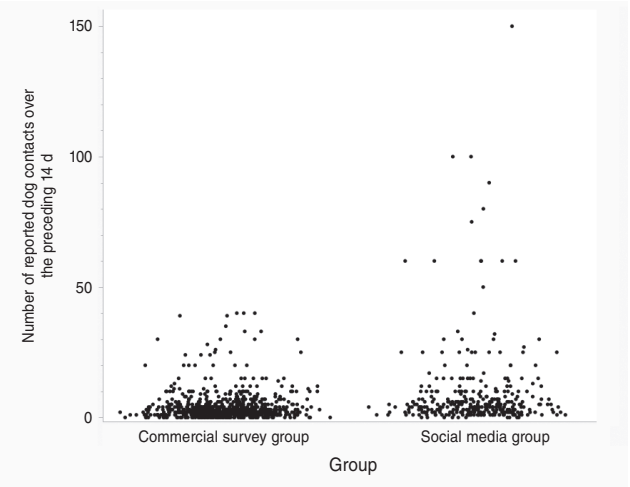

FIGURE 1. Scatterplot of the number of dog contacts in the preceding 14 d, based on responses from owners surveyed *via* a commercial survey (n = 829) and social media (n = 329).

RESULTS

Data were obtained for 1058 dogs. The commercial survey group consisted of 550 households with a total of 829 dogs. The social media group consisted of 205 households with 329 dogs. The median number of dogs owned per household was greater in the commercial survey group than in the social media group (median: 2 *versus* 1, $P < 0.0001$).

There were no differences in numbers of dog contacts in the preceding 24 h in the commercial *versus* social media groups ($P = 0.19$), but there were significantly more contacts reported by the social media group for the past 7 d ($P < 0.0001$) and 14 d ($P < 0.0001$) (Table 1, Figure 1). When individual contact types were evaluated, significant differences in numerous dog contact activities were identified, with the social media group reporting significantly more common participation in group puppy training, organized canine events, or pack walks; and the commercial survey group reporting significantly more visits to off-leash dog parks, day care, groomers, or family members that owned a dog (Table 2). The distribution of the numbers of different queried contact activities is presented in Figure 2. Because of these differences, data and analysis are reported separately for the 2 groups.

Commercial survey group

There were responses from 550 dog owners: 388 (71%) from the United States and 162 (29%) from Canada. These reported data from a total of 829 dogs, 617 (74%) from the United States and 212 (26%) from Canada.

There was a median of 1 dog per household [range: 1 to 10, interquartile range (IQR): 1]. Median dog age was 5 y (range: < 1 to 18 y, IQR: 5 y). Contact numbers and activities are reported in Tables 1 and 2. Dogs had a median of 1 type of queried contact (*e.g.,* day care), with a range of 0 to 10. Two hundred sixty-four (32%) dogs had none of the reported contact activities (Figure 2). One hundred sixty-one (19%) dogs had no reported dog contacts in the

TABLE 2. Reported participation in specific activities by dogs in the preceding 14 d.

Activity	Social media group (n = 329 dogs)	Commercial survey group (n = 829)	P-value
Boarding	10 (3.0%)	40 (4.8%)	0.20
Group puppy training	58 (18%)	42 (5.1%)	< 0.001
Organized event (*e.g.*, show, agility)	52 (16%)	32 (3.9%)	< 0.001
Veterinary clinic visit	84 (26%)	180 (22%)	0.16
Pack walk	56 (17%)	79 (9.5%)	0.0005
Visit to a friend or neighbor that has a dog	84 (26%)	228 (28%)	0.51
Visit to an off-leash dog park	35 (11%)	194 (23%)	< 0.0001
Day care	17 (5.2%)	102 (12%)	0.0002
Groomer	11 (3.3%)	184 (22%)	< 0.0001
Visit to a family member that has a dog	46 (14%)	231 (28%)	< 0.0001

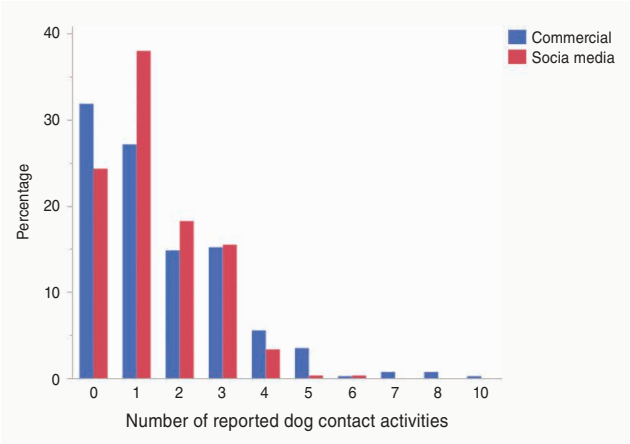

FIGURE 2. Number of selected activities engaged in by dogs in the preceding 14 d, based on responses from owners surveyed *via* a commercial survey (n = 829) and social media (n = 329).

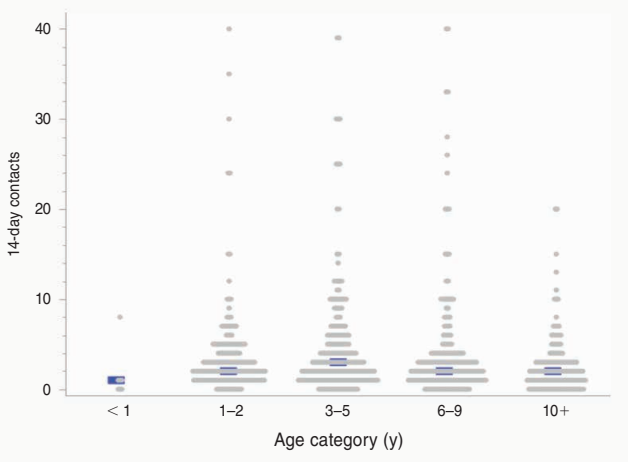

FIGURE 3. Scatterplot and median values for the number of 14-day dog contacts, presented by dog age, based on responses from owners surveyed *via* a commercial survey (n = 829).

preceding 24 h, whereas 116 (14%) and 101 (12%) had no reported contacts in the preceding 7 and 14 d.

Dogs from the United States had significantly greater numbers of contacts in the preceding 24 h (USA median: 2, Canada median: 1; $P < 0.0001$) but no difference in the preceding 7 d (both medians: 2, $P = 0.39$) and 14 d (both medians: 2, $P = 0.42$). Dogs from Canada were more likely than dogs from the United States to have participated in a pack walk (17% *versus* 7.1%, $P = 0.0002$). There were no differences between countries in boarding ($P = 0.14$), group puppy or training class ($P = 0.72$), organized events, visiting a veterinary clinic ($P = 0.34$), visiting a friend or neighbor who owns a dog ($P = 0.72$), visiting a family member who owns a dog ($P = 1.0$), visiting an off-leash park ($P = 0.35$), visiting a day care ($P = 0.90$) or visiting a groomer ($P = 0.50$). There was also no difference in the median number of queried contact activities (median: 1, $P = 0.52$).

Impact of dog age. Dog age was significantly associated with a greater number of contacts in the preceding 14 d ($P = 0.029$) but not the preceding 24 h ($P = 0.66$) or 7 d ($P = 0.15$). When age was categorized (< 1 y, 1 to 2 y, 3 to 5 y, 6 to 9 y, ≥ 10 y), there were significantly more 14-day contacts for 1 to 2 *versus* ≥ 10-year-old dogs ($P = 0.023$) and 3 to 5 *versus* 10-year-old dogs ($P = 0.0005$). However, though statistically significant, the biological relevance was likely minimal, as the median values were identical (2 contacts) (Figure 3). Contacts with large numbers of dogs tended to occur in dogs between 1 and 9 y of age.

When age was evaluated as a continuous variable, age was not significantly associated with boarding ($P = 0.33$), group puppy or training classes ($P = 0.14$), participating in an organized event ($P = 0.83$), visiting a veterinary clinic ($P = 0.40$), participating in a pack walk ($P = 0.41$), visiting a friend or neighbor with a dog ($P = 0.11$), visiting an off-leash park ($P = 0.56$), attending a day care ($P = 0.22$),

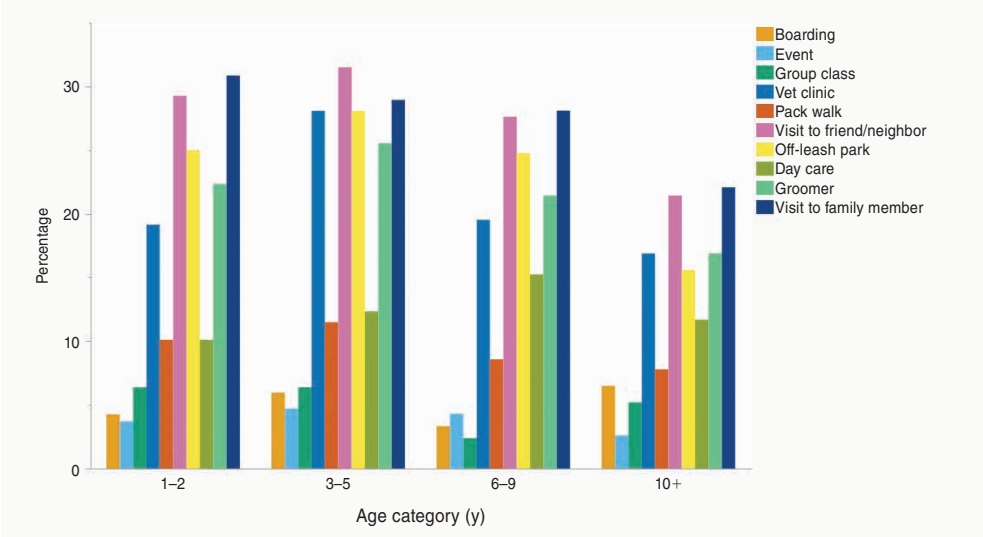

FIGURE 4. Participation in selected activities in the preceding 14 d, presented by dog age, as reported by owners surveyed *via* a commercial survey (n = 829).

TABLE 3. Dog contact activities significantly associated with the number of reported individual dog contacts; commercial survey group (n = 829 dogs).

Timeframe	Significant variable	Association	*P*-value
24 h	Day care	Increased	< 0.0001
	Off-leash dog park	Increased	0.005
7 d	Day care	Increased	< 0.0001
	Off-leash dog park	Increased	< 0.0001
	Visit to family member with a dog	Increased	0.014
14 d	Day care	Increased	< 0.0001
	Off-leash dog park	Increased	< 0.0001
	Visit to family member with a dog	Increased	0.02
	Group puppy training	Increased	0.035
	Boarding	Decreased	0.02

visiting a groomer (P = 0.20), or visiting a family member with a dog (P = 0.14). When age was categorized, there was a significant association of age with visiting an off-leash dog park (P = 0.032), but not with other activities (all $P < 0.10$) (Figure 4).

Risk factor analysis. Factors significantly associated with contact numbers for all timeframes are presented in Table 3. Visiting a day care or visiting an off-leash dog park were consistently associated with a greater number of contacts. Conversely, boarding was associated with a lower number of contacts in the preceding 14 d. A multivariable analysis of factors associated with being in the top quartile of dog contact numbers in the preceding 14 d is presented in Table 4.

TABLE 4. Multivariable analysis of factors associated with being in the top quartile of dog contacts over the preceding 14 d; commercial survey group (n = 829 dogs).

Variable	Odds ratio (95% CI)	*P*-value
Number of dogs in the household	1.5 (1.2 to 1.8)	0.0001
Boarding	0.07 (0.01 to 0.55)	0.012
Off-leash dog park	3.5 (2.2 to 5.6)	< 0.0001
Day care	3.1 (1.6 to 5.8)	0.004
Visit to family member with dog	2.9 (1.8 to 4.6)	< 0.0001

Social media group

The 205 respondents provided information for 329 dogs (1 to 11 dogs per household, median: 2, IQR: 2). Respondents were from Canada (n = 119, 58%), the United States (n = 83, 40%), Argentina (n = 1, 0.5%), Australia (n = 1, 0.5%), and Switzerland (n = 1, 0.5%).

Numbers of dog contacts (including household contacts) over 1, 7, and 14 d are shown in Table 1. Fifty-one (16%) dogs had no reported contacts with any other dogs in the preceding 24 h, whereas 17 (5.2%) had no contacts in the preceding 7 d and 10 (3.0%) had no contacts in the preceding 14 d.

In the preceding 14 d, 249 (76%) dogs had participated in at least 1 of the queried contact activities (Table 2).

A country comparison was made only between Canada and the United States. There were no differences in the reported number of dog contacts in the preceding 24 h (Canada median: 2, USA median: 1; P = 0.48) or 7 d (Canada median: 3, USA median: 3; P = 0.14), but there was a difference in the number of 14-day contacts (Canada median: 5, USA median: 4; P = 0.014). Canadian dogs were

TABLE 5. Dog contact activities significantly associated with the number of reported individual dog contacts; social media group (n = 329 dogs).

Timeframe	Significant variable	Association	*P*-value
24 h	Day care	Increased	< 0.0001
	Group puppy training	Increased	0.04
7 d	Day care	Increased	< 0.0001
	Off-leash dog park	Increased	< 0.0001
14 d	Day care	Increased	< 0.0001
	Off-leash dog park	Increased	0.0003
	Organized event	Increased	0.01
	Boarding	Decreased	0.004

TABLE 6. Multivariable analysis of factors associated with being in the top quartile of dog contacts over the preceding 14 d; social media group (n = 329 dogs).

Variable	Odds ratio (95% CI)	*P*-value
Group puppy or training class	2.6 (1.2 to 5.6)	0.014
Organized event	6.8 (3.0 to 15.1)	< 0.0001
Off-leash dog park	7.8 (3.3 to 18.8)	< 0.0001
Day care	26.2 (6.3 to 110)	< 0.0001

more likely than United States dogs to have participated in a pack walk (22 *versus* 10%, $P = 0.007$) or visited a friend with a dog (30 *versus* 17%, $P = 0.006$). There were no other differences in selected contact activities (all $P > 0.13$).

Risk factor analysis. Factors significantly associated with contact numbers for all timeframes are shown in Table 5. As with the commercial survey group, visiting a day care or visiting an off-leash dog park were consistently associated with a greater number of contacts, and boarding was associated with a lower number of contacts in the preceding 14 d. A multivariable analysis of factors associated with being in the top quartile of dog contact numbers is shown in Table 6.

DISCUSSION

Dog contacts were relatively low for most dogs, with median direct contact numbers of 1 to 4 dogs, depending on timeframe and group, including household contacts. Many dogs had no reported contacts over the studied periods or had none of the queried higher-risk contact activities. Although this study could not capture all potential sources of pathogen exposure, dogs with few or no high-risk contacts were likely at limited risk of acquisition of canine pathogens that are transmitted through close contact. However, at the other extreme, some dogs had a substantial number of contacts (1 dog had contact with an estimated 150 dogs in 14 d) and multiple dogs participated in all queried contact activities. Number of contacts itself is not the only factor associated with disease risk, as some individual contacts may pose greater or lesser risk. It is plausible that dogs with small numbers of high-risk contacts could be at greater risk of disease than those with large numbers of lower-risk contacts. This is why there is a need to look at specific types of known or perceived high-risk contacts in addition to absolute contact numbers. However, it is reasonable to assume that, in general terms, more contacts mean more risk. These issues highlight the nonhomogeneous nature of pathogen risk, which is a factor that needs to be considered when modelling disease transmission, developing interventions, and communicating risk.

Timeframes chosen for investigation were empirical but were chosen to understand temporal trends and to include timeframes that would be relevant for common infectious diseases. The 14-day timeframe encompassed main pathogen-shedding periods of many relevant infectious diseases, such as canine influenza virus, canine parvovirus, canine parainfluenza viruses, and canine respiratory coronavirus. It was also empirically chosen as a timeframe to determine how much contacts increased with time while avoiding problems with recall bias that can arise with longer timeframes. It is impossible to query timeframes related to all potential communicable disease risks because some pathogens can be shed for prolonged periods of time (*e.g.*, canine distemper virus, *Bordetella bronchiseptica*) or throughout life *(e.g., Brucella canis)*.

This study queried "direct physical contact" as the highest-risk and most readily identifiable type of contact. This may have resulted in an underestimation of exposure because of close but non-direct contacts (such as aerosol, droplet, and airborne exposures) and indirect transmission from shared environments or fomites. Querying all possible exposures is challenging, and the decision for this study was to focus on the highest-risk form of contact, direct contact.

There may be seasonal effects on dog contact behaviors, and those were not investigated in this study. This survey was conducted in March and April, and weather factors, particularly in colder climates, could affect the number of certain types of dog contact (*e.g.*, off-leash dog park visits). The study period also included a major holiday (Easter) that could have influenced certain contact types, such as visiting family members with dogs.

Off-leash park and day care were the variables most consistently and strongly associated with increased number of contacts and were associated with a dog being in the top quartile of dog contacts in both survey groups. This

was unsurprising because of the potential for contact with numerous dogs, particularly in parks. Day care-associated infectious disease risk has been well described in dogs, including for pathogens such as *Leptospira* spp., canine papillomavirus, and canine influenza virus (10–12). Less is known about dog parks, likely because of limited investigation and challenges in identifying parks as a source of exposure. However, the potential for contact with large numbers of dogs of unknown health status creates ample risk.

Interestingly, boarding was associated with fewer reported contacts through different analyses. Although perhaps counterintuitive, this could reflect a reduction in the occurrence and frequency of other activities, such as visiting an off-leash dog park, for dogs at a boarding kennel with their contacts restricted to a small number of co-boarders. However, boarding is a known risk factor for pathogen exposure (13) and, though it may be associated with fewer contacts, those contacts may be of higher risk than average. It is also possible that owners do not know how many dogs might be encountered during boarding, resulting in underestimation of contacts.

Potential biases of survey populations must always be considered, as obtaining a truly representative sample is challenging. We chose to use 2 approaches to maximize enrolment and reduce the effect of biases that could be present in a single population. The social media population could plausibly have been overrepresented by people with a greater interest in infectious diseases, based on their following of the author or his infectious disease website. The commercial survey group would not have had the same bias but was still a subset of the population and likely had its own biases. Regardless, the goal was not to have an exact understanding of dog contacts but rather to provide basic information about the types and numbers of contacts, so to assist with risk assessment and communications. The differences in contact numbers and patterns between the 2 groups demonstrate the need to consider the effect of recruitment on results and the value of using broad or multiple recruitment methods.

The data obtained here only captured certain components of contact networks and pathogen transmission risk. This type of approach can be complemented with other methods to assess contact networks and specific types and durations of contacts, such as structured and detailed interviews of dog owners. Those can provide more detailed and potentially more accurate data, but may be impractical, particularly with large numbers of dogs or wide geographic areas. Electronic (*e.g.,* GPS-based) tracking systems have also been employed (3), but those provide information about geographic range with little or no information about dog-dog interactions. Newer methods such as video observation with artificial intelligence assessment may offer advantages for assessing certain types of contacts, but they are not without logistical and ethical concerns and can only evaluate selected aspects of contacts.

Dog contact networks are just one component of pathogen transmission risk, since transmission from other sources or indirect contacts can occur. However, direct contacts likely pose the greatest overall transmission risk for most infectious diseases of dogs, and an understanding of the number and type of contacts is needed to properly investigate risks and to identify optimal control approaches. These data provide important baseline information about contact numbers and types for 2 populations of pet dogs in Canada and the United States. Although there will likely be regional, seasonal, and other sources of variation, information identified in this study can be used for preliminary disease modelling and for consideration of risk and intervention approaches for common and emerging infectious diseases of dogs. CVJ

REFERENCES

1. Chen S, Lanzas C. Distinction and connection between contact network, social network, and disease transmission network. Prev Vet Med 2016;131:8–11.
2. Eames K, Bansal S, Frost S, Riley S. Six challenges in measuring contact networks for use in modelling. Epidemics 2015;10:72–77.
3. Brookes VJ, VanderWaal K, Ward MP. The social networks of free-roaming domestic dogs in island communities in the Torres Strait, Australia. Prev Vet Med 2020;181:104534.
4. Kittisiam T, Phimpraphai W, Kasemsuwan S, Thakur KK. Analyses of contact networks of community dogs on a university campus in Nakhon Pathom, Thailand. Vet Sci 2021;8:299.
5. Laager M, Mbilo C, Madaye EA, *et al.* The importance of dog population contact network structures in rabies transmission. PLoS Negl Trop Dis 2018;12:e0006680.
6. Muinde P, Bettridge JM, Sousa FM, *et al.* Who let the dogs out? Exploring the spatial ecology of free-roaming domestic dogs in western Kenya. Ecol Evol 2021;11:4218–4231.
7. Warembourg C, Fournié G, Abakar MF, *et al.* Predictors of free-roaming domestic dogs' contact network centrality and their relevance for rabies control. Sci Rep 2021;11:12898.
8. Wilson-Aggarwal JK, Ozella L, Tizzoni M, *et al.* High-resolution contact networks of free-ranging domestic dogs *Canis familiaris* and implications for transmission of infection. PLoS Negl Trop Dis 2019;13:e0007565.
9. Westgarth C, Gaskell RM, Pinchbeck GL, Bradshaw JW, Dawson S, Christley RM. Walking the dog: Exploration of the contact networks between dogs in a community. Epidemiol Infect 2009;137:1169–1178.
10. Iverson SA, Levy C, Yaglom HD, *et al.* Clinical, diagnostic, and epidemiological features of a community-wide outbreak of canine leptospirosis in a low-prevalence region (Maricopa County, Arizona). J Am Vet Med Assoc 2021;258:616–629.

11. Barrell EA, Pecoraro HL, Torres-Henderson C, Morley PS, Lunn KF, Landolt GA. Seroprevalence and risk factors for canine H3N8 influenza virus exposure in household dogs in Colorado. J Vet Intern Med 2010;24:1524–1527.
12. Lane HE, Weese JS, Stull JW. Canine oral papillomavirus outbreak at a dog daycare facility. Can Vet J 2017;58:747–749.
13. Anderson TC, Crawford PC, Dubovi EJ, Gibbs EP, Hernandez JA. Prevalence of and exposure factors for seropositivity to H3N8 canine influenza virus in dogs with influenza-like illness in the United States. J Am Vet Med Assoc 2013;242:209–216.

ARTICLE

Risk factors associated with Johne's disease in a captive wood bison herd

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ABSTRACT

Objective and animals

Mycobacterium avium subsp. *paratuberculosis* (Map) has been identified in a wide range of domestic and wild ruminants. Captive wildlife, including *Bison* spp., can experience Johne's disease-related epidemiological scenarios similar to those seen in cattle. To date, there is no epidemiological information about Map in captive wood bison *(Bison bison athabascae)* herds. The objective of this study was to examine age, sex, and location-specific density as potential risk factors associated with Map positivity in a captive wood bison herd located in Alberta.

Procedure and results

Fecal samplings were obtained from October 2021 to October 2022, DNA was extracted, and qPCR targeting IS*900* and F57 followed by liquid culture confirmation was completed. Within-herd prevalence of IS*900*/F57 qPCR in October 2021 was 4.7%; prevalence of IS*900* qPCR with culture confirmation was 6.8%. Regression analysis using 3 different outcomes based on different diagnostic approaches was employed: "base scenario" (positive result by fecal IS*900* qPCR with culture confirmation of IS*900*/F57 qPCR), "scenario 1" (positive result by fecal IS*900* qPCR with culture confirmation of IS*900* qPCR), and "scenario 2" (positive result by fecal F57 qPCR with culture confirmation of F57 qPCR). Wood bison in the age group ≥ 6 to 9 y were more likely to be Map-positive in all scenarios. Location B (higher animal density) was significantly associated with animals being Map-positive in base scenario and scenario 1.

Conclusion and clinical relevance

This study identified risk factors related to Johne's disease in a captive wood bison herd. The findings can be used to initiate more studies in both the commercial and wild wood bison herds.

RÉSUMÉ

Facteurs de risque associés à la paratuberculose chez un troupeau de bisons des bois en captivité

Objectif et animaux

Mycobacterium avium subsp. *paratuberculosis* (Map) a été identifié chez une large variété de ruminants domestiques et sauvages. Les animaux sauvages en captivité, y compris les bisons, peuvent connaître des scénarios épidémiologiques liés à la paratuberculose semblables à ceux observés chez les bovins. À ce jour, il n'existe aucune information épidémiologique sur Map dans les troupeaux de bisons des bois (*Bison bison athabascae*) en captivité. L'objectif de cette étude était d'examiner l'âge, le sexe et la densité spécifique à l'emplacement comme facteurs de risque potentiels associés à la positivité de Map dans un troupeau de bisons des bois en captivité situé en Alberta.

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Procédure et résultats

Des échantillons fécaux ont été obtenus d'octobre 2021 à octobre 2022, l'ADN a été extrait et une qPCR ciblant IS*900* et F57 suivie d'une confirmation par culture en bouillon a été réalisée. La prévalence au sein du troupeau de IS*900*/F57 par qPCR en octobre 2021 était de 4,7 %; la prévalence de IS*900* par qPCR avec confirmation par culture était de 6,8 %. Une analyse de régression utilisant 3 résultats différents basés sur différentes approches diagnostiques a été utilisée : « scénario de base » (résultat positif par qPCR fécale pour IS*900* avec confirmation par culture de la qPCR IS*900*/F57), « scénario 1 » (résultat positif par qPCR fécale pour IS*900* avec confirmation par culture de la qPCR IS*900*) et « scénario 2 » (résultat positif par qPCR fécale pour F57 avec confirmation par culture de la qPCR F57). Les bisons des bois dans le groupe d'âge ≥ 6 à 9 ans étaient plus susceptibles d'être positifs à la Map dans tous les scénarios. L'emplacement B (densité animale plus élevée) était significativement associé à des animaux positifs à Map dans le scénario de base et le scénario 1.

Conclusion et pertinence clinique

Cette étude a identifié des facteurs de risque liés à la paratuberculose dans un troupeau de bisons des bois en captivité. Les résultats peuvent être utilisés pour réaliser d'autres études dans les troupeaux de bisons des bois commerciaux et sauvages.

(Traduit par Dr Serge Messier)

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INTRODUCTION

Johne's disease (JD) is an infectious granulomatous enteritis affecting domestic and wild ruminants worldwide and transmitted to susceptible individuals through the fecal-oral route. Infected wild and domestic animals usually develop diarrhea and weight loss 2 to 5 y after infection. Johne's disease is caused by *Mycobacterium avium* subsp. *paratuberculosis* (Map), a slow-growing mycobacterium. It can be diagnosed through Map detection in tissues or fecal samples, or serology testing to identify antibodies (1). Detection of Map can be based on culture techniques that are relatively time-consuming or by targeting different genes *via* polymerase chain reaction (PCR), with different sensitivity and specificity levels reported (2). *Mycobacterium avium* subsp. *paratuberculosis* has been identified in a wide range of domestic and wild ruminants (3), and sometimes nonruminants (4), in almost all countries in the world. Affected domestic species are mainly dairy and beef cattle, but of importance to Canada are also examples in wildlife; *e.g.,* bighorn sheep (5), plains bison *(Bison bison bison)* (6), and elk (7).

There are 2 subspecies of bison recognized in North America: plains bison and wood bison *(Bison bison athabascae)*. These species have morphological differences; wood bison have larger body size and darker and heavier pelage compared to plains bison. Moreover, the location of the highest point of the hump in plains bison is centered over the front legs, but in wood bison is forward of the front legs (8). In addition, the geographic distribution differs between subspecies: plains bison are most abundant in the Great Plains across Canada, the United States, and Mexico, whereas wood bison herds are located mainly in boreal forests in northern Canada and Alaska (9).

Bison are among the susceptible species, and wood bison in particular are a keystone species in Canada. Extensive conservation efforts are in progress to ensure their recovery. However, infectious diseases are reported as a threat to the population (8), with JD a possible threat for success in the recovery of animal numbers. Previous studies reported that captive plains bison herds developed clinical signs and lesions similar to cattle (6). *Mycobacterium avium* subsp. *paratuberculosis* in free-ranging wood bison has been documented in Canada, where Map DNA was reported in fecal samples from apparently healthy individuals (10,11). However, there is anecdotal information about clinical cases of JD in captive wood bison.

Disease-control efforts around the world have focused on prevention of oral-fecal transmission by separating cows and calves (dairy), testing and culling positive animals, implementing strict biosecurity measures, protecting susceptible animals, adopting low-risk replacement strategies, and vaccinating in countries where permitted (12). Captive wildlife experience JD transmission similar to that

TABLE 1. Age distribution in a wood bison herd during sampling events in October 2021 and October 2022.

	October 2021				
	n	Median (y)	IQR	Min	Max
Location A					
Cows	38	7.5	5, 12	3	16
Bulls	6	8	4, 12	4	12
Calves	16	< 6 mo	< 6 mo	< 6 mo	< 6 mo
Location B					
Cows	77	6	4, 9	3	16
Subadult females	36	1	1, 2	1	2
Bulls/subadult males	24	2	1, 3	1	8
Calves	37	< 6 mo	< 6 mo	< 6 mo	< 6 mo
Total	234				
	October 2022				
	n	Median (y)	IQR	Min	Max
Location A					
Cows	39	6	4, 10	3	13
Bulls	3	5	5, 9	5	9
Calves	22	< 6 mo	< 6 mo	< 6 mo	< 6 mo
Location B					
Cows	58	6	5, 10	3	16
Subadult females	24	2	2, 2	1	2
Bulls/subadult males	13	2	2, 2	1	4
Calves	32	< 6 mo	< 6 mo	< 6 mo	< 6 mo
Total	191				

IQR — Interquartile range.

described in beef cattle, as cow-calf pairs are not separated and cattle graze on potentially contaminated pastures (3).

To date, there is no epidemiological information about Map in captive wood bison herds. Identification of risk factors for JD in captive wood bison would be beneficial to help focus the control efforts for this disease. Our study was executed in a captive bison herd with a known history of clinical JD in which animals exhibited diarrhea and weight loss with confirmed laboratory diagnosis. Therefore, the objective of this study was to examine potential risk factors; *e.g.*, age, sex, pregnancy diagnosis, and Map infection status of the dam. All may be associated with Map-positive laboratory diagnoses in this wood bison herd and would help to inform control efforts.

MATERIALS AND METHODS

This research was approved by the Veterinary Sciences Animal Care Committee (VSACC) of the University of Calgary (Calgary, Alberta) (#AC21-0187).

Animals and housing

The study was conducted in a captive wood bison herd located close to Fort McMurray in Alberta. The first detection of several clinical JD cases in the herd was in 2019, when animals were observed with signs of weight loss and diarrhea. The diagnosis was confirmed by a commercial laboratory (Prairie Diagnostic Services, Saskatoon, Saskatchewan) using fecal direct quantitative PCR (qPCR). Additional diagnostic tests carried out at the University of Calgary identified a Type-II (cattle) strain, specifically secondary clade. Animals were grazed on 336 ha of land with double fencing to keep wildlife species out that had not been used for grazing except for this wood bison herd. Pastures were divided into "location A" with 141 ha, "location B" with 171 ha, and "location C" with 24 ha (used only in summer months). Location A housed 44 animals (excluding calves), whereas location B had 137 animals (excluding calves), indicating a higher density in the latter (0.3 *versus* 0.8 animals per ha). The subgroups were relatively stable and maintained at either location A or B; however, some animals (mainly calves and 1- to 2-year-old females) could be transferred from one location to another for management reasons such as reproduction. Calves remained with their mothers for ~8 to 10 mo. This corresponds with the calving season, which takes place between April and June, with weaning typically occurring in February of the following year. From May to late October, the animals were grazing; during winter months, their feed was supplemented

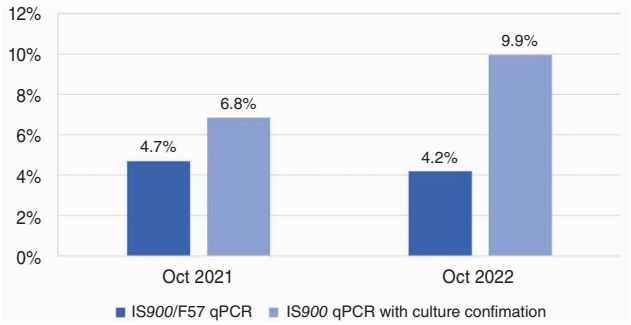

FIGURE 1. Within-herd prevalence of *Mycobacterium avium* subsp. *paratuberculosis* in wood bison, using IS*900*/F57 quantitative polymerase chain reaction (qPCR) (deep blue) and IS*900* qPCR with culture confirmation (light blue), from fecal samples taken in October 2021 and October 2022.

with a mix of meadow bromegrass *(Bromus biebersteinii),* timothy *(Phleum pratense),* and alfalfa *(Medicago sativa)*. In addition, water was provided in troughs year-round, and natural sources of water were also available.

Sample collection

Fecal samples were collected from the whole herd in October 2021 and October 2022. In addition, 2 opportunistic samplings were undertaken — one during February 2022, when the focus was on adult cows; and the other in September 2022, when a herd subgroup consisting of cows, subadults, and bulls was targeted. Each fecal sample from the October 2021 and 2022 and February 2022 samplings was obtained directly from the rectum (with a clean, shoulder-length plastic glove for every individual) while the bison was on a scale attached to a hydraulic bison squeeze (Berlinic Mfg., Quill Lake, Saskatchewan). In September 2022, each sample was collected directly from the ground and was obtained by observing the animal ID and collecting the sample immediately after defecation, using a sterile scoop. Samples were transferred from the gloves or sterile scoops into sterile containers and maintained in clean coolers with ice packs. Samples were maintained at 4°C and sent to the Faculty of Veterinary Medicine, University of Calgary, arriving 1 to 3 d after collection and refrigerated at 4°C until analyzed within 5 d.

Laboratory analysis of fecal samples

All laboratory JD diagnostic tests were conducted in a BSL-2 laboratory at the Faculty of Veterinary Medicine, University of Calgary. Testing for JD in fecal samples is certified through the U.S. Department of Agriculture National Veterinary Services Laboratories by successfully completing the annual JD direct PCR proficiency panel.

DNA extraction from fecal samples and quantitative polymerase chain reaction

Fecal samples were processed individually. The MagMAX Total Nucleic Acid Isolation Kit (Applied Biosystems by Thermo Fisher Scientific, Vilnius, Lithuania) was used, following the manufacturer's protocol. The DNA from feces and fecal culture was analyzed by qPCR targeting the IS*900* gene, plus F57 when positive for IS*900*. In addition, an internal amplification control was used, and all the sequences were identical to those previously described for Map detection (13,14). Briefly, each reaction contained 10 μL of TaqMan Fast Advanced Master Mix (Applied Biosystems), 10 pmol of each primer, 10 pmol of probes for IS*900* and for F57, 500 copies of the internal control plasmid, and 2 μL of DNA template. For qPCR, thermocycler (CFX96 Thermal Cycler; Bio-Rad, Hercules, California, USA) conditions were 50°C for 2 min and 95°C for 20 s for initial denaturation; followed by 42 cycles of 95°C for 3 s and 60°C for 30 s; and finally, 72°C for 5 min. Negative controls were included in each reaction. Samples were run in duplicate, and average quantification cycle Cq was recorded, with Cq values < 37 considered positive.

Map fecal culture

Culture was conducted on fecal samples that were positive by direct fecal IS*900* qPCR, using a modified TREK ESP II liquid culture system (TREK para-JEM; TREK Diagnostic Systems, Cleveland, Ohio, USA) as previously described (11). Two grams of each sample were added to distilled water. Samples were mixed and allowed to settle for 30 min. In the first decontamination step, 25 mL of 0.9% HPC (Thermo Fisher Scientific, Waltham, Massachusetts, USA) and half-strength BHI (BD Diagnostics, Franklin Lakes, New Jersey, USA) were added to 5 mL of the settled mixture and samples were incubated at 37°C for 24 h. Samples were then centrifugated at $3000 \times g$ for 20 min. The pellet was resuspended in 3 mL of antibiotic brew [1 mL para-JEM AS (Thermo Fisher Scientific), 1 mL full-strength BHI, and 1 mL ddH_2O] and incubated overnight at 37°C. Finally, 1 mL of the antibiotic brew and 2 mL of enrichment media were added [1 mL para-JEM GS (Thermo Fisher Scientific) and 1 mL para-JEM EYS (Thermo Fisher Scientific)] to para-JEM culture bottles and incubated at 37°C for 49 d. After incubation, IS*900* and F57 qPCR was used as described herein. Results were considered positive when a $Cq < 37$ was obtained for either F57 or IS*900* genes.

TABLE 2. Univariate logistic regression of variables considered associated with a *Mycobacterium avium* subsp. *paratuberculosis*-positive status of fecal samples taken from wood bison in a farmed herd.

	Base scenario				
Variable	Positive *(n)*	Negative *(n)*	Total *(n)*	OR (95% CI)	*P*-value
Age group (y)					
≥ 1 to 2 (reference)	6	41	47	—	—
≥ 3 to 5	16	37	53	2.9 (1.0 to 8.3)	0.041[b]
≥ 6 to 9	14	32	46	2.9 (1.0 to 8.6)	0.043[b]
≥ 10 to 16	5	30	35	1.1 (0.3 to 4.1)	0.842
Sex					
Male (reference)	3	28	31	—	—
Female	38	112	150	3.2 (0.9 to 11.0)	0.070[b]
Map infection status of the dam					
Negative (reference)	31	77	108	—	—
Positive	5	22	27	0.5 (0.2 to 1.6)	0.289
Location					
Location A (reference)	2	42	44	—	—
Location B	39	98	137	8.4 (1.9 to 36.2)	0.005[b]
Pregnancy rate[a]					
Nonpregnant (reference)	4	39	43	—	—
Pregnant	9	76	85	1.3 (0.4 to 4.4)	0.7

OR — Odds ratio; 95% CI — 95% confidence interval.
"Base scenario" reflected a positive test result: IS*900* qPCR and culture confirmation F57 qPCR.
[a] Variable analyzed in a separate univariate model since only results from October 2021 were included due to pregnancy diagnosis made in October 2021.
[b] Variables used for the multivariable analysis ($P \leq 0.20$).

Data analysis

Logistic regression analysis was conducted. Animals ≥ 1 y ($n = 181$) from the whole herd were included in the regression analysis. Three scenarios were analyzed using the different laboratory results for categorization of positive animals. The base scenario was a common commercial definition of a positive animal (IS*900* screen with F57 confirmation). Scenario 1 lacked F57 confirmation (more chance of false positive test results). Scenario 2 was based on F57 (more chance of false negative test results). In more detail, the scenarios were as follows: in "base scenario," a positive result was fecal IS*900* qPCR with culture confirmation of IS*900* and F57 qPCR. In "scenario 1," a positive result was fecal IS*900* qPCR with culture confirmation of IS*900* qPCR. Finally, in "scenario 2," a positive result was fecal IS*900* and F57 qPCR with culture confirmation of F57 qPCR. Furthermore, we used the following criteria: if an animal was tested > 1 time during the 4 sampling time points, only the first positive result was considered in the statistical analysis.

Variables were defined as follows. "Age" was categorized into 4 groups: ≥ 1 to 2 y, ≥ 3 to 5 y, ≥ 6 to 9 y, and ≥ 10 to 16 y, to create biologically relevant groups based on susceptibility as well as more even data distribution. Since ages were reported annually, if an animal tested positive between October 2021 and February 2022, its age as recorded in fall 2021 was used. For animals testing positive between September 2022 and October 2022, the age as recorded in 2022 was used. The remaining variables were dichotomized: "sex" (female/male), "location" (A/B), "Map infection status of the dam" (positive/negative), "pregnancy diagnosis during 2021" (pregnant/nonpregnant, for adult females only).

Variables with more than 30% missing values were not included in the analysis. Statistical analysis was carried out using STATA 17.0 software (2021; StataCorp, College Station, Texas, USA). The outcome variable was "animal Map status" (positive/negative). A descriptive analysis was initially undertaken to explore all variables. Univariate analysis was used to assess unconditional association between the dependent variable "animal Map status" and each independent variable, using logistic regression. Associations with $P \leq 0.20$ were considered in the multivariable logistic regression model.

Confounding was evaluated by assessing the β-coefficients of the variables of the adjusted model compared with the non-adjusted model. Confounders were retained if a change > 10% was observed. Two-way interactions were examined if > 2 variables were retained as significant risk factors in the final model ($P < 0.05$). A stepwise backward elimination process was used. Furthermore, multicollinearity among the

TABLE 3. Multivariable logistic regression to identify age group, sex, and location associated with a positive *Mycobacterium avium* subsp. *paratuberculosis* status of fecal samples taken from a wood bison herd.

	Base scenario		
Variable	aOR (95% CI)	Standard error	*P*-value
Age group (y)			
≥ 1 to 2 (reference)	—	—	—
≥ 3 to 5	4.1 (1.4 to 12.5)	2.3	0.013[a]
≥ 6 to 9	6.5 (1.9 to 22.1)	4.0	0.002[a]
≥ 10 to 16	3.7 (0.6 to 21.2)	3.3	0.141
Sex			
Male (reference)	—	—	—
Female	4.7 (0.9 to 22.7)	3.7	0.05[a]
Location			
Location A (reference)	—	—	—
Location B	9.9 (2.1 to 46.9)	7.9	0.004[a]

aOR — Adjusted odds ratio; 95% CI — 95% confidence interval.
Hosmer-Lemeshow test ($P = 0.81$).
"Base scenario" reflected a positive test result: IS*900* qPCR and culture confirmation F57 qPCR.
[a] $P \leq 0.05$.
Logistic regression model was adjusted for confounding of Map infection status of the cow.

explanatory variables was tested by measuring the variance inflation factor of each variable. The maximum association of any one explanatory variable with the others was < 50%. The results from the final model were presented as odds ratio (OR) or adjusted odd ratio (aOR) with 95% confidence intervals (95% CIs). Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test.

RESULTS

Samplings and age distributions at different time points

During October 2021, the total number of animals sampled was 234, including cows (n = 115), bulls (n = 30), subadult females (n = 36), and calves (n = 53) (Table 1). In February 2022, 59 samples from cows were collected. During sampling in September 2022, 77 samples were collected, including from cows (n = 39), bulls (n = 10), subadult females (n = 17), and subadult males (n = 11). Finally, during October 2022, 191 samples were collected from cows (n = 97), bulls (n = 13), subadult females (n = 24), subadult males (n = 3), and calves (n = 54). In October 2021, the median age in location A for cows was 7.5 y, the median age for bulls was 8 y, and the median age for all calves was < 6 mo. In location B, the median age for cows was 6 y, for bulls was 2 y, for subadult females was 1 y, and for all calves was < 6 mo. By October 2022, the median age in location A for cows was 6 y, for bulls was 5 y, and for all calves was < 6 mo. In location B, the median age for cows was 6 y, for bulls/yearlings was 2 y, for subadult females was 2 y, and for all calves was < 6 mo (Table 1).

Within-herd prevalence and opportunistic sampling prevalence

In October 2021, the prevalence of IS*900*/F57 qPCR was 4.7% (11/234) and the prevalence of IS*900* qPCR with culture confirmation was 6.8% (16/234). During October 2022, the within-herd prevalence of IS*900*/F57 qPCR was 4.2% (8/191) and the prevalence of IS*900* qPCR with culture confirmation was 9.9% (19/191) (Figure 1).

During February 2022, the opportunistic sampling prevalence of IS*900*/F57 qPCR was 28.8% (17/59) and the prevalence of IS*900* qPCR with culture confirmation was the same, at 28.8% (17/59). During September, the opportunistic sampling prevalence of IS*900*/F57 qPCR was 5.2% (4/77) and the prevalence of IS*900* qPCR with culture confirmation was 13.0% (10/77).

Statistical analysis

There were 181 animals (> 1 y old) included in the logistic regression analysis.

Base scenario. Sex, location, and age were associated with Map animal status ($P \leq 0.20$) (Table 2) and were selected for the multivariable analysis (Table 3). The age groups ≥ 3 to 5 y (OR = 4.1; 95% CI: 1.4 to 12.5) and ≥ 6 to 9 y (OR = 6.6; 95% CI: 1.9 to 22.1) were significantly associated with animals being Map-positive, in comparison with the reference group (≥ 1 to 2 y). Furthermore, females had a significantly higher OR for a positive Map status (OR = 4.7; 95% CI: 0.9 to 22.7). In addition, animals in location B were significantly more likely to have a positive Map status in the herd (OR = 9.9; 95% CI: 2.1 to 46.9).

Scenario 1. Sex, location, and age were associated with Map animal status ($P \leq 0.20$) (Table 4) and were selected for the multivariable analysis (Table 5). The age groups ≥ 3 to 5 y (OR = 5.8; 95% CI: 1.9 to 17.2) and ≥ 6–9 y (OR = 6.9; 95% CI: 2.1 to 22.7) were significantly associated with animals being Map-positive, in comparison with the reference group (≥ 1 to 2 y). Furthermore, female sex was significantly associated with a positive Map status in the herd (OR = 7.1; 95% CI: 1.5 to 34.1). In addition, animals in location B were significantly more likely to have a positive Map status in the herd (OR = 13.9; 95% CI: 2.9 to 65.7).

Scenario 2. Sex and age were associated with Map animal status ($P \leq 0.20$) (Table 6) and were selected for the

TABLE 4. Univariate logistic regression of variables considered associated with a *Mycobacterium avium* subsp. *paratuberculosis*-positive status of fecal samples taken from wood bison in a farmed herd.

	Scenario 1				
Variable	Positive *(n)*	Negative *(n)*	Total *(n)*	OR (95% CI)	*P*-value
Age group (y)					
≥ 1 to 2 (reference)	7	40	47	—	—
≥ 3 to 5	20	33	53	3.4 (1.3 to 9.1)	0.013[b]
≥ 6 to 9	15	31	46	2.7 (1.0 to 7.6)	0.049[b]
≥ 10 to 16	5	30	35	0.95 (0.3 to 3.3)	0.939
Sex					
Male (reference)	3	28	31	—	—
Female	44	106	150	3.8 (1.1 to 13.4)	0.033[b]
Map infection status of the dam					
Negative (reference)	35	73	108	—	—
Positive	7	20	27	0.7 (0.3 to 1.8)	0.516
Location					
Location A (reference)	2	42	44	—	—
Location B	45	92	137	10.2 (2.3 to 44.3)	0.002[b]
Pregnancy rate[a]					
Nonpregnant (reference)	5	38	43	—	—
Pregnant	11	74	85	1.3 (0.3 to 3.5)	0.832

OR — Odds ratio; 95% CI — 95% confidence interval.
"Scenario 1" reflected a positive test result: IS*900* qPCR and culture confirmation.
[a] Variable analyzed in a separate univariate model since only results from October 2021 were included due to pregnancy diagnosis made in October 2021.
[b] Variables used for the multivariable analysis ($P \leq 0.20$).

multivariable analysis (Table 7). Location was not analyzed because all positive animals were in location B. Only the age group ≥ 6 to 9 y (OR = 7.5; 95% CI: 1.8 to 30.5) was significantly associated with animals being Map-positive, in comparison with the reference group (≥ 1 to 2 y).

No significant association between pregnancy diagnosis and Map status during October 2021 was reported in any of the scenarios. Furthermore, no significant association between Map infection status of the dam and Map status of the animals in the herd was reported.

TABLE 5. Multivariable logistic regression to identify age group, sex, and location associated with a positive *Mycobacterium avium* subsp. *paratuberculosis* status of wood bison based on IS*900* test positivity.

	Scenario 1		
Variable	aOR (95% CI)	Standard error	*P*-value
Age group (y)			
≥ 1 to 2 (reference)	—	—	—
≥ 3 to 5	5.8 (1.9 to 17.2)	3.2	0.001[a]
≥ 6 to 9	6.9 (2.1 to 22.7)	4.2	0.002[a]
≥ 10 to 16	3.5 (0.6 to 19.8)	3.1	0.160
Sex			
Male (reference)	—	—	—
Female	7.1 (1.5 to 34.1)	5.6	0.015[a]
Location			
Location A (reference)	—	—	—
Location B	13.9 (2.9 to 65.7)	11.3	0.001[a]

aOR — Adjusted odds ratio; 95% CI — 95% confidence interval.
Hosmer-Lemeshow test ($P = 0.94$).
"Scenario 1" reflected a positive test result: IS*900* qPCR and culture confirmation.
[a] $P \leq 0.05$.
Logistic regression model was adjusted for confounding of Map infection status of the dam.

DISCUSSION

Mycobacterium avium subsp. *paratuberculosis* has been described in a wide range of wildlife species, including plains bison, focusing primarily on pathology (6,15–16). In contrast, there have been limited studies in wood bison (10,11). The present study was conducted with the intention of providing epidemiological information on JD within a captive wood bison herd.

In the present study, herd prevalence was 6.8% at the beginning and 9.9% at the end of the study, using IS*900* qPCR with confirmed culture. Although no studies have specifically examined the within-herd prevalence of JD in wood bison for direct comparison, it is noteworthy that management practices for these animals maintained in captivity can be similar to those used in domestic cattle. In dairy industries across North America, the within-herd prevalence ranged between 2.6 and 16%, based on serum ELISA (17,18). Furthermore, with culture used as

TABLE 6. Univariate logistic regression of variables considered associated with a *Mycobacterium avium* subsp. *paratuberculosis*-positive status of fecal samples taken from wood bison in a farmed herd.

	Scenario 2				
Variable	Positive *(n)*	Negative *(n)*	Total *(n)*	OR (95% CI)	*P*-value
Age group (y)					
≥ 1 to 2 (reference)	3	46	49	—	—
≥ 3 to 5	8	44	52	2.8 (0.7 to 11.2)	0.15[b]
≥ 6 to 9	12	34	46	5.4 (1.4 to 20.7)	0.014[b]
≥ 10 to 16	3	31	34	1.5 (0.3 to 7.8)	0.642
Sex					
Male (reference)	2	29	31	—	—
Female	24	126	150	2.8 (0.6 to 12.3)	0.184[b]
Map infection status of the dam					
Negative (reference)	18	90	108	—	—
Positive	5	22	27	1.1 (0.4 to 3.4)	0.819
Location					
Location A (reference)	0	44	44	—	—
Location B	26	111	137	—	—
Pregnancy rate[a]					
Nonpregnant (reference)	3	40	43	—	—
Pregnant	4	81	85	0.6 (0.1 to 3.1)	0.596

OR — Odds ratio; 95% CI — 95% confidence interval.
"Scenario 2" reflected a positive test result: F57 qPCR and culture confirmation.
[a] Variable analyzed in a separate univariate model since only results from October 2021 were included due to pregnancy diagnosis made in October 2021.
[b] Variables used for the multivariable analysis ($P \leq 0.20$).

TABLE 7. Multivariable logistic regression to identify age group and sex associated with a positive *Mycobacterium avium* subsp. *paratuberculosis* status of fecal samples taken from wood bison in a farmed herd.

	Scenario 2		
Variable	aOR (95% CI)	Standard error	*P*-value
Age group (y)			
≥ 1 to 2 (reference)	—	—	—
≥ 3 to 5	3.0 (0.7 to 12.4)	2.1	0.129
≥ 6 to 9	7.5 (1.8 to 30.5)	5.3	0.005[a]
≥ 10 to 16	1.2 (0.1 to 15.3)	1.7	0.795
Sex			
Male (reference)	—	—	—
Female	5.4 (0.7 to 44.1)	5.8	0.11

aOR — Adjusted odds ratio; 95% CI — 95% confidence interval.
Hosmer-Lemeshow test (P = 0.76).
"Scenario 2" reflected a positive test result: F57 qPCR and culture confirmation.
[a] $P \leq 0.05$.
Logistic regression model was adjusted for confounding of Map infection status of the dam.

a diagnostic technique in culled dairy cattle, the reported within-herd prevalence could be up to 16.1% (19). Conversely, the within-herd seroprevalence reported in beef cattle is lower across Canada, ranging between 1.1 and 2.1% (20). However, a prevalence up to 9% was reported in beef cattle in the United States (21–22). When comparing the JD prevalence in this wood bison herd to that in domestic cattle, results seemed to be between those of dairy and beef cattle. Regarding management practices, this herd aligned more closely with beef cattle, as calves are grazed with their females and not weaned until later, allowing the maintenance of fecal-oral transmission from infectious animals to susceptible animals (calves). Unfortunately, most prevalence studies are based on ELISA, a diagnostic technique with lower sensitivity in comparison to qPCR, resulting in underestimations of the prevalence (22).

In the logistic regression models, 3 scenarios were examined, using different diagnostic approaches for defining a positive animal. The diagnostic approaches were based on 2 genes for qPCR, F57 and IS*900*. A higher concentration of Map must be present in the sample to be positive by F57 qPCR compared to IS*900* qPCR. Kralik *et al* (2011) reported a detection limit of 1.03×10^4 for F57 qPCR and 6.87×10^2 Map cells per gram of fecal sample for IS*900* qPCR (23). Therefore, with the objective to identify changes in the age of occurrence, sex, and location, we defined a more conservative approach using only F57 qPCR and culture confirmation with F57 qPCR, a second scenario (base scenario) with a combination of IS*900* qPCR and culture confirmation with F57 qPCR, and a less conservative approach using only IS*900* qPCR and culture confirmation with IS*900* qPCR.

Animals in the age groups ≥ 3 to 5 y and ≥ 6 to 9 y in base scenario and scenario 1 were more likely to be positive for Map. However, the odds were slightly higher in scenario 1. On the other hand, in scenario 2, only animals in the group ≥ 6 to 9 y were more likely to be Map-positive (OR = 7.5). The higher odds reported in scenario 1 in comparison with the base scenario are explained by IS*900* being a gene with multiple copies and a higher Se in comparison with F57, since only 1 F57 copy is located in the Map genome. However, it is worth noting that IS*900*-like sequences can be present in other *Mycobacterium* species, such as *M. porcinum* (24), *M. cookii* (25), and *M. scrofulaceum* (26), resulting in false positive results. Hence, caution must be taken when drawing conclusions on the best test for culling decisions and the consequences of over- and underestimating the true disease status. The age group ≥ 6 to 9 y was significant in all scenarios; this was similar to what has been described in plains bison, which are JD-positive by culture and demonstrated a mean age of 8.2 y (range: 4 to 20 y) (6). Although not common, young animals < 1 year old can have positive results using qPCR and culture as a diagnostic test (27–28). In our study, we had no positive results in animals < 1 y. This result can be attributed to the fact that these animals were only tested once before being removed from the herd as part of management decisions. Therefore, the limited testing frequency before their removal might have contributed to the absence of positive findings in this age group.

In addition, animals in location B were more likely to be Map-positive (OR = 9.9; base scenario). This association can be attributed to the considerable difference in animal density between the 2 locations. Location B had, on average, more animals per ha than location A (0.8 and 0.3 animals per ha, respectively). This observation aligned with findings in domestic cattle, for which increased animal density (> 100 animals) has been linked to elevated risk of Map transmission and calf exposure (29–30). Although this is not new information, according to the Canadian Bison Association (2021), the average herd size of captive bison in Canada is 151 animals, increasing the likelihood of higher Map prevalence, especially if pasture availability is limited (31).

Our study was conducted using a single wood bison herd with specific characteristics, and caution should be exercised when drawing comparisons or conclusions for the Canadian bison industry as a whole. To provide external validity, this study should be repeated in more herds. In conclusion, this study provided insights into age and density as risk factors for JD in a captive wood bison herd that can be used to implement JD control programs. The effects of different diagnostic approaches can further inform control strategies as well as potentially assist when health status of wild animals is essential — *e.g.,* in translocation events. Furthermore, more studies in wood bison herds would strengthen the external validity of the findings and could be focused on identification of additional risk factors (*e.g.,* genetic susceptibility, effects of health status and stress) at herd level as well as the effects of implementing control measures.

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REFERENCES

1. Whittington RJ, Marshall DJ, Nicholls PJ, Marsh IB, Reddacliff LA. Survival and Dormancy of *Mycobacterium avium* subsp. *paratuberculosis* in the environment. Appl Environ Microbiol 2004;70:2989–3004.
2. Barkema HW, Orsel K, Nielsen SS, *et al.* Knowledge gaps that hamper prevention and control of *Mycobacterium avium* subspecies *paratuberculosis* infection. Trans Emer Dis 2018;65:125–148.
3. Manning EJB. Paratuberculosis in captive and free-ranging wildlife. Vet Clin North Am Food Anim Pract 2011;27:621–630.
4. Beard PM, Daniels MJ, Henderson D, *et al.* Paratuberculosis infection of nonruminant wildlife in Scotland. J Clin Microbiol 2001;39:1517–1521.
5. Forde T, Kutz S, de Buck J, *et al.* Occurrence, diagnosis, and strain typing of mycobacterium avium subspecies paratuberculosis infection in rocky mountain bighorn sheep *(Ovis canadensis canadensis)* in southwestern Alberta. J Wildl Dis 2012;48:1–11.
6. Buergelt CD, Layton AW, Ginn PE, *et al.* The pathology of spontaneous paratuberculosis in the North American bison *(Bison bison)*. Vet Pathol 2000;37:428–438.
7. Forde T, Pruvot M, De Buck J, Orsel K. A high-morbidity outbreak of Johne's disease in game-ranched elk. Can Vet J 2015;56:479–483.
8. ECCC. Recovery Strategy for the Wood Bison *(Bison bison athabascae)* in Canada. Species at Risk Act Recovery Strategy Series. Ottawa, Ontario: Environment and Climate Change Canada, 2018.
9. Sanderson EW, Redford KH, Weber B, *et al.* 2008. The ecological future of the North American bison: Conceiving long-term, large-scale conservation of wildlife. Conserv Biol 2008;22:252–266.

10. Sibley JA, Woodbury MR, Appleyard GD, Elkin B. *Mycobacterium avium* subspecies *paratuberculosis* in bison *(Bison bison)* from northern Canada. J Wildl Dis 2007;43:775–779.
11. Forde T, De Buck J, Elkin B, *et al*. Contrasting results of culture-dependent and molecular analyses of *Mycobacterium avium* subsp. *paratuberculosis* from wood bison. Appl Environ Microbiol 2013;79:4448–4454.
12. Whittington R, Donat K, Weber MF, *et al*. Control of paratuberculosis: Who, why and how. A review of 48 countries. BMC Vet Res 2019;15:198.
13. Slana I, Kralik P, Kralova A, Pavlik I. On-farm spread of *Mycobacterium avium* subsp. *paratuberculosis* in raw milk studied by IS*900* and F57 competitive real time quantitative PCR and culture examination. Int J Food Microbiol 2008; 128:250–257.
14. Mortier RAR, Barkema HW, Bystrom JM, *et al*. Evaluation of age-dependent susceptibility in calves infected with two doses of *Mycobacterium avium* subspecies *paratuberculosis* using pathology and tissue culture. Vet Res 2013;44:94.
15. Huntley JFJ, Whitlock RH, Bannantine JP, Stabel JR. Comparison of diagnostic detection methods for *Mycobacterium avium* subsp. *paratuberculosis* in North American bison. Vet Pathol 2005;42:42–51.
16. Stabel J, Palmer M, Whitlock R. Immune responses after oral inoculation of weanling bison or beef calves with a bison or cattle strain of *Mycobacterium avium* subsp. *paratuberculosis*. Proc 7th Int Colloq Paratuberculosis 2003:90–94.
17. Thorne JG, Hardin LE. Estimated prevalence of paratuberculosis in Missouri, USA cattle. Prev Vet Med 1997;31:51–57.
18. Vanleeuwen JA, Keefe GP, Tremblay R, Power C, Wichtel JJ. Seroprevalence of infection with *Mycobacterium avium* subspecies *paratuberculosis,* bovine leukemia virus, and bovine viral diarrhea virus in maritime Canada dairy cattle. Can Vet J 2001;42:195–198.
19. McKenna SLB, Keefe GP, Barkema HW, *et al*. Cow-level prevalence of paratuberculosis in culled dairy cows in Atlantic Canada and Maine. J Dairy Sci 2004;87:3770–3777.
20. Johnson P, Marfleet T, Waldner C, Parker S, Campbell J. Seroprevalence of *Mycobacterium avium* spp. *paratuberculosis* in cow-calf herds located in the prairie provinces of Canada. Can Vet J 2022;63:1247–1251.
21. Hill BB, West M, Brock KV. An estimated prevalence of Johne's disease in a subpopulation of Alabama beef cattle. J Vet Diagnostic Investig 2003;15:21–25.
22. Roussel AJ, Libal MC, Whitlock RL, Hairgrove TB, Barling KS, Thompson JA. Prevalence of and risk factors for paratuberculosis in purebred beef cattle. J Am Vet Med Assoc 2005;226: 773–778.
23. Kralik P, Slana I, Kralova A, Babak V, Whitlock RH, Pavlik I. Development of a predictive model for detection of *Mycobacterium avium* subsp. *paratuberculosis* in faeces by quantitative real time PCR. Vet Microbiol 2011;149:133–138.
24. Taddei R, Barbieri I, Pacciarini ML, Fallacara F, Belletti GL, Arrigoni N. *Mycobacterium porcinum* strains isolated from bovine bulk milk: Implications for *Mycobacterium avium* subsp. *paratuberculosis* detection by PCR and culture. Vet Microbiol 2008;130:338–347.
25. Englund S, Bölske G, Johansson KE. An IS*900*-like sequence found in a *Mycobacterium* sp. other than *Mycobacterium avium* subsp. *paratuberculosis*. FEMS Microbiol Lett 2002;209: 267–271.
26. Cousins DV, Whittington R, Marsh I, Masters A, Evans RJ, Kluver P. Mycobacteria distinct from *Mycobacterium avium* subsp. *paratuberculosis* isolated from the faeces of ruminants possess IS*900*-like sequences detectable by IS*900* polymerase chain reaction: Implications for diagnosis. Mol Cell Probes 1999;13:431–442.
27. Wolf R, Orsel K, De Buck J, Barkema HW. Calves shedding *Mycobacterium avium* subspecies *paratuberculosis* are common on infected dairy farms. Vet Res 2015;46:71.
28. Bolton MW, Pillars RB, Kaneene JB, Mauer WA, Grooms DL. Detection of *Mycobacterium avium* subspecies *paratuberculosis* in naturally exposed dairy heifers and associated risk factors. J Dairy Sci 2011;94:4669–4675.
29. Krieger M, Eisenberg S, Donat K, Campe A. High-impact risk factors for *Mycobacterium avium* ssp. *paratuberculosis* in dairy herds in Germany. Animals 2023;13:1889.
30. Puerto-Parada M, Arango-Sabogal JC, Paré J, *et al*. Risk factors associated with *Mycobacterium avium* subsp. *paratuberculosis* herd status in Québec dairy herds. Prev Vet Med 2018;152:74–80.
31. Canadian Bison Association [Internet]. Bison Census 1996–2021 [updated 2021]. Available from: https://www.canadianbison.ca. Last accessed February 6, 2025.

BRIEF COMMUNICATION
COMMUNICATION BRÈVE

Survey of the antimicrobial susceptibility of *Escherichia coli* isolated from horses admitted to the Western College of Veterinary Medicine, Saskatoon, Saskatchewan

Maria C. Polo, Florence D. Huby, Fabienne D. Uehlinger, Joseph E. Rubin

ABSTRACT

Objective

Antimicrobial resistance is a serious threat in human and veterinary medicine. Among the most problematic resistant organisms are the extended-spectrum β-lactamase (ESBL)-producing Enterobacterales, which are resistant to the 3rd-generation cephalosporins. The purpose of this study was to determine the frequency of colonization of horses admitted to the Western College of Veterinary Medicine with resistant *Escherichia coli*.

Animals and procedure

Rectal swabs were collected from 60 horses admitted between November 2021 and March 2022. Swabs were selectively cultured for *E. coli,* which was identified using standard biochemical tests. The antimicrobial susceptibility of isolates was determined, and resistant isolates were screened using PCR for the presence of ESBL.

Results

Escherichia coli was isolated from all 60 horses. Although pansusceptibility was the most commonly identified phenotype, multidrug-resistant isolates possessing a CTX-M-type ESBL were recovered from 1 horse. Resistance to trimethoprim + sulfamethoxazole was the most commonly encountered resistant phenotype.

Conclusion and clinical relevance

Although resistance was uncommon, the identification of ESBL-producing isolates highlighted the importance of continued surveillance to monitor the emergence of resistance in equine populations.

RÉSUMÉ

Étude de la sensibilité aux antimicrobiens d'*Escherichia coli* isolé chez des chevaux admis au Western College of Veterinary Medicine, Saskatoon, Saskatchewan

Objectif

La résistance aux antimicrobiens est une menace sérieuse en médecine humaine et vétérinaire. Parmi les organismes résistants les plus problématiques figurent les entérobactéries productrices de bêta-lactamases à spectre étendu (ESBL), qui sont résistantes aux céphalosporines de 3e génération. Le but

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de cette étude était de déterminer la fréquence de colonisation des chevaux admis au Western College of Veterinary Medicine par des *Escherichia coli* résistants.

Animaux et procédure

Des écouvillons rectaux ont été effectués sur 60 chevaux admis entre novembre 2021 et mars 2022. Les écouvillons ont été cultivés de manière sélective pour *E. coli*, qui a été identifié à l'aide de tests biochimiques standards. La sensibilité aux antimicrobiens des isolats a été déterminée et les isolats résistants ont été examinés par PCR pour la présence d'ESBL.

Résultats

Escherichia coli a été isolé chez les 60 chevaux. Bien que la pansensibilité était le phénotype le plus fréquemment identifié, des isolats multirésistants possédant une ESBL de type CTX-M ont été retrouvés chez 1 cheval. La résistance au triméthoprime-sulfaméthoxazole était le phénotype de résistance le plus fréquemment rencontré.

Conclusion et pertinence clinique

Bien que la résistance fût rare, l'identification d'isolats producteurs d'ESBL a souligné l'importance d'une surveillance continue pour détecter l'émergence de la résistance dans les populations équines.

(Traduit par Dr Serge Messier)

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Antimicrobial resistance (AMR) is a serious threat to human and animal health. Although the epidemiology of AMR is well-documented in human medicine and food animals, poor definition of the current situation in companion animals, including horses, highlights the need for more surveillance targeting these populations. Antimicrobial-resistance surveillance is essential to quantitatively understand the risk of AMR to animal and human health, aid in the detection of new resistance mechanisms, and develop evidence-based antimicrobial stewardship guidelines (1).

The β-lactam antimicrobials (penicillins, cephalosporines, and carbapenems) are used extensively in veterinary and human medicine. Broad-spectrum β-lactamases, including the extended-spectrum β-lactamases (ESBLs) and AmpC-type enzymes (CMY-2), which are capable of hydrolyzing the 3rd-generation cephalosporins, are an increasingly encountered serious threat to the effectiveness of β-lactam antimicrobials (2). Genes encoding these enzymes are often colocated on plasmids with genes conferring resistance to other drug families, resulting in multidrug resistance and severely limited therapeutic options (3). Among the Enterobacterales, resistance gene-bearing plasmids are recognized as an import mechanism of resistance dissemination within and between populations of humans and animals (4). Extended-spectrum β-lactamase-producing *Escherichia coli,* which are increasingly identified in veterinary and human medicine, play a major role in ESBL dissemination (5). Among animals, ESBL-producing *E. coli* have been increasingly isolated from bovine mastitis, meat products, broiler-chicken feces, and swine and dairy farms over the last 2 decades (6). A limited number of studies also described the isolation and identification of ESBL producers from companion animals, including horses; these organisms often possess resistance genes that are closely related to those in humans from the same region (5,7).

In western Canada, the frequency of AMR in *E. coli* isolated from horses has not been described. The objective of this study was therefore to describe the antimicrobial susceptibility of *E. coli* isolated from the feces of horses presented to the Western College of Veterinary Medicine (Saskatoon, Saskatchewan). Rectal swabs were collected from a convenience sampling of 60 horses brought to the Veterinary Medical Centre (VMC) at the Western College of Veterinary Medicine between November 2021 and March 2022. Horses were included in the study regardless of age, sex, or reason for presentation to the VMC. To minimize sampling bias, only 1 horse per premise was included. Although the study animals were not specifically recruited to represent Saskatchewan's equine population, all horses presented to the VMC during the study period were eligible for inclusion, providing a snapshot of the local caseload. Since all horses enrolled in this study were admitted to the VMC for veterinary care, it is reasonable to speculate that biases in our population would manifest as an overestimation of resistance among *E. coli* from horses.

TABLE 1. Antimicrobial minimum inhibitory concentration distribution of *Escherichia coli* (*n* = 181) isolated from horses (*n* = 60) presented to the Veterinary Medical Centre at the Western College of Veterinary Medicine (Saskatoon, Saskatchewan).

	Minimum inhibitory concentration (μg/mL)									
Drug	0.5	1	2	4	8	16	32	64	Number (%), isolates	Number (%), horses
Ampicillin			63	99	15	1	3		3 (1.7)	1 (1.7)
Amox/clav					177		4		4 (2.2)	2 (3.3)
Cefazolin			177	1			3		3 (1.7)	1 (1.7)
Ceftriaxone		178						3	3 (1.7)	1 (1.7)
Cefuroxime				140	33	5	3		3 (1.7)	1 (1.7)
Cefepime			178				3		3 (1.7)	1 (1.7)
Cefoxitin			119	55	4		3		3 (1.7)	1 (1.7)
Aztreonam			177			1	3		3 (1.7)	1 (1.7)
Ciprofloxacin	178				3				3 (1.7)	1 (1.7)
Levofloxacin	178				3				3 (1.7)	1 (1.7)
Gentamicin		156	21	1		3			3 (1.7)	1 (1.7)
Tobramycin		114	62	2		3			3 (1.7)	1 (1.7)
Tetracycline			176			5			5 (2.8)	2 (3.3)
TMS	155			26					26 (14.4)	9 (15)

Amox/clav — Amoxicillin/clavulanic acid; TMS — Trimethoprim sulfamethoxazole.
The distribution of minimum inhibitory concentrations (MICs) for each drug is presented; the number of isolates with each MIC is indicated numerically in each cell. The concentrations of each drug tested are indicated with shading. Those isolates with MICs above the highest concentration tested were uninhibited. Resistance breakpoints are indicated by vertical lines.

For each horse, metadata, including previous antimicrobial administration in the last 6 mo, age, sex, breed information, and geographic location of the home premises, were collected using a short questionnaire. This study was approved by the University of Saskatchewan Animal Research Ethics Board (Protocol # 20210093).

A rectal swab (BBL CultureSwab; Becton Dickinson, Franklin Lakes, New Jersey, USA) was collected from each horse before initiation of any therapy and within 2 h after admission to the hospital. Within 8 h of collection, swabs were cultured for *E. coli* on MacConkey (Becton Dickinson) and CHROMagar ESBL (CHROMagar, Paris, France) media and incubated overnight at 35°C. Up to 3 colonies with *E. coli*-like morphology were subcultured to blood agar and identified biochemically. All isolates were then frozen at −80°C in trypticase soy broth with 10% glycerol before antimicrobial susceptibility testing.

Antimicrobial minimum inhibitory concentrations (MICs) were determined by broth microdilution using the MicroScan system with the Gram-negative MIC Panel #42 (Thermo Fisher Scientific, Mississauga, Ontario). Antimicrobial susceptibility tests were conducted and interpreted according to the Clinical and Laboratory Standards Institute guidelines and manufacturer's instructions (8). Isolates resistant to 3rd-generation cephalosporins were screened for broad-spectrum β-lactamases (extended-spectrum and AmpC β-lactamases) by PCR. The CTX-M and CMY-2-type β-lactamases were detected and amplicons were sequenced for allelic identification as previously described (9). Isolates possessing broad-spectrum β-lactamases were further characterized by multi-locus sequence-typing using a previously published protocol (10).

Data collected were entered into a Microsoft Excel spreadsheet (Microsoft Corporation, Redmond, Washington, USA), and associations between dichotomous variables were compared with Fisher's exact test, using the freely available GraphPad platform (https://www.graphpad.com/quickcalcs/contingency1.cfm). A value of $P \leq 0.05$ was considered statistically significant. The proportion of horses from which *E. coli* resistant to any antimicrobial was detected was compared between sex, antimicrobial administration in the previous 6 mo (yes/no), history of boarding (*i.e.,* housing with other horses belonging to different owners), and age (≤ 7 years old and > 7 years old). This age categorization was chosen to include an equal number of horses in each age group that were treated with systemic antimicrobials in the previous 6 mo. Furthermore, a history of antimicrobial usage in the previous 6 mo was compared by sex and age.

TABLE 2. Distribution of the isolation from horses of pansusceptible *Escherichia coli versus E. coli* resistant to any antimicrobial.

Variable	Category	*E. coli* resistant to at least one drug	No *E. coli* resistant to any drug	*P*-value
Previous antimicrobial usage	Yes	4	7	0.07
	No	6	43	
History of boarding	Yes	0	4	1.0
	No	10	46	
Age category	≤ 7 y old	4	12	0.43
	> 7 y old	6	38	
Sex	Male	8	25	0.16
	Female	2	25	

A total of 60 horses were sampled, including 27 mares, 28 geldings, and 5 stallions, with an age range from 7 mo to 40 y. Thirty-nine of the 60 horses were quarter horses. There were 4 Arabians, 4 Canadian warmbloods, and 4 thoroughbred horses; the remaining 9 horses were miniature horses, Appaloosas, Belgians, Clydesdales, Percherons, Friesians, or paint horses. Eighteen percent (11/60) of horses had received either local (n = 1) or systemic (n = 10) antimicrobials in the previous 6 mo, including β-lactams, aminoglycosides, or trimethoprim sulfamethoxazole. Of the 11 horses that received antimicrobials, 36% (4/11) were administered a combination of ≥ 2 drugs. Only 7% (4/60) of horses had a history of boarding. *Escherichia coli* were recovered from all 60 horses; 2 isolates were saved from each of 2 horses, 6 isolates from 1 horse, and 3 isolates from the remaining 57 horses, for a total of 181 isolates.

Pansusceptible isolates (n = 153/181) were recovered from most (46/60) horses carrying *E. coli,* and no resistance to the carbapenems or amikacin was identified. Resistance to trimethoprim sulfamethoxazole was most common (14.4%, 26/181) and was identified in *E. coli* isolates from 9 horses (Table 1). Three isolates of multidrug-resistant *E. coli* producing CTX-M and CMY-2-like β-lactamases were identified from a single horse, which was a 12-year-old quarter horse gelding admitted with a penile prolapse. Multi-locus sequence-typing revealed this isolate to be ST244, a strain unrelated to epidemic strains. No statistically significant associations between the presence of resistant *E. coli* and previous antimicrobial usage (P = 0.07), history of boarding (P = 1.0), age categories (P = 0.43), or sex (P = 0.16) were demonstrated (Table 2).

This is the first study in western Canada describing the antimicrobial susceptibility of equine *E. coli,* including the presence of strains producing broad-spectrum β-lactamases. Although most organisms identified were pansusceptible, indicating that resistance is still uncommon in this region, ESBL-producing *E. coli* were recovered from 1 horse. Interestingly, this horse had not received antimicrobials in the previous 6 mo and had no history of boarding, suggesting community acquisition. Although no statistically significant associations with the presence of resistant organisms were identified, the association between previous antimicrobial usage and the presence of resistant organisms trended towards statistical significance (P = 0.07). Although the current study may have been insufficiently powered to detect factors associated with resistance, a recent study examining racehorses in Ontario determined that previous antimicrobial usage was associated with colonization with ESBL-producing *E. coli* (11).

The low frequency of ESBL-producing *E. coli* in this study was consistent with the results of a similar investigation describing the antimicrobial susceptibility of *E. coli* isolated from the remote population of Sable Island horses in eastern Canada. In this study, the majority of horses were only colonized with pansusceptible *E. coli* (139/146, 95%), and isolates from a single horse possessed a CTX-M-type ESBL (12). In contrast, higher rates of resistance were shown in more populated regions of central Canada. A recent study from Quebec described comparatively high frequencies of multidrug resistance (46.3%) and ESBL/AmpC-positive *E. coli* (7.3%) among healthy horses (13). Similarly, among racehorses in Ontario, a much higher frequency of colonization with ESBL-producing *E. coli* (12%) was demonstrated than in the current study (11).

Colonization with resistant *E. coli* has the potential to pose severe therapeutic challenges in cases of infection and the risk for nosocomial and zoonotic transmission in equine clinical settings (14). Currently, there is very little published data describing the transmission of ESBL-producing organisms between humans and companion animals (5).

Although companion-animal ownership has been identified as a risk factor for human ESBL colonization (15), further studies are required to determine the frequency of and risk factors for transmission.

As this is the first study to describe the presence of an ESBL-producing *E. coli* from a horse in western Canada, these results provide valuable baseline data for future investigations. Continued surveillance of AMR in community *E. coli* isolates is necessary to provide evidence in support of antimicrobial stewardship initiatives and to serve as an early warning for the emergence of resistance.

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REFERENCES

1. FAO. Monitoring and surveillance of antimicrobial resistance in bacteria from healthy food animals intended for consumption. Bangkok, Thailand: Food and Agriculture Organization of the United Nations, 2019. Available from: https://openknowledge.fao.org/handle/20.500.14283/ca6897en. Last accessed January 14, 2025.
2. Pitout JDD. Infections with extended-spectrum β-lactamase-producing *Enterobacteriaceae*. Drugs 2010;70:313–333.
3. Nóbrega DB, Brocchi M. An overview of extended-spectrum beta-lactamases in veterinary medicine and their public health consequences. J Infect Dev Ctries. 2014;8:954–960.
4. Carattoli A. Plasmids and the spread of resistance. Int J Med Microbiol 2013;303:298–304.
5. Rubin JE, Pitout JDD. Extended-spectrum β-lactamase, carbapenemase and AmpC producing *Enterobacteriaceae* in companion animals. Vet Microbiol 2014;170:10–18.
6. Watson E, Jeckel S, Snow L, *et al*. Epidemiology of extended spectrum beta-lactamase *E. coli* (CTX-M-15) on a commercial dairy farm. Vet Microbiol 2012;154:339–346.
7. Greig J, Rajić A, Young I, Mascarenhas M, Waddell L, LeJeune J. A scoping review of the role of wildlife in the transmission of bacterial pathogens and antimicrobial resistance to the food chain. Zoonoses Public Health 2015;62:269–284.
8. Clinical and Laboratory Standards Institute [Internet]. CLSI M100-ED34 Performance Standards for Antimicrobial Susceptibility Testing [updated 2024]. Available from: http://em100.edaptivedocs.net/GetDoc.aspx?doc=CLSI%20M100%20ED34:2024&scope=user. Last accessed January 14, 2025.
9. Ghosh KK, Lebert LA, McEwen SA, *et al*. Extended-spectrum β-lactamase and AmpC β-lactamase-producing *Escherichia coli* isolates from chickens raised in small flocks in Ontario, Canada. Microb Drug Resis 2019;25:1250–1256.
10. Wirth T, Falush D, Lan R, *et al*. Sex and virulence in *Escherichia coli:* An evolutionary perspective. Mol Microbiol 2006;60:1136–1151.
11. Shnaiderman-Torban A, Navon-Venezia S, Paitan Y, *et al*. Extended spectrum β lactamase-producing *Enterobacteriaceae* shedding by race horses in Ontario, Canada. BMC Vet Res 2020;16:479.
12. Timonin ME, Poissant J, McLoughlin PD, Hedlin CE, Rubin JE. A survey of the antimicrobial susceptibility of *Escherichia coli* isolated from Sable Island horses. Can J Microbiol 2017;63:246–251.
13. de Lagarde M, Fairbrother JM, Arsenault J. Prevalence, risk factors, and characterization of multidrug resistant and ESBL/AmpC producing *Escherichia coli* in healthy horses in Quebec, Canada, in 2015–2016. Animals 2020;10:523.
14. Walther B, Klein KS, Barton AK, *et al*. Extended-spectrum beta-lactamase (ESBL)-producing *Escherichia coli* and *Acinetobacter baumannii* among horses entering a veterinary teaching hospital: The contemporary "Trojan Horse." PLoS One 2018;13:e0191873.
15. Schmitt K, Kuster SP, Zurfluh K, *et al*. Transmission chains of extended-spectrum beta-lactamase-producing *Enterobacteriaceae* at the companion animal veterinary clinic–household interface. Antibiotics 2021;10:171.

BRIEF COMMUNICATION
COMMUNICATION BRÈVE

Porcine circovirus type 2 (PCV2) genomic diversity in Canadian swine diagnostic samples collected from 2021 to 2023: Highlighting PCV2d and PCV2e genotypes

Marika Koszegi, Chantale Provost, Valérie Grenier St-Sauveur, Vincent Baby, Carl A. Gagnon

ABSTRACT

Objective

In light of growing concerns regarding potential increases in porcine circovirus type 2 (PCV2)-associated diseases in Quebec between 2021 and 2022, the objective was to characterize PCV2 genotype diversity in Canada.

Samples and procedure

A total of 54 analyzed samples (2021 to 2023) either were submitted by veterinarians to the Molecular Diagnostic Laboratory (MDL) of the Centre de Diagnostic Vétérinaire de l'Université de Montréal (CDVUM) or were collected from pigs necropsied at the Quebec Ministry of Agriculture, Fishery and Food (MAPAQ) laboratories. The sick pigs from which samples were collected had various clinical signs, including those of PCV2-associated diseases. Whole-genome sequencing for PCV2 was done either by Sanger sequencing from 2 PCR amplicons covering the entire viral genome or by high-throughput sequencing.

Results

The main PCV2 subtype identified as circulating in Canada was PCV2d (48.1%). Only 1 strain clustered into the PCV2e subtype (1.9%). In previous reports, PCV2b was the main subtype present in the field. However, only 5 PCV2b sequences (9.3%) were identified in 2022 and 2023.

Conclusion and clinical relevance

This is apparently the first official identification of PCV2d and 2e genotypes in diseased Canadian pigs. Results also provided an overview of PCV2 strains now circulating in Canada compared to reports from 2007 to 2008.

RÉSUMÉ

Diversité génomique du circovirus porcin de type 2 (PCV2) à partir d'échantillons diagnostiques porcins du Canada prélevés de 2021 à 2023 : mise en évidence des génotypes PCV2d et PCV2e

Objectif

À la suite des préoccupations grandissantes vis-à-vis une possible augmentation des maladies associées au PCV2 au Québec entre 2021 et 2022, l'objectif de cette étude a été de caractériser la diversité des génotypes du PCV2 au Canada.

Molecular Diagnostic Laboratory (MDL), Centre de diagnostic vétérinaire de l'Université de Montréal (CDVUM) (Koszegi, Provost, Grenier St-Sauveur, Baby, Gagnon) and Swine and Poultry Infectious Diseases Research Centre (CRIPA-FRQ) (Gagnon), Faculté de médecine vétérinaire (FMV), Université de Montréal, 3200, rue Sicotte, Saint-Hyacinthe, Québec J2S 2M2.

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Échantillons et procédure

Un total de 54 échantillons obtenus entre 2021 et 2023 ont soit été envoyés directement par les vétérinaires praticiens au Laboratoire de diagnostic moléculaire (LDM) du Centre de Diagnostic Vétérinaire de l'Université de Montréal (CDVUM) ou les animaux ont été nécropsiés aux laboratoires du Ministère de l'Agriculture, des Pêcheries et de l'Alimentation du Québec (MAPAQ). Les animaux malades, desquels les échantillons ont été prélevés, démontraient différents symptômes incluant ceux liés aux maladies associées au PCV2. Le séquençage du génome entier du PCV2 a été réalisé soit par la méthode de type Sanger à partir de deux amplicons de PCR représentant la totalité du génome viral, soit par séquençage à haut débit.

Résultats

Le principal sous-type de PCV2 retrouvé au Canada a été le PCV2d (48,1 %). Une seule souche virale a été associée au PCV2e (1,9 %). D'anciennes études avaient démontré que le sous-type majoritairement présent dans les fermes était le PCV2b. Cependant, seulement cinq séquences de PCV2b (9,3 %) ont été identifiées en 2022 et 2023.

Conclusion et pertinence clinique

Il s'agit possiblement de la première identification officielle des génotypes PCV2d et PCV2e chez des porcs canadiens infectés. Les résultats obtenus donnent également un aperçu global des souches de PCV2 qui circulent actuellement au Canada comparativement aux précédents rapports de 2007-2008.

(Traduit par les auteurs)

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Postweaning multisystemic wasting syndrome (PMWS) was first identified in Canada in 1991 (1) and afterward was reported worldwide. The etiologic agent of this syndrome, the porcine circovirus (PCV), causes wasting, dyspnea, skin paleness, respiratory distress, diarrhea, late-term abortion, and stillbirth in infected swine (1,2). This circovirus is a small, non-enveloped virus (diameter: ~17 nm) with a single-stranded DNA circular genome, 1700 to 2000 nucleotides in length (3). Its genome contains 3 major open reading frames (ORF): ORF1, encoding for the replicase protein; ORF2, encoding for the capsid protein; and ORF3, encoding for a protein that has a role in virus-induced apoptosis (4).

Pathogenicity of PCV varies depending on the species involved and its genotypes. For example, PCV type 1 species (PCV1) is considered nonpathogenic in infected pigs, whereas the emergence of PMWS in 2004 in Quebec was linked with the appearance of a new viral subtype of PCV type 2 species named PCV2b (5,6). In 2015, a new PCV species, PCV3, was identified in the United States, from pigs with clinical signs of myocarditis, multisystemic inflammation, porcine dermatitis, nephropathy syndrome, and reproductive failure (7). More recently, in 2019, a 4th species, called PCV4, was identified in China following an increase in pigs with severe PMWS clinical signs (8). Both PCV2 and PCV3 species have been reported to circulate in Canadian swine farms (9,10).

The PCV2 viral species is divided into several subtypes (or genotypes): PCV2a to PCV2h (11). Subtype PCV2d was first identified in Asia and the United States in the early 2010s (12–14). It has since spread worldwide, but its presence had not yet been officially reported in Canada. Subtype PCV2e was first identified in a Mexican swine sample collected in 2014, and 1 y later in samples from swine in the United States (15). Moreover, a PCV recombinant virus, named PCV1/2a, was reported in Canada and probably emerged from a faulty inactivated chimeric vaccine containing the ORF1 of PCV1 and ORF2 of PCV2a (16).

Until now, only PCV2a, PCV2b, and PCV1/2a subtypes have officially been reported in Canada (5,9,16). However, a recent upsurge of PCV2-associated diseases cases was observed throughout Quebec. Therefore, our hypothesis was that a new PCV2 strain subtype was involved in those clinical cases. The entire viral genomes of 54 PCV2-positive cases were sequenced.

Samples used for Sanger and high-throughput sequencing came from sick pigs on various farms located across Canada. Samples included in the present study were submitted from 2021 to 2023 to the Molecular Diagnostic Laboratory (MDL) of the Centre de Diagnostic Vétérinaire de l'Université de Montréal (CDVUM). Samples were submitted by swine veterinarians; the Quebec Ministry of Agriculture, Fishery and Food (MAPAQ); and other Canadian diagnostic laboratories. The project was approved

by the Faculté de médecine vétérinaire Institutional Animal Care Committee (Université de Montréal, Saint-Hyacinthe, Quebec) (Protocol # 24-Rech-2290), following the guidelines of the Canadian Council on Animal Care.

Lungs and other tissue samples were collected at the necropsy and were subsequently sent to MDL for PCV2 qPCR testing and, in several cases, for detection of other pathogens such as porcine reproductive and respiratory syndrome virus (PRRSV), swine influenza A virus (IAV-S), *Mycoplasma hyopneumoniae,* and *Streptococcus suis*. Therefore, most of the PCV2-positive sequenced samples were also positive for various combinations of pathogens listed above, illustrating the clinical phenomenon called the "porcine respiratory diseases complex" (PRDC). Consequently, in addition to classical PMWS clinical signs, various clinical signs (and degrees of severity) were reported by swine veterinarians.

Serum, saliva, processing fluids (from castration), and tongue-tip fluid samples were sent directly to MDL or to other Canadian diagnostic laboratories by veterinarians for PCV2 qPCR diagnostic assay. Tissues samples were homogenized in a PBS solution with glass beads using a bead-beater apparatus. Viral DNA was then extracted on either a KingFisher Flex (Thermo Fisher Scientific) or a QIAcube (Qiagen) apparatus, using custom protocols to extract both DNA and RNA. The PCV2 qPCR-positive samples were processed for whole viral genome sequencing. For 50 samples, the entire PCV2 viral genome was amplified using 2 distinct PCR assays providing 2 overlapping amplicons that were subsequently sequenced using Sanger methodology (Plateforme de séquençage et de génotypage des génomes; CHU de Québec-Université Laval Research Center, Québec, Quebec). Briefly, 2 PCR assays were run using a Fast Cycling PCR Kit (Cat. #20374; Qiagen) reagents as recommended by the manufacturer and using the following PCR cycling conditions: initial denaturation at 94°C for 10 min, followed by 45 cycles of the 3-steps temperature cycle (94°C for 1 min, 55°C for 1 min, and 72°C for 2 min), and a final extended elongation period of 10 min at 72°C. To obtain both amplicons and for their subsequent sequencing, we used the following sets of primers: SEQ.PCV.1NF: 5′-GGACCCCAACCCCATAAAA-3′ and SEQ.PCV.1NR: 5′-CCCTCACCTATGACCCCTATGT-3′; SEQ.PCV.2NF: 5′-TGTTTTCGAACGCAGTGCC-3′ and SEQ.PCV.2NR: 5′-CCGTTGTCCCTGAGATCTAGGA-3′. For the remaining samples, viral DNA was extracted using a viral DNA extraction kit (Zymo Research), as recommended by the manufacturer. Then, DNA was amplified using the

TABLE 1. Canadian porcine circovirus type 2 (PCV2) strain genotype classification.

		2021			2022		2023							2021 to 2023
		Sequenced cases			Sequenced cases		Sequenced cases							Total sequenced cases (%)
Genotype	2006 to 2007[a] No. cases (%)	ON[c]	QC	Total (%)	QC	Total (%)	AB	SK	MB	ON	QC	n.a.[d]	Total (%)	
PCV2a	5 (4.13)	1	2	3 (33.3)	1	1 (10)		6	4	4	3	1	18 (51.4)	22 (40.7)
PCV2b	112 (92.56)			0 (0)	1	1 (10)		1	2	1			4 (11.4)	5 (9.3)
PCV2d	Not tested[b]		6	6 (66.7)	8	8 (80)	4			1	7		12 (34.3)	26 (48.1)
PCV2e	Not tested[b]			0 (0)		0 (0)	1						1 (2.9)	1 (1.9)
PCV2a and 2b	4 (3.31)			0 (0)		0 (0)							0 (0)	0 (0)
Total	121 (100)	1	8	9 (100)	10	10 (100)	5	7	6	6	10	1	35 (100)	54 (100)

[a] PCV2a and 2b multiplex qPCR results previously reported in Gagnon *et al*, 2008 (9).
[b] Genotypes unknown in 2006 to 2007.
[c] Sequenced cases from the following Canadian provinces: Alberta (AB), Saskatchewan (SK), Manitoba (MB), Ontario (ON), and Quebec (QC).
[d] Province of origin not disclosed (not available; n.a.).

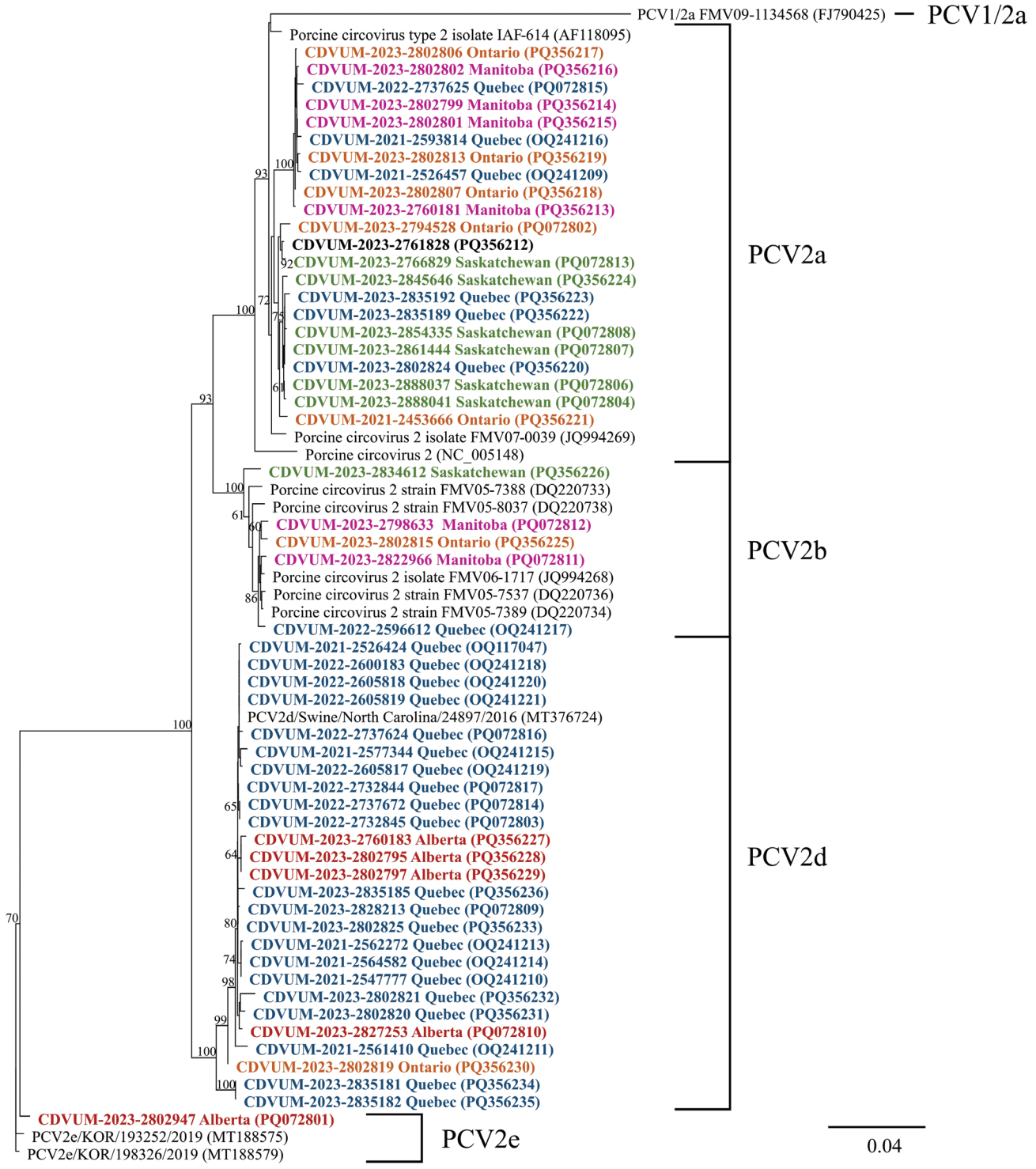

FIGURE 1. Nucleotide phylogenetic tree of porcine circovirus type 2 (PCV2) whole-viral genomes. The 2021 to 2023 Canadian PCV2 sequences are compared to other reference sequences available in GenBank, including several previously reported Canadian PCV2 sequences. The PCV2 sequences subtype classifications (PCV2a, PCV2b, PCV2d, PCV2e, and PCV1/2a) are indicated with bars at the right side of their identification numbers. The Canadian PCV2 nucleotide sequences of the present study are shown in different colors and bold text (Alberta in red, Saskatchewan in green, Manitoba in pink, Ontario in orange, Quebec in blue, and unknown origin in black), with year of sample collection (*i.e.*, 2021 to 2023) and GenBank accession number in round brackets. The distance between each of the PCV2 sequences is represented by the scale bar located at the bottom right. Only bootstrap values ≥ 60 are presented.

rolling circle amplification (RCA) technique with EquiPhi29 enzyme (Thermo Fisher Scientific) and the same primers described. Amplified DNA was quantified using a Qubit fluorometer high-sensitivity DNA kit (Thermo Fisher Scientific) and library preparation was done with Nextera XT kit (Illumina). Library quality was assessed using the Agilent Bioanalyzer (Agilent). Sequencing was done using the MiSeq System (Illumina) and bioinformatics analyses were computed with CLC Genomics Workbench (version 24.0; Qiagen) and Geneious Prime (version 2022.1.1; Dotmatics). Alignments were done using MAFFT multiple sequence alignment program (mafft.cbrc.jp/alignment/software) and trees were created using PhyML (atgc.lirmm.fr/phyml) with 1000 bootstrap replicates.

Between 2021 and 2023, a total of 1730 samples were tested at MDL for PCV2 (607, 562, and 561 in 2021, 2022, and 2023, respectively) using a qPCR-specific diagnostic assay. Of those samples, 21.91% (n = 379) were positive for PCV2 (27.70, 25.80, and 12.48% in the 3 consecutive years), with Ct values ranging from 4.12 to 33.98. Porcine circovirus type 2 whole-genome sequencing was attempted on 62 clinical samples and was successfully obtained with 54 samples (9, 10, and 35 in the 3 y), all collected from independent clinical cases and from which various types of samples were tested (13 sera, 21 tissue samples including lungs alone or within a pool of diverse tissues, 11 saliva, 7 processing fluid, 1 tongue-tip fluid, and 1 unknown). In addition, samples were collected from various pig farms in Alberta (n = 5), Saskatchewan (n = 7), Manitoba (n = 6), Ontario (n = 7) and Quebec (n = 28), including a sample from an unknown province of origin.

Twenty-six PCV sequences were classified as PCV2d, 22 as PCV2a, 5 as PCV2b, and 1 as PCV2e (Table 1). Interestingly, the dominant subtype in the cases selected has shifted from PCV2b (with a prevalence of 92.56% in 2006 to 2007 compared to 9.3% in 2021 to 2023) to PCV2d, representing 48.1% of all cases sequenced from 2021 to 2023 (Table 1).

Unfortunately, surveillance/identification of PCV2 genotypes conducted from 2010 to 2020 was insufficient to determine the exact time of PCV2d genotype appearance in Canada. The fact that PCV2d was already the predominant genotype in diagnostic samples in Quebec in 2021 (Table 1) implied that it probably emerged in Canada before 2021. Interestingly, PCV2d seemed to be the most dominant genotype in Alberta and Quebec, whereas PCV2a was the most dominant genotype in the 3 other provinces, Saskatchewan, Manitoba, and Ontario (Table 1 and Figure 1).

Of note, the temporal distribution of sequences according to their genotype classification was not uniform and could be explained by the fact that the numbers of sequences obtained per year and per province were not similar. This finding may also be partly a consequence of various types of PCV2 vaccine being used in the field. Within Canadian PCV2d strains, whole-genome sequences shared a nucleotide identity from 97.96 to 100% (Figure 1). Compared to other PCV2d entire viral genomes available in GenBank, the Canadian sequences shared a nucleotide identity varying from 98.39 to 100%. For Canadian PCV2a sequences compared to other PCV2a sequences available in GenBank, the shared nucleotide identity varied from 96.83 to 99.89%. For Canadian PCV2b sequences compared to other PCV2b sequences, the shared nucleotide identity varied from 98.42 to 99.83%. For the only Canadian PCV2e entire viral genome sequenced, the closest sequence available was the reference strain PCV2e/KOR/193252/2019 (GenBank accession: MT188575) with a nucleotide identity of 99.38%. To our knowledge, this is the first report of a Canadian PCV2e whole viral genome. Interestingly, the PCV2e-positive case appeared in 2023 in Alberta and represented 1.9% of all sequenced PCV2-positive cases (Table 1).

To our knowledge, this is the first time that PCV2d and PCV2e genotypes are officially reported in Canada. Moreover, the genomic characterization of the PCV2 strains circulating from 2021 to 2023 contributed to knowledge on PCV2 strain prevalence in the Canadian swine population. However, the recently highlighted genomic diversity of Canadian PCV2 strains combined with commercial PCV2 vaccines available in Canada, which should offer at least some level of cross-protection against the rising PCV2d genotype (17,18), could not alone explain the increase in PCV2 cases.

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REFERENCES

1. Harding JCS. Post-weaning multisystemic wasting syndrome (PMWS): Preliminary epidemiology and clinical presentation. 28th Ann Meet Proc Am Assoc Swine Pract 1997:503.
2. Harding JCS, Clark EG. Recognizing and diagnosing postweaning multisystemic wasting syndrome (PMWS). J Swine Health Prod 1997;5:201–203.
3. Fenaux M, Halbur PG, Gill M, Toth TE, Meng XJ. Genetic characterization of type 2 porcine circovirus (PCV-2) from pigs with postweaning multisystemic wasting syndrome in different geographic regions of North America and development of a differential PCR-restriction fragment length polymorphism assay to detect and differentiate between infections with PCV-1 and PCV-2. J Clin Microbiol 2000;38:2494–2503.
4. Liu J, Chen I, Kwang J. Characterization of a previously unidentified viral protein in porcine circovirus type 2-infected cells and its role in virus-induced apoptosis. J Virol 2005;79:8262–8274.
5. Gagnon CA, Tremblay D, Tijssen P, Venne MH, Houde A, Elahi SM. The emergence of porcine circovirus 2b genotype (PCV-2b) in swine in Canada. Can Vet J 2007;48:811–819.
6. Carman S, Cai HY, DeLay J, *et al.* The emergence of a new strain of porcine circovirus-2 in Ontario and Quebec swine and its association with severe porcine circovirus associated disease — 2004–2006. Can J Vet Res 2008;72:259–268.
7. Phan TG, Giannitti F, Rossow S, *et al.* Detection of a novel circovirus PCV3 in pigs with cardiac and multi-systemic inflammation. Virol J 2016;13:184.
8. Zhang HH, Hu WQ, Li JY, *et al.* Novel circovirus species identified in farmed pigs designated as porcine circovirus 4, Hunan province, China. Transbound Emerg Dis 2020;67:1057–1061.
9. Gagnon CA, Del Castillo JR, Music N, Fontaine G, Harel J, Tremblay D. Development and use of a multiplex real-time quantitative polymerase chain reaction assay for detection and differentiation of porcine circovirus-2 genotypes 2a and 2b in an epidemiological survey. J Vet Diag Invest 2008;20:545–558.
10. Zhang M, Liu CC, Huang Y, *et al.* Phylogenetic analysis of porcine circovirus 3 circulating in Canadian pigs. Vet Med Sci 2022;8:1969–1974.
11. Giovanni Franzo JS. Porcine circovirus 2 (PCV-2) genotype update and proposal of a new genotyping methodology. PLoS One 2018;13:e0208585.
12. Xiao CT, Halbur PG, Opriessnig T. Global molecular genetic analysis of porcine circovirus type 2 (PCV2) sequences confirms the presence of four main PCV2 genotypes and reveals a rapid increase of PCV2d. J Gen Virol 2015;96:1830–1841.
13. Xiao CT, Halbur PG, Opriessnig T. Complete genome sequence of a novel porcine circovirus type 2b variant present in cases of vaccine failures in the United States. J Virol 2012;86:12469.
14. Guo L, Fu Y, Wang Y, *et al.* A porcine circovirus type 2 (PCV2) mutant with 234 amino acids in capsid protein showed more virulence *in vivo,* compared with classical PCV2a/b strain. PLoS One 2012;7:e41463.
15. Harmon KM, Gauger PC, Zhang J, Pineyro PE, Dunn DD, Chriswell AJ. Whole-genome sequences of novel porcine circovirus type 2 viruses detected in swine from Mexico and the United States. Genome Announce 2015;3:e01315.
16. Gagnon CA, Music N, Fontaine G, Tremblay D, Harel J. Emergence of a new type of porcine circovirus in swine (PCV): A type 1 and type 2 PCV recombinant. Vet Microbiol 2010;144:18–23.
17. Opriessnig T, Karuppannan AK, Halbur PG, *et al.* Porcine circovirus type 2a or 2b based experimental vaccines provide protection against PCV2d/porcine parvovirus 2 co-challenge. Vaccine 2020;38:1975–1981.
18. Yu C, Cao M, Wei Y, *et al.* Evaluation of cross-immunity among major porcine circovirus type 2 genotypes by infection with PCV2b and PCV2d circulating strains. Vet Microbiol 2023;283:109796.

REVIEW ARTICLE
COMPTE RENDU

Salmonella Dublin in dairy cattle: Review of state of the science

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ABSTRACT

Background

Salmonella enterica subsp. *enterica* serovar Dublin (*Salmonella* Dublin) is a Gram-negative bacterium of increasing importance to the Canadian cattle sector.

Objective, animal, and procedure

The objective of this narrative literature review was to provide an overview of the epidemiology of *Salmonella* Dublin in cattle, highlight risk factors associated with infection, discuss diagnostic methods, and review prevention and control strategies, with a specific focus on the Canadian context.

Results

Approximately 3 to 30% of dairy farms are positive for *Salmonella* Dublin, depending on the province. This bacterium can cause high levels of morbidity and mortality and is best controlled by preventing carrier cattle from gaining access to uninfected farms.

Conclusion and clinical relevance

Salmonella Dublin is an emerging pathogen and action is necessary to control its spread.

RÉSUMÉ

***Salmonella* Dublin chez les vaches laitières : état des connaissances scientifiques**

Contexte

Salmonella enterica sous-espèce *enterica* sérovar Dublin (*Salmonella* Dublin) est une bactérie à Gram négatif d'importance croissante pour le secteur bovin canadien.

Objectif, animal et procédure

L'objectif de cette recension de la littérature était de fournir un aperçu de l'épidémiologie de *Salmonella* Dublin chez les bovins, de mettre en évidence les facteurs de risque associés à l'infection, de discuter des méthodes de diagnostic et de passer en revue les stratégies de prévention et de contrôle, en mettant l'accent sur le contexte canadien.

Résultats

Environ 3 à 30 % des fermes laitières sont positives à *Salmonella* Dublin, selon la province. Cette bactérie peut entraîner des niveaux élevés de morbidité et de mortalité et la meilleure façon de la contrôler est d'empêcher les bovins porteurs d'accéder aux fermes non infectées.

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Conclusion et pertinence clinique

Salmonella Dublin est un agent pathogène émergent et des mesures sont nécessaires pour contrôler sa propagation.

(Traduit par Dr Serge Messier)

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INTRODUCTION

Salmonella enterica subsp. *enterica* serovar Dublin (*Salmonella* Dublin) is a Gram-negative bacterium that can cause disease in cattle. *Salmonella* Dublin was first isolated in Canada in Alberta in 1993 (1), and since this time, it has been reported in all Canadian provinces. It is host-adapted to cattle; however, it is important to note that it can infect other species, including humans (2). The objective of this narrative literature review was to provide an overview of the epidemiology of *Salmonella* Dublin, highlight risk factors associated with infection, discuss diagnostic methods to identify its presence, and review strategies for its prevention and control, with a specific focus on the Canadian context. For this narrative review, a comprehensive search was done using PubMed and Google Scholar online databases, using keywords such as "*Salmonella* Dublin," "cattle," "bovine," "risk factors," "epidemiology," "diagnosis," "treatment," and "control."

CLINICAL SIGNS OF *SALMONELLA* DUBLIN IN CATTLE

Salmonella Dublin causes a variety of clinical presentations in cattle, depending on animal age and whether the pathogen is endemic in the herd. Predominantly, *Salmonella* Dublin affects calves from 2 to 12 wk of age and is associated with high morbidity and mortality (3). Acute infection of a naïve herd can lead to calves with clinical signs of septicemia, endotoxic shock, and sudden death (4). It can also cause respiratory disease unresponsive to treatment, and less commonly, joint inflammation, arthritis, and gangrenous necrosis of distal extremities due to cold agglutination (4–6). In adult dairy cattle, acute disease has been associated with reproductive losses, mainly last-trimester abortions due to bacteremia, as well as fever, diarrhea, depression, and reductions in milk yield and appetite (2,4). However, clinical signs in adult cattle may be unnoticed as they are subtle, with few animals affected.

TREATMENT

Supportive therapy is essential for cattle with acute *Salmonella* Dublin infection. Often, calves will be dehydrated and require fluid therapy, either oral electrolytes or intravenous fluids, depending on the severity of infection (2,3). Nonsteroidal anti-inflammatory drugs are also recommended as they can aid in mitigating direct endotoxin-mediated effects and host systemic inflammatory responses (2). Other supportive therapies include ensuring that calves are housed in a clean, dry environment and are offered high-quality feed or milk, depending on their weaning status, as a source of calories.

Antimicrobial therapy for treatment of *Salmonella* is controversial. Although antimicrobial therapy can increase the odds of recovery, it may fail to limit or reduce the duration of fecal shedding. However, this finding is extrapolated from other species (2). In cattle, there was higher risk of fecal shedding in adults and heifers compared to calves after antimicrobial therapy (7); however, that study was conducted in cattle predominantly infected with *Salmonella typhimurium* and its results may not be directly relevant for *Salmonella* Dublin. Providing antimicrobials is often recommended for calves with acute *Salmonella* Dublin infections, due to the risk of bacteriemia and spread of bacteria to vital organs (2). Another complicating factor is the prominent level of antimicrobial resistance in *Salmonella* Dublin, as highlighted in the next section of this article. Antimicrobial selection should be based on culture and sensitivity, but this is not always possible (2,3). Despite treatment, the case fatality rate can be high [*e.g.,* 26.4% case fatality in an outbreak of *Salmonella* Dublin in dairy calves (8)].

ANTIMICROBIAL RESISTANCE

Among the reasons for the lack of therapeutic success is the rising prevalence of multidrug-resistant *Salmonella* Dublin. In Ontario, increasing levels of resistance were identified from cattle samples submitted to a diagnostic laboratory from 2007 to 2020. This resistance was primarily driven by *Salmonella* Dublin, which was more likely to be resistant to ampicillin and ceftiofur compared to other *Salmonella* spp. (9). Results for Alberta were comparable to those for Ontario, with high levels of multidrug resistance (93.8%), including ceftiofur resistance (68.8%), in *Salmonella* Dublin isolates collected from cattle operations from 2006 to 2014 (10).

TABLE 1. Herd-level prevalence of *Salmonella* Dublin in dairy cattle in recent studies in Canada and elsewhere.

Province/country	Prevalence (%)	Year sampled	Method for prevalence estimation	Reference
Ontario, Canada	2.7	2021 to 2022	Tested all bulk tank milk samples in Ontario (*n* = 3286) at a single time point with ELISA (positivity ≥ 35%).	Perry, 2023 (50)
Quebec, Canada	2.7	2019 to 2020	Tested bulk tank milk samples of a convenience sample of 302 dairy herds from 3 regions in Quebec at 2 time points with ELISA (positivity ≥ 35%).	Um *et al,* 2022 (51)
Alberta, Canada	15.6	2021 to 2022	Tested all bulk tank milk samples in Alberta (*n* = 489) at 4 time points with ELISA (positivity ≥ 35%). Farms were considered positive if ≥ 1 of the samples was positive.	Shaukat *et al,* 2024 (52)
British Columbia, Canada	29.7	2022 to 2023	Tested 461 bulk tank milk samples in British Columbia at 4 time points with ELISA (positivity ≥ 35%). Farms were considered positive if ≥ 1 of the samples was positive.	Himsworth, 2023 (53)
Tyrol, Austria	25.8	2019 to 2022	Tested bulk tank milk samples with ELISA (positivity ≥ 35%) from 3386 dairy farms in Tyrol at 3 time points (2019, 2020, and 2022). Farms were considered positive if ≥ 1 samples was positive.	Hofer *et al,* 2024 (54)
Salzburg, Austria	18.2	2022	Tested bulk tank milk samples from 3119 dairy farms in Salzburg at a single time point with ELISA (positivity ≥ 35%).	Hofer *et al,* 2024 (54)
Denmark	9	2021	Bulk tank samples from all Danish herds tested (ELISA) every 3 mo.	Nielsen *et al,* 2021 (55)
Great Britain	38	2020 to 2021	Bulk tank samples from stratified sample of 401 herds tested quarterly with ELISA (for a herd to be negative, average of the previous 4 results < 25 ODC% and the last of 4 samples < 20 ODC% higher than average of the previous 3).	Henderson *et al,* 2022 (56)
Sweden	1	2013	Bulk tank samples from all Swedish dairy farms (*n* = 4683) tested once with ELISA (positivity ≥ 20%).	Ågren *et al,* 2016 (57)
New York, USA	1	2013	Bulk tank samples from nearly all New York dairy farms (*n* = 4896) tested once with ELISA (positivity ≥ 35%).	Cummings *et al,* 2018 (58)
USA	8	2014	Bulk tank samples from 230 farms across USA tested with ELISA.	Lombard *et al,* 2015 (59)

ODC% — Optical density coefficient.

ZOONOSIS

Of particular concern, *Salmonella* Dublin is zoonotic, with high genetic similarity between human and cattle isolates reported (11). Infected humans can have severe disease, characterized by acute gastroenteritis and septicemia (5). Compared to infection with other *Salmonella* spp. in humans, *Salmonella* Dublin has the highest case fatality rate and more severe clinical outcomes (12), likely due to increasing multidrug resistance. In Canada, the proportion of multidrug-resistant *Salmonella* Dublin isolates from human cases increased from 0% in 2003 to 64% in 2015, coinciding with increased septicemia (11). Although human infections are rare, there have been documented increases in the numbers of human cases of *Salmonella* Dublin in both Canada and the United States (11,12), with a 7.6× increase in the incidence of *Salmonella* Dublin in humans in the United States from 1968 to 2013 (12).

Transmission from cattle to humans usually occurs after consumption of unpasteurized or improperly pasteurised milk from infected cattle or undercooked meat (13). *Salmonella* Dublin was recently identified in 3.6% of livers from milk-fed veal calves at 2 Quebec abattoirs (14), highlighting the importance of proper food handling and preparation. However, infected cattle are also a source of infection if animal feces or fluids are accidently ingested (2).

As human cases originate from cattle, it is critical to control and reduce levels of *Salmonella* Dublin in cattle populations. In Denmark, a decline in human cases coincided with implementation of control measures against *Salmonella* Dublin in the cattle industry (13).

EPIDEMIOLOGY IN CANADA AND GLOBALLY

Prevalence

Salmonella Dublin has often been deemed an emerging disease in Canada because the proportion of infected dairy farms is relatively low when compared to those for other infectious diseases; *e.g., Mycobacterium avium* subsp. *paratuberculosis* (46%) (15) or bovine leukemia virus (78%) (16). Recent estimates of herd positivity for *Salmonella* Dublin in Canada and elsewhere and the methods used to determine positivity are described in Table 1. Because many studies or surveillance projects rely on bulk tank milk samples, it is likely that more farms are positive at the herd level when using a combination of testing strategies, as outlined below.

Transmission

Salmonella Dublin is mostly transmitted *via* the fecal-oral route in cattle herds (17). However, susceptible cattle can become infected by ingesting milk, saliva, nasal secretions, or birth fluids from infected animals or contaminated environments (4,17). There is also evidence that vertical transmission can occur, with calves being congenitally infected at birth (18); however, most *in-utero* infections lead to abortion or stillborn calves (19). Aerosolized transmission, in which bacterial uptake occurs through respiratory or conjunctiva routes, may also be possible, especially for calves in tightly confined housing (20). This is an important consideration when pressure-washing or -cleaning, as organic matter can become aerosolized and infect nearby animals or humans (21).

Risk factors for transmission

Several risk factors have been identified, as summarized by Velasquez-Munoz *et al* (3) and Henderson and Mason (22).

Individual-animal risk factors. The likelihood of animals developing clinical disease is affected by virulence, exposure dose, host age, host immunity, and stressors. The minimum dose of bacteria that results in a clinical infection is estimated to be 10^6 colony-forming units in calves from 0 to 6 mo of age (17); however, this likely varies with age. Because calves are born with immature immune systems, neonates are highly susceptible to all diseases, including *Salmonella* Dublin infection. Thus, contact of newborn calves with *Salmonella* Dublin in feces, colostrum, or other body fluids within calving pens or calf housing is likely to lead to infection and development of clinical disease (3). Other high-risk groups include peripartum cattle or cattle with concomitant disease (*e.g.,* liver flukes or bovine viral diarrhea virus) (23).

Between-herd risk factors. One of the most important factors for introducing and spreading *Salmonella* Dublin within a herd is introductions [purchases or departures and reintroductions (24)]. The reproduction rate of *Salmonella* Dublin was 1.1 to 2.7 in young Danish dairy calves (25) and 2.5 in Dutch dairy herds (26), meaning that 1 to 3 susceptible cattle could become infected with *Salmonella* Dublin due to introduction of a shedding animal (3). Therefore, it is crucial to screen the herd of origin or test individuals before introducing them to a herd. Other risk factors associated with between-farm transmission include livestock-based visitors not being required to wear protective clothing (27) and neighbouring farms being positive (28).

Within-herd risk factors. Factors associated with within-herd spread are related to hygiene and contact of young calves with feces. The calving pen is a suspected area of transmission due to increased shedding of *Salmonella* Dublin around the time of calving in carriers and the high susceptibility of newborn calves. Adding bedding more frequently to the calving area and having ≤ 3 cows per pen in the calving area were associated with lower risk of *Salmonella* Dublin positivity at the herd level (24). Calving in individual calving pens was also associated with a reduced risk of being positive for *Salmonella* Dublin (29), whereas delayed separation of newborn calves from their dams (30), overstocking the calving pen (29,31), and calving in a hospital area for sick cows (23) were associated with an increased risk of being positive. Another factor associated with *Salmonella* Dublin positivity is not isolating sick calves from herd-mates (30,32). Herd size also affects risk, with larger herds having greater probabilities of being positive (33). Furthermore, wild mice have been identified as a reservoir for *Salmonella* Dublin, promoting environmental persistence and highlighting the importance of rodent control (34).

Risk factors for becoming a latent carrier

After an acute infection, surviving cattle can become latent carriers, persistently infected with *Salmonella* Dublin, with the bacteria remaining in lymphoid tissue and being continuously or intermittently shed into the environment through feces, milk, or colostrum (2,35). These persistent infections are characterized by an absence of clinical signs of disease, apart from reduced milk production (4).

Latent carriers are often responsible for spreading the bacterium within and between herds (31,36). Furthermore, when the immune system is challenged or the host undergoes stress, *e.g.,* calving or transportation, shedding may increase (35).

Cattle were at the highest risk of becoming persistently infected when they were initially infected from 12 mo of age to their first calving and in the peripartum period (37). Furthermore, cattle were more likely to become carriers when there were few shedders in the herd, as low doses of *Salmonella* Dublin may increase the risk of becoming a carrier (37). This is most likely due to cattle in high-prevalence herds having better cell-mediated immunity (22). However, in the same study, cattle were also more likely to become carriers if there was an active clinical outbreak in the herd (37), likely due to persistent infections being more common in cattle that are clinically ill (30). As these latent carriers are an important source of infection for herds, management practices aiming to reduce their numbers within an infected herd or prevent introduction into a naïve herd are critical for control (36).

Diagnosis

Individual-animal diagnosis. In live cattle, there are 2 approaches to identify *Salmonella* Dublin infection: bacterial culture and detection of an immune response *via* an enzyme-linked immunosorbent assay (ELISA). A polymerase chain reaction (PCR) test has also been suggested (3) and is used by many Canadian laboratories; however, compared to bacterial culture, real-time PCR did not provide a sensitive alternative for detection of *Salmonella* Dublin in fecal samples (38). Furthermore, bacterial culture isolates are needed to assess antimicrobial susceptibility and, in some cases, are needed for *Salmonella* grouping and serotyping. Hence, the use of PCR is currently not well-validated in live animals and additional research is needed in this area. Any animal with a suspected case of *Salmonella* Dublin that dies should be necropsied.

Bacterial culture.

Fecal culture. Fecal culture is likely most relevant for cattle with acute infections or clinically ill cattle, with a sensitivity of 60 to 100%, as these cattle shed large numbers of the bacteria (> 100 colony-forming units/g) (39). However, sensitivity is only 6 to 14% for detection of subclinical cattle (40), perhaps due to intermittent shedding or low concentrations shed by subclinical or reinfected cattle (4,40). Specificity is assumed to be 100% (with potential for cross-contamination or errors).

TABLE 2. Sensitivity and specificity for *Salmonella* Dublin ELISA on bovine serum, based on age and optical density coefficient cut points.

Age (d)	ODC% cutoff	Sensitivity (%)	Specificity (%)
100 to 300	25[a]	85	88
	35[b]	79	98.5
	50[a]	77	95
> 300	25[a]	73	76
	35[b]	62	94
	50[a]	59	89

ODC% — Optical density coefficient.
Literature sources: [a] (40), [b] (43).

Blood culture. Blood culture has been used to detect *Salmonella* Dublin in untreated, clinically affected calves (4); however, it requires specific transport media and sterile collection of blood, which may not be practical in the field. Sensitivity and specificity have not been explored, although the technique has been used successfully in outbreak investigations (5).

Detection of an immune response. Enzyme-linked immunosorbent assays are commonly used to detect immunoglobulins directed against *Salmonella* Dublin-specific O-antigens in blood or milk (41). After initial infection, antibodies became detectable 10 to 15 d after an experimental inoculation (41); however, on commercial dairy farms with natural exposure to *Salmonella* Dublin, seroconversion was estimated to be 36 d (range: 11 to 67 d) (25). It is also possible to have cross-reaction between *Salmonella* serovars sharing O-antigens, *e.g., Salmonella* Typhimurium, creating false positives (42). Interpretation of ELISA results depends on a semiquantitative measurement of antibody concentration expressed in optical density coefficient (ODC%) (3). It is noteworthy that all test validations for individual-animal detection of *Salmonella* Dublin using ELISA were compared to fecal culture, which is known to be an imperfect test.

Serum ELISA. The accuracy of ELISA depends on cattle age and ODC% cutoff used. Serum ELISA is not recommended in calves < 3 mo of age due to interference from maternally derived antibodies and limited production of *Salmonella*-specific antibodies before 11 to 12 wk of age (40,43). In this age group, if an ODC% cutoff of 25 was used, sensitivity was 46% and specificity was 89%; however, at an ODC% cutoff of 50, sensitivity was 21% and specificity was 96% (40). Testing cattle from 100 to 300 d of age resulted in the highest test performance (40), though there was a reasonable level of performance for cattle aged > 300 d. Sensitivity and specificity of the test

TABLE 3. Herd sensitivity and herd specificity for dairy herd test strategies for *Salmonella* Dublin, based on individual animal results [adapted from Nielsen, 2013 (44)].

Herd testing procedure	Herd sensitivity (%) (95% CI)	Herd specificity (%) (95% CI)
Serology of all young stock < 1 y (if 1 positive, herd considered positive)[a]	96 (92 to 100)	NR
Serology of all young stock from 4 to 6 mo (if 1 positive, herd considered positive)[a]	91 (85 to 97)	NR
Serology of animals with current or previous signs of salmonellosis[a]	80 (71 to 88)	NR
Serology of 10 youngest animals > 3 mo old[b,c]	85 (60 to 99) (depended on herd size and herd prevalence)	NR
Serology of 2 rounds of testing 10 animals > 3 mo old (starting from the youngest available to the next oldest, until 10 sampled)[c]		
≥ 1 positive on any of the 2 rounds of sampling	69 (30 to 97)	77 (71 to 82)
≥ 2 positive on any of the 2 rounds of sampling	34 (10 to 84)	94 (90 to 98)
≥ 3 positive on any of the 2 rounds of sampling	18 (3 to 54)	98 (95 to 100)
Fecal culture of cattle with current or recent signs of salmonellosis[a]	3 (27 to 49)	NR

NR — Not recorded.
Literature sources: [a] (60), [b] (4), [c] (57).

TABLE 4. Herd sensitivity and herd specificity for dairy herd test strategies for *Salmonella* Dublin, using pooled samples [adapted from Nielsen, 2013 (44)].

Herd testing procedure	Herd sensitivity (%) (95% CI)	Herd specificity (%) (95% CI)
Culture of manure-pit sample[a]	44 (33 to 56)	NR
Single bulk tank sample with ELISA at ODC% of 20[a]	54 (44 to 65)	98 (96 to 100)
Single bulk tank sample with ELISA at ODC% of 15[b]	41 (16 to 89)	92 (88 to 96)
Single bulk tank sample with ELISA at ODC% of 35[b]	16 (4 to 44)	98 (96 to 99)
2 bulk tank samples with ELISA at ODC% of 15[c]		
≥ 1 positive on any of the 2 rounds of sampling	79 (45 to 99)	89 (80 to 93)
≥ 2 positive on any of the 2 rounds of sampling	49 (20 to 87)	98 (93 to 100)
2 bulk tank samples with ELISA at ODC% of 35[c]		
≥ 1 positive on any of the 2 rounds of sampling	69 (31 to 98)	93 (88 to 98)
≥ 2 positive on any of the 2 rounds of sampling	13 (4 to 38)[e]	99 (97 to 100)[e]
4 bulk tank samples with ELISA at ODC% of 15[c]		
≥ 1 positive on any of the 2 rounds of sampling	86 (59 to 100)[e]	88 (80 to 93)[e]
≥ 2 positive on any of the 2 rounds of sampling	64 (27 to 95)[e]	96 (93 to 99)[e]
4 bulk tank samples with ELISA at ODC% of 35[c]		
≥ 1 positive on any of the 2 rounds of sampling	41 (15 to 84)	99 (96 to 100)
≥ 2 positive on any of the 2 rounds of sampling	24 (10 to 55)[e]	99 (97 to 100)[e]
Average of 4 bulk tank measurements over 5 to 12 mo[d]	95 (83 to 96)	96 (92 to 98)

NR — Not recorded; ODC% — Optical density coefficient.
Literature sources: [a] (60), [b] (52), [c] (61), [d] (62).
[e] Estimated from graphic in Um *et al*, 2023 (61).

at various ODC% cut points are shown in Table 2, with a cut point of 35 ODC% recommended. The PrioCHECK *Salmonella* antibody bovine Dublin ELISA test (Thermo Fisher Scientific, Waltham, Massachusetts, USA) was used in the studies highlighted herein and is the most widely used test in Canada.

Individual milk ELISA. The accuracy of the milk ELISA for individual cows was evaluated in a PhD thesis by Nielsen (2013) (44), using fecal samples as a reference. Using an ODC% cutoff of 25, milk ELISA had an estimated sensitivity of 77 to 78% and a specificity of 65 to 86%, whereas at a cutoff of 50, sensitivity was 42 to 43% and specificity was 81 to 94%. Although a cutoff of 35 is commonly used for diagnosing individual cows with *Salmonella* Dublin, the estimated sensitivity and specificity have not been described in the literature.

TABLE 5. Herd sensitivity and herd specificity for dairy herd test strategies for *Salmonella* Dublin, using a combination of testing strategies.

Herd testing procedure	Herd sensitivity (%) (95% CI)	Herd specificity (%) (95% CI)
Combination of bulk tank milk ELISA and serology of all calves 4 to 6 mo old[a]	99 (96 to 100)	NR
Combination of bulk tank milk ELISA and serology of cattle with current or previous signs of salmonellosis[a]	91 (85 to 97)	NR
2 visits at 6 mo interval; herd considered positive if bulk milk ODC% ≥ 35 and/or ≥ 1/10 cattle positive on ELISA[b]	87 (64 to 99)	92 (87 to 97)

NR — Not recorded; ODC% — Optical density coefficient.
Literature sources: [a] (60), [b] (61).

Necropsy examination. There are no lesions specific to *Salmonella* Dublin. Findings could include an icteric appearance of the overall carcass, petechiae over serosal surfaces, enlarged liver (sometimes with a yellow or orange appearance), enlarged mesenteric lymph nodes, generalized pneumonia with consolidation or surface petechia, splenomegaly, or peritonitis (22). If *Salmonella* Dublin is suspected, lung, liver, spleen, kidney, mesenteric lymph nodes, and intestine should be submitted for bacterial culture and histologic examination.

Identifying carriers. Individual identification of carriers is critical to reduce *Salmonella* Dublin in herds, as carriers are important in maintenance of endemic herd infections. Carrier detection often involves repeated sampling, as shedding can be intermittent. Several options have been evaluated, with varying success.

Fecal culture. Fecal culture has low sensitivity (6 to 14%) for detection of carriers (40) and is not recommended. However, sensitivity may improve during periods of stress; *e.g.*, calving and transportation (22).

Serology. Repeated ELISA testing is often used to detect carriers. Several strategies have been proposed for identifying carriers, including collecting 3 serum samples over 120 d, with those exceeding the ODC% cut point at each sampling classified as carriers (22). Others have used 2 serum samples 2 mo apart, with those with positive results on both tests defined as carriers, as a better predictor compared to a single sample or 2 tests done 1 mo apart (45). A study in Denmark (46) used a different approach to flag cattle based on risk status, in which high-risk cattle were those with ≥ 2 samples > 80 ODC% with a minimum of 120 d in between, the most recent sample > 80 ODC%, and the average of the last up to 4 samples > 80 ODC%. Cattle were characterized as medium risk if the most recent ELISA and the average of the last up to 4 samples were > 50 ODC%, but not high enough to be high risk. Animals with ELISA values < 50 ODC% in the most recent sample were classified as low risk. Overall, though schemes to classify carrier status exist, additional research is needed to optimize detection of carriers.

Herd-level diagnosis. There are several approaches to identify the herd status of *Salmonella* Dublin.

Sampling multiple cattle. This option could include testing all cattle in the herd or testing randomly selected cattle with ELISA, and designating the herd as positive if at least 1 test is positive. The latter testing method is most economical. Testing options in which results from individual cattle are used to identify herd status (44) are outlined in Table 3.

Testing pooled milk or manure samples. Most surveillance systems have used bulk tank milk samples due to ease of sample collection and economics. Potential strategies to determine herd-level status for *Salmonella* Dublin are described in Table 4. It is important to note that a single bulk tank milk test has low sensitivity, potentially leading to misclassification of infected farms as negative. Therefore, multiple bulk tank samples should be used to more accurately determine herd status.

Combination of testing strategies. A combination testing approach is likely the best strategy where young animals and bulk tank milk samples are used. Three strategies evaluated to identify herd status for *Salmonella* Dublin using bulk tank milk samples and serology are described in Table 5.

Control of *Salmonella* Dublin

Noninfected herds. The focus for herds that are not infected is to prevent *Salmonella* Dublin from entering the herd. Maintaining a closed herd by not allowing cattle to leave and return and not purchasing animals is critical; however, this may not be practical, as only 41% of Canadian

dairy farms did not add cattle or have cattle return to their herds in 2014 (47). Therefore, cattle should not be purchased from unknown sources or from test-positive herds (33). Furthermore, a high level of biosecurity is needed for visitors that encounter cattle, including veterinarians, nutritionists, and drovers. Ongoing surveillance is also needed, including necropsies completed in cases of unexpected calf mortality.

Infected herds. As outlined by Nielsen (44), and Nielsen and Nielsen (30), a structured approach can be used to control *Salmonella* Dublin. Nielsen (44) outlined the following 5 steps:

1. Complete risk-scoring, such as scoring outlined by Nielsen and Nielsen (30), to determine transmission routes within the herd and into the herd.
2. Determine a plan of action.
3. Change management practices in areas of high importance.
4. Interpret repeated test results to identify individuals that could be culled or managed.
5. Complete diagnostic tests in various age groups to monitor progress, including bulk tank milk testing alongside individual testing, in calves aged 100 to 300 d.

Below are some specific areas that are important considerations when dealing with *Salmonella* Dublin (3).

Farm personnel. Due to the zoonotic risk, when farms are initially diagnosed with *Salmonella* Dublin, training should be provided to farm staff and families regarding the importance of personal protective equipment (coveralls, washable boots, gloves) when working with cattle, as well as good hand hygiene and avoiding raw, unpasteurized milk. If farms are using pressure-washers, goggles and masks should be considered, as the organism can become aerosolized (21). Before leaving the farm, individuals should remove protective equipment and clean and disinfect boots. As farm personnel can act as fomites, clean equipment, boots, coveralls, and gloves can reduce transmission when working with young, susceptible cattle.

Management of neonates. As young calves are especially susceptible to *Salmonella* Dublin, a focus on their early life management is essential. Because latent carriers often begin shedding in the peripartum period (37), the calving area should be kept as clean as possible to minimize contact of newborn calves with manure. Hence, removing newborn calves from manure contact as soon as possible can break transmission. Reducing stocking density in the calving area (24) and not using the calving area to house sick cows also reduce the risk of *Salmonella* Dublin exposures. Pasteurization of colostrum and milk reduces the levels of *Salmonella enterica* species and other pathogens (48). Furthermore, housing that avoids contact of young calves with older cattle also reduces risk of transmission (31).

Vaccination. No vaccines for *Salmonella* Dublin are currently available in Canada. Some autologous vaccines have been tried, but none have been rigorously evaluated in published studies.

Isolation of clinically ill cattle. This is often suggested to reduce environmental contamination by shedding. However, this alone is not sufficient, as cattle without clinical signs can still shed bacteria (25).

Sanitation. Cleaning and disinfection of the environment is a critical control point. Furthermore, cleaning equipment (*e.g.,* bottles, nipples, and manure-handling equipment) and housing is also important, as *Salmonella* Dublin can survive well in the environment. First, remove organic material (*i.e.,* manure, feed, bedding), as it can inactivate disinfectants. Second, rinse surfaces with water and apply a detergent, followed by a disinfectant agent at the concentration and contact time described on the label. *Salmonella* is susceptible to most disinfectants when proper steps are completed (49). Daily removal of manure, cleaning and disinfection of the calving area at least twice monthly, and adding new bedding weekly controlled *Salmonella* Dublin in endemic herds (31).

CONCLUSION

Salmonella Dublin is an emerging disease in Canadian dairy herds. Currently, the true prevalence in many Canadian provinces is unknown due to the use of only single bulk tank milk samples to detect positive herds. To best manage this disease, Canadian dairy producers should maintain closed herds. For those that have this pathogen present, the focus should be on reducing exposure of young, susceptible calves to manure. CVJ

REFERENCES

1. Guerin MT, Martin SW, Darlington GA, Rajic A. A temporal study of *Salmonella* serovars in animals in Alberta between 1990 and 2001. Can Vet J 2005;69:88–99.
2. Holschbach CL, Peek SF. *Salmonella* in dairy cattle. Vet Clin North Am Food Anim Pract 2018;34:133–154.
3. Velasquez-Munoz A, Castro-Vargas R, Cullens-Nobis FM, Mani R, Abuelo A. Review: *Salmonella* Dublin in dairy cattle. Front Vet Sci 2023;10:1331767.
4. Nielsen LR. Review of pathogenesis and diagnostic methods of immediate relevance for epidemiology and control of *Salmonella* Dublin in cattle. Vet Microbiol 2013;162:1–9.
5. McDonough PL, Fogelman D, Shin SJ, Brunner MA, Lein DH. *Salmonella enterica* serotype Dublin infection: An emerging

infectious disease for the northeastern United States. J Clin Microbiol 1999;37:2418–2427.
6. Loeb E, Toussaint MJM, Rutten VPMG, Koeman JP. Dry gangrene of the extremities in calves associated with *Salmonella* Dublin infection: A possible immune-mediated reaction. J Comp Path 2006;134:366–369.
7. Warnick LD, Kanistanon K, McDonough PL, Power L. Effect of previous antimicrobial treatment on fecal shedding of *Salmonella enterica* subsp. *enterica* serogroup B in New York dairy herds with recent clinical salmonellosis. Prev Vet Med 2003;56:285–297.
8. Guizelini C, Tutija JF, Morais DR, *et al.* Outbreak investigation of septicemic salmonellosis in calves. J Infect Dev Ctries 2020;14:104–108.
9. Uyama T, Renaud D, LeBlanc S. Observational study on antimicrobial resistance in *Escherichia coli* and *Salmonella* isolates from Ontario calf samples submitted to a diagnostic laboratory from 2007 to 2020. Can Vet J 2022;63:260–268.
10. Otto SG, Ponich KL, Cassis R, Goertz C, Peters D, Checkley SL. Antimicrobial resistance of bovine *Salmonella enterica* spp. *enterica* isolates from the Alberta Agriculture and Forestry Disease Investigation Program (2006–2014). Can Vet J 2018; 59:1195–1201.
11. Mangat CS, Bekal S, Avery BP, *et al.* Genomic investigation of the emergence of invasive multidrug-resistant *Salmonella enterica* serovar Dublin in humans and animals in Canada. Antimicrob Agents Chemother 2019;63:e00108.
12. Harvey R, Friedman CR, Crim SM. Epidemiology of *Salmonella enterica* serotype Dublin infections among humans, United States, 1968–2013. Emerg Infect Dis 2017;23:1493–1501.
13. Leekitcharoenphon P, Vigre H, Kaas RS, Aarestrup FM. Trends in *Salmonella* Dublin over time in Denmark from food and animal related isolates. Infect Genet Evol 2023;113:105475.
14. Arsenault JG, Côté P, Turgeon E, *et al.* Prevalence and antimicrobial resistance of *Salmonella* Dublin and thermotolerant Campylobacter in liver from veal calves in Québec, Canada. Foodborne Pathog Dis 2024;21:19–26.
15. Corbett CS, Naqvi SA, Bauman CA, *et al.* Prevalence of *Mycobacterium avium* ssp. paratuberculosis infections in Canadian dairy herds. J Dairy Sci 2018;101:11218–11228.
16. Nekouei O, VanLeeuwen J, Sanchez J, Kelton D, Tiwari A, Keefe G. Herd-level risk factors for infection with bovine leukemia virus in Canadian dairy herds. Prev Vet Med 2015;119: 105–113.
17. Nazer AH, Osborne AD. Experimental *Salmonella* Dublin to calves from adult carrier cows. Vet Rec 1977;92:112–115.
18. Hanson DL, Loneragan GH, Brown TR, Nisbet DJ, Hume ME, Edrington TS. Evidence supporting vertical transmission of *Salmonella* in dairy cattle. Epidemiol Infect 2016;144: 962–967.
19. Hinton M. *Salmonella* Dublin abortion in cattle: Studies on clinical aspects of condition. Br Vet J 1974;130:556–563.
20. Hardman PM, Wathes CM, Wray C. Transmission of *Salmonella* among calves penned individually. Vet Rec 1991;129:327–329.
21. Peek SF, Mcguirk SM, Sweeney RW, Cummings KJ. Rebhun's Diseases of Dairy Cattle. St. Louis, Missouri: Elsevier, 2018: 249–356.
22. Henderson K, Mason C. Diagnosis, and control of *Salmonella* Dublin in dairy herds. In Pract 2017;39:158–168.
23. Fossler CP, Wells SJ, Kanenne JB, *et al.* Herd-level factors associated with isolation of *Salmonella* in a multi-state study of conventional and organic dairy farms: II. *Salmonella* shedding in calves. Prev Vet Med 2005;70:279–291.
24. Perry KV, Kelton DF, Dufour S, Miltenburg C, Umana Sedo SG, Renaud DL. Risk factors for *Salmonella* Dublin on dairy farms in Ontario, Canada. J Dairy Sci 2023;106:9426–9439.
25. Nielsen LR, van den Borne B, van Schaik G. *Salmonella* Dublin infection in young dairy calves: Transmission parameters estimated from field data and an SIR-model. Prev Vet Med 2007;79:46–58.
26. Van Schaik G, Klinkenberg D, Veling J, Stegeman A. Transmission of *Salmonella* in dairy herds quantified in the endemic situation. Vet Res 2007;38:861–869.
27. Van Schaik G, Schukken YH, Nielen M, Dijkhuizen AA, Barkema HW, Benedictus G. Probability of and risk factors for introduction of infectious diseases into Dutch SPF farms: A cohort study. Prev Vet Med 2002;54:279–289.
28. Wedderkopp A, Strøger U, Lind P. *Salmonella* Dublin in Danish dairy herds: Frequency of change to positive serological status in bulk tank milk ELISA in relation to serostatus of neighbouring farm. Acta Vet Scandinavia 2001;42:295–302.
29. Losinger WC, Wells SJ, Garber LP, Hurd H, Thomas LA. Management factors related to *Salmonella* shedding by dairy heifers. J Dairy Sci 1995;78:2464–2472.
30. Nielsen LR, Nielsen SS. A structured approach to control of *Salmonella* Dublin in 10 Danish dairy herds based on risk scoring and test-and-manage procedures. Food Res Int 2012;45:1158–1165.
31. Nielsen TD, Vesterbaek LL, Kudah AB, Borup KJ, Nielsen LR. Effect of management on prevention of *Salmonella* Dublin exposure of calves during a one-year control programme in 84 Danish dairy herds. Prev Vet Med 2012;105:101–109.
32. Nielsen LR. *Salmonella* Dublin faecal excretion probabilities in cattle with different temporal antibody profiles in 14 endemically infected dairy herds. Epidemiol Infect 2013;141:1937–1944.
33. Nielsen LR, Dohoo I. Survival analysis of factors affecting incidence risk of *Salmonella* Dublin in Danish dairy herds during a 7-year surveillance period. Prev Vet Med 2012;107:60–69.
34. Tablante NL, Lane VM. Wild mice as potential reservoirs of *Salmonella* Dublin in a closed dairy herd. Can Vet J 1989;30: 590–592.
35. Counter DE and Gibson EA. *Salmonella* Dublin infection in self-contained dairy herds in East Anglia: Excretion at calving. Vet Rec 1980;107:191–193.
36. Kent E, Okafor C, Caldwell M, Walker T, Whitlock B, Lear A. Control of *Salmonella* Dublin in a bovine dairy herd. J Vet Int Med 2021;35:2075–2080.
37. Nielsen LR, Schukken YH, Gröhn YT, Ersbøll AK. *Salmonella* Dublin infection in dairy cattle: Risk factors for becoming a carrier. Prev Vet Med 2004;65:47–62.
38. Jensen AN, Nielsen LR, Baggesen DL. Use of real-time PCR on faecal samples for detection of sub-clinical *Salmonella* infection in cattle did not improve the detection sensitivity compared to conventional bacteriology. Vet Microbiol 2013; 163:373–377.
39. Richardson A, Fawcett AR. *Salmonella* Dublin infection in calves: Value of rectal swabs in diagnosis and epidemiological studies. Br Vet J 1973;129:151–156.
40. Nielsen LR, Toft N, Ersbøll AK Evaluation of an indirect serum ELISA and a bacteriological faecal culture test for diagnosis of *Salmonella* serotype Dublin in cattle using latent class models. J Appl Microbiol 2004;96:311–319.
41. Robertsson JA. Humoral antibody responses to experimental and spontaneous *Salmonella* infections in cattle measured by ELISA. J Vet Med B 1984;31:367–380.
42. Konrad H, Smith BP, Dilling GW, House JK. Production of *Salmonella* serogroup D (O9)-specific enzyme-linked immunosorbent assay antigen. Am J Vet Res 1994;55:1647–1651.
43. Nielsen LR, Ersbøll AK. Age-stratified validation of an indirect *Salmonella* Dublin serum enzyme-linked immunosorbent assay for individual diagnosis in cattle. J Vet Diagn Invest 2004; 16:212–218.

44. Nielsen LR. *Salmonella* Dublin in cattle: Epidemiology, design and evaluation of surveillance and eradication programmes [DrMedVet dissertation]. Copenhagen, Denmark: University of Copenhagen, 2013.
45. Spier SJ, Smith BP, Tyler JW, Cullor JS, Dilling GW, Da Pfaff L. Use of ELISA for detection of immunoglobulins G and M that recognize *Salmonella* Dublin lipopolysaccharide for prediction of carrier status in cattle. Am J Vet Res 1990;51:1900–1904.
46. Nielsen LR, Dohoo I. Culling decisions of dairy farmers during a 3-year *Salmonella* control study. Prev Vet Med 2011;100:29–37.
47. Denis-Robichaud J, Kelton DF, Bauman CA, Barkema HW, Keefe GP, Dubuc J. Biosecurity and herd health management practices on Canadian dairy farms. J Dairy Sci 2019;102:9536–9547.
48. Stabel JR, Hurd S, Calvente L, Rosenbusch RF. Destruction of *Mycobacterium paratuberculosis, Salmonella* spp., and *Mycoplasma* spp. in raw milk by a commercial on-farm high-temperature short-time pasteurizer. J Dairy Sci 2004;87:2177–2183.
49. Møretrø T, Vestby LK, Nesse LL, Storheim SE, Kotlarz K, Langsrud S. Evaluation of efficacy of disinfectants against *Salmonella* from the feed industry. J Appl Microbiol 2009;106:1005–1012.
50. Perry K. Exploring the prevalence and identifying risk factors associated with *Salmonella* Dublin in Ontario dairy herds [MSc dissertation]. Guelph, Ontario: University of Guelph, 2023. Available from: https://atrium.lib.uoguelph.ca/server/api/core/bitstreams/e886764e-7590-4607-b5d1-2a60dd5d87bc/content. Last accessed January 6, 2025.
51. Um MM, Castonguay MH, Arsenault J. Estimation of the accuracy of an ELISA test applied to bulk tank milk for predicting herd-level status for *Salmonella* Dublin in dairy herds using Bayesian latent class models. Prev Vet Med 2022;206:105699.
52. Shaukat W, de Jong E, McCubbin KD, *et al.* Herd-level prevalence of bovine leukemia virus, *Salmonella* Dublin, and *Neospora caninum* in Alberta, Canada, dairy herds using ELISA on bulk tank milk samples. J Dairy Sci 2024;107:8313–8328.
53. Himsworth C. *Salmonella* Dublin Investigation and Management Program (SDIMP): Final Bulk Milk Testing Report. *Salmonella* Dublin Investigation and Management Program, 2023. Available from: https://www.sdublinbc.ca/reports. Last accessed January 6, 2025.
54. Hofer K, Trockenbacher B, Sodoma E, Khol JL, Dünser M, Wittek T. Establishing a surveillance programme for *Salmonella* Dulbin in Austrian dairy herds by comparing herd-level *versus* individual animal detection methods. Prev Vet Med 2024;230:106277.
55. Nielsen LR, Houe H, Nielsen SS. Narrative review comparing principles and instruments used in three active surveillance and control programmes for non-EU-regulated diseases in the Danish cattle population. Front Vet Sci 2021;8:685857.
56. Henderson K, Mason C, Brülisauer F, Williams P. Determining the prevalence of antibodies to *Salmonella* Dublin in dairy herds in Great Britain by quarterly bulk tank testing. Prev Vet Med 2022;208:105776.
57. Ågren EC, Lewerin SS, Wahlström H, Emanuelson U, Frössling J. Low prevalence of *Salmonella* in Swedish dairy herds highlight differences between serotypes. Prev Vet Med 2016;125:38–45.
58. Cummings KJ, Virkler PD, Wagner B, Lussier EA, Thompson BS. Herd-level prevalence of *Salmonella* Dublin among New York dairy farms based on antibody testing of bulk tank milk. Zoonoses Public Hlth 2018;65:1003–1007.
59. Lombard JE, Thompson BS, Virkler PD, Wagner B, Kristensen RC, Fossler CP. *Salmonella* Dublin antibodies in bulk-tank milk on U.S. dairy operations. ADSA-ASAS Joint Annual Meeting 2015. Orlando, Florida, USA, July 12 to 16, 2015.
60. Veling J, Barkema HW, van der Schans J, van Zijderveld F, Verhoeff J. Herd-level diagnosis for *Salmonella enterica* subsp. *enterica* serovar Dublin infection in bovine dairy herds. Prev Vet Med 2002;53:31–42.
61. Um MM, Castonguay MH, Arsenault J, *et al.* Accuracy of testing strategies using antibody-ELISA tests on repeated bulk tank milk samples and/or sera of individual animals for predicting herd status for *Salmonella* Dublin in dairy cattle. Prev Vet Med 2023;220:106048.
62. Warnick LD, Nielsen LR, Nielsen J, Greiner M. Simulation model estimates of test accuracy and predictive values for the Danish *Salmonella* surveillance program in dairy herds. Prev Vet Med 2006;77:284–303.

ANSWERS TO QUIZ CORNER
CORRIGÉ DU TEST ÉCLAIR

1. B) Supportive care is the first and best treatment for severe coliform mastitis (*i.e.,* caused by *Escherichia coli, Klebsiella* spp., *Enterobacter* spp.). Lipopolysaccharide (LPS) endotoxins released from the bacteria trigger immune-related sepsis. Treat with IV fluids and oral calcium. Nonsteroidal anti-inflammatory agents administered early in the course of disease can decrease the severity of clinical signs and improve treatment outcomes.

The subsequent use of antimicrobial therapy in severe coliform mastitis cases is controversial, but about half of endotoxic cases are also bacteremic. Some studies suggest improved patient outcomes with IV oxytetracycline or IM cephalosporins. Both are extra-label drug use and require Food Animal Residue Avoidance Databank (FARAD) approved drug withdrawal times in the US and Canada (https://cgfarad.usask.ca/index.php).

Pirlimycin is a lincosamide that targets Gram-positive *Staphlyococcus* spp. and *Streptococcus* spp. and would not be indicated in this case.

Prevent new cases of environmental mastitis pathogens with antiseptic teat dip before and after each milking, good bedding management, and fly control. Vaccinate dry cows with bacterin toxoid to aid in prevention of coliform mastitis and endotoxemia.

Expect 1 to 2 cases of clinical mastitis per 100 cows/month in well-managed herds.

Reference

1. Peek S, Divers TJ, Rebhun's Diseases of Dairy Cattle, 3rd ed., St. Louis, Missouri: Elsevier, 2018:432–438.

1. B) Les soins de soutien sont le premier et le meilleur traitement de la mammite à coliformes (causée par *Escherichia coli* ou des bactéries du genre *Klebsiella* ou *Enterobacter*) grave. Les lipopolysaccharides libérés par les bactéries déclenchent une forte réaction immunitaire et peuvent entraîner un choc endotoxique. Il convient de traiter la vache par une fluidothérapie intraveineuse et du calcium oral. L'administration d'anti-inflammatoires non stéroïdiens au début de la maladie peut réduire la gravité des signes cliniques et améliorer les résultats du traitement.

Le recours à l'antibiothérapie dans les cas de mammite à coliformes grave est controversé, mais environ la moitié des animaux qui présentent une endotoxémie présentent aussi une bactériémie. Certaines études indiquent que l'administration d'oxytétracycline par voie intraveineuse ou d'une céphalosporine par voie intramusculaire améliore l'état des patients. Dans les deux cas, il s'agit d'une utilisation non conforme aux indications homologuées et il convient de respecter les périodes de retrait recommandées par le programme FARAD (Food Animal Residue Avoidance Databank) aux États-Unis et au Canada (https://cgfarad.usask.ca/index.php).

La pirlimycine est un lincosamide qui cible les bactéries à Gram positif des familles des staphylocoques et des streptocoques, et elle n'est pas indiquée dans un cas comme celui-ci.

La prévention des nouveaux cas de mammite causée par des agents pathogènes de l'environnement passe par l'emploi d'un bain de trayons antiseptique avant

2. D) This is hypertrophic osteodystrophy (HOD). Lesions center around the metaphysis, causing lameness, and HOD is often accompanied by systemic signs of fever, lethargy, diarrhea, and anorexia. It is seen in fast-growing, large breed dogs, and the etiology is unknown.

Diagnose based on classic radiographic findings: "double physis" sign of an irregular radiolucent zone in the metaphysis, parallel and adjacent to the physis, and metaphyseal periosteal reactions in chronic cases.

Hypertrophic osteodystrophy is very painful, so treatment includes analgesics (NSAIDs), rest, and supportive care. Although HOD is self-limiting, some dogs suffer recurrences until they are 8 to 10 months of age. Prognosis is generally good, except in cases with severe systemic disease: *e.g.*, systemic inflammatory response syndrome. Damage to growth plates can result in angular limb deformities.

Panosteitis is another painful inflammatory condition of unknown etiology commonly seen in young, rapidly growing, large breed dogs. Dogs have pain on palpation of long bones. Differentiate from HOD by radiographic changes: multifocal intramedullary densities of long bones and, sometimes, irregular endosteal surface.

References

1. Cohn L, Côté E. Clinical Veterinary Advisor: Dogs and Cats. 4th ed., St. Louis, Missouri: Elsevier, 2020:508–509.
2. Selman J, Towle Millard H. Hypertrophic osteodystrophy in dogs. J Small Anim Pract 2022;63:3–9. doi: 10.1111/jsap.13413. Epub 2021 Sep 7. PMID: 34490906.

The questions and answers are provided by Zuku Review, online veterinary test prep.

Les questions et les réponses sont gracieusement fournies par le site de préparation aux examens vétérinaires Zuku Review.

et après chaque traite, une bonne gestion de la litière, et la lutte contre les mouches. La vaccination des vaches taries au moyen d'un vaccin de type bactérine et anatoxine peut être envisagée pour aider à prévenir la mammite à coliformes et l'endotoxémie.

Dans les troupeaux bien gérés, on peut s'attendre à constater 1 ou 2 cas de mammite clinique pour 100 vaches par mois.

Référence

1. Peek S, Divers TJ, Rebhun's Diseases of Dairy Cattle, 3rd ed., St. Louis, Missouri: Elsevier, 2018:432–438.

2. D) Le chiot souffre d'ostéodystrophie hypertrophique. Les lésions affectent la métaphyse et provoquent une boiterie. L'ostéodystrophie hypertrophique s'accompagne souvent de signes systémiques tels que fièvre, léthargie, diarrhée et anorexie. Elle est observée chez les chiens de grande race à croissance rapide et son étiologie est inconnue.

Le diagnostic repose sur les observations radiographiques classiques : signe de la « double physe » caractérisé par une zone radiotransparente irrégulière dans la métaphyse qui est parallèle et adjacente à la physe, et réactions périostées métaphysaires dans les cas chroniques.

Comme l'ostéodystrophie hypertrophique est très douloureuse, le traitement comprend des analgésiques (AINS), du repos et des soins de soutien. Bien qu'il s'agisse d'une affection autolimitante, certains chiens présentent des récidives jusqu'à l'âge de 8 à 10 mois. Le pronostic est généralement bon, sauf en cas de maladie systémique grave (syndrome de réponse inflammatoire systémique, par exemple). Les dommages causés aux plaques de croissance peuvent entraîner des déformations angulaires des membres.

La panostéite est une autre affection inflammatoire douloureuse d'étiologie inconnue qui est souvent observée chez les jeunes chiens de grande race à croissance rapide. Les chiens qui en sont atteints présentent une douleur à la palpation des os longs. La panostéite peut être distinguée de l'ostéodystrophie hypertrophique à la radiographie (densités intramédullaires multifocales des os longs et, parfois, surface endostéale irrégulière).

Références

1. Cohn L, Côté E. Clinical Veterinary Advisor: Dogs and Cats. 4th ed., St. Louis, Missouri: Elsevier, 2020:508–509.
2. Selman J, Towle Millard H. Hypertrophic osteodystrophy in dogs. J Small Anim Pract 2022;63:3–9. doi: 10.1111/jsap.13413. Publication en ligne le 7 septembre 2021. PMID: 34490906.

FOOD ANIMAL MATTERS
DÉFIS DU SECTEUR BIOALIMENTAIRE

Food Animal Matters: Fire, Part 2

Robert Tremblay

In a previous Food Animal Matters column, 2 veterinarians described their personal experiences in dealing with fires affecting livestock (1). In this column, Dr. Andrew MacLeod relates his experiences as both a food animal veterinarian and a volunteer firefighter. Dr. Chris Riley offers advice on preparedness for barn and range fires from the perspective of someone deeply involved in planning for emergency situations.

Dr. Andrew MacLeod, Linwood Veterinary Services, Saint Clements, Ontario

The mission statement of our fire service is to:

i. preserve human life,
ii. save property, and
iii. minimize environmental impact

One way of achieving this is through prevention. Homeowners and school-aged children will be familiar with fire prevention activities. Because extinguishing barn fires requires substantial resources and because overall losses are often substantial, many rural departments are developing and delivering fire prevention programs aimed at reducing the incidence of barn fires (2).

The allocation of resources, assumption of risk, and prioritization of actions during a fire or rescue event are applied using this mission as a template. Animals are considered as property. It is quite possible that fire departments may not be focused as intently on animal rescue or be willing to place people in vulnerable situations to save animals. Farmers or pet owners may not agree. There are 2 main reasons why fire departments take this approach. First, firefighting is inherently dangerous; most fire departments balance the risks needed to achieve a benefit (*e.g.*, risk a little if you can only save a little; to risk a lot, a lot must potentially be saved). Second, many fire departments lack sufficient training in animal handling or technical rescue of animals. Although specialized training courses for animal handling and rescue are available, they are expensive, time-consuming, and can be difficult for firefighting personnel to access. In addition, animal rescue often requires fire departments to own or have access to specialized equipment.

During a barn fire, there is a potential for many animals to be released at once. This would require enough people, trailers, and potentially handling facilities (real or improvised) to corral and eventually transport the animals away from the fire to interim facilities. Most farmers likely won't have a pre-planned strategy for executing such a massive evacuation. Having a basic plan and contact information for people with livestock handling skills and equipment such as portable panels and trailers are useful should an emergency evacuation of livestock become necessary (3).

Dr. Chris Riley, Department of Clinical Studies, University of Guelph, Guelph, Ontario

Part 1 of this Food Animal Matters topic (1) shared some sobering experiences from our colleagues and their clients. Such experiences are traumatizing for farmers and veterinarians. Sharing them can lead to retraumatization (4). Disastrous events can and do happen on farms and ranches, so it is essential to recognize the need for preparation and prevention (planning). Equally important is the role of the attending veterinarian in supporting themselves and their

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clients during (response) and after (recovery) such traumatic events. I will further elaborate on some concepts arising from the experiences shared by Drs. McLeod and Wasilow (1).

Prevention and preparation advice for your clients

Barn fires. New barn construction should utilize less flammable building materials and select interior finishes that are less likely to produce toxic smoke, which is a common cause of death in fires. Strategically located firewalls reduce the speed of the spread of fire. Barns should have at least 2 animal exits that can be opened from the outside and, ideally, open to holding pens or containment paddocks at > 30 m from the building. Doorways and aisles should be kept free of vehicles and other obstacles. More valuable animals should be housed close to these exits, where they may be more likely to be rescued.

Farmers are multi-talented and skilled "contractors." Nevertheless, electrical, plumbing, heating and ventilation systems should be professionally designed and installed to reduce fire risk (5). Electrical inspection should be conducted bi-annually to ensure there are no exposed wires due to damage by vermin and all electrical equipment is in good repair (6). Faulty wiring should be replaced rather than "patched up." Electrical fires are a particular risk in older cattle barns as electrical wire sheathing degrades (7). Only industrial appliances should be used in the barn (5). Lightning rods may prevent fires in areas prone to lightning strikes.

Many barns store hay in areas above or adjacent to livestock. This has practical handling advantages but poses a substantial risk of burn injuries and smoke inhalation. Locate combustible feeds and bedding separately from the animals.

Fire extinguishers should be located throughout the barn in areas that are easy to access near the entry and exit points of the barn. Ensure that all staff are trained to operate them. Extinguishers should be inspected annually and serviced regularly according to local regulations. Ideally, new barns should be designed with a fire detection and suppression system suitable for livestock facilities (6). In some cases, these may be retrofitted. A separate water source not dependent on barn plumbing should be located near the barn so that the fire can be fought using portable pumps and hoses before the arrival of the fire department. The fire department may also use these when on scene. Local water sources have been used to save many barns and homes.

Wildfires. Barn fires affect individual farms; wildfires threaten entire communities. The veterinarian's role is often limited to providing first aid to animals during the incident and supporting survivors and their owners. Veterinarians should be familiar with the local emergency management agency; this may vary depending on the scale of the wildfire.

Farmers should seek advice from their local fire authority on the size and design of fire breaks and other aspects of wildfire readiness around their property, especially in regions at risk of wildfires. The same applies to veterinarians and veterinary clinics in at-risk zones. The loss of the Jasper Veterinary Clinic during the summer wildfires in Alberta is a sober reminder of the risk we share with farming families. As in barn construction, consideration should be given to the flammability of construction materials. Ventilation outlets/inlets and windows facing prevailing winds should be closeable and possibly shuttered to counter the intense radiant heat of wildfires. Embers often carry fire into buildings, so eaves should be closed. Ideally, a sprinkler system should be installed in the eaves of buildings in high wildfire-risk zones. Gas-powered pumps in shielded locations allow for continued operation during power failures. A separate backup water source should be available. Fire-resistant hoses are recommended.

Pastures should not be allowed to become overgrown or mature to the point of providing combustible material that speeds the fire across pastures. The layout of pastures should be such that it is easy to corral animals down the corridors that give access to the safety of a shelter, possibly a large open water source, or to a loading chute for evacuation. Turning cattle onto roadways poses a safety hazard to emergency responders seeking to bring in resources to fight the fire.

Before a wildfire emergency and if your client plans to evacuate livestock, they must identify which animals to save, how many they can transport to safety, the means of evacuation, to where they will be transported, and how they will be fed and watered (8). Biosecurity is also a consideration.

Planning

Barn fires. The prevention and preparation steps noted constitute the first steps in planning. Health and safety planning is a key part of successful farm management and should include plans that anticipate possible adverse event scenarios. Performing a paper-based exercise with your clients to develop a fire safety and response plan is advised. Aim to ensure human safety and optimal animal welfare outcomes.

Each person's role in the plan should be clearly identified before a fire. Examples of these roles include organizing a water pump and hoses for the fire, evacuating the animals from the barn if it is safe, appointing someone responsible for the safety of the people on-site, and providing first aid if needed. It is important to have a communication strategy that includes notification of the local emergency response agencies. A good plan also discusses what personal protective equipment should be kept onsite and the firefighting resources available. You may discuss the process of triage, expected injuries such as smoke inhalation and burns, and your general approach to determining an animal's likelihood of survival and productivity based on injury patterns. The importance of this is that it prepares the farmer for the possible mental trauma that comes with these situations. A mock exercise should be performed once a plan has been agreed upon. This exercise identifies any weaknesses or overlooked items in the plan and familiarizes those involved with the roles they will perform in the event of a barn fire. The post-exercise discussion should be frank and nonjudgmental.

Wildfires. Farmers, ranchers, and veterinarians are resourceful and adaptable. However, wildfires often go beyond the resources available and may involve losing property, animals, and people. Notwithstanding the previous suggestions, wildfires are usually managed by emergency response agencies governed by provincial statutes and municipal regulations. Farmers, ranchers, and veterinarians can best support the community response by participating in emergency management and incident management system training. These may provide opportunities to participate in a multiagency response and allow for controlled entry under the authority of the incident commander to attend to animal welfare in the controlled zone. Alternatively, veterinarians may work as part of a team to establish shelters for displaced animals and provide care and treatment for evacuated animals. How to establish such a shelter is beyond the scope of this article; however, some general principles, in addition to those related to veterinary care, include site security, proper animal identification, control of access to the evacuated animals, biosecurity, volunteer management and logistics (9).

Response

In the previous column (1), Dr. Wasilow provided excellent advice that does not require repeating. However, a brief mention of triage as a multi-casualty burn incident is appended to her advice. Humane euthanasia should be considered for livestock with the following injuries (10,11):

- any animal moribund or unable to rise
- animals in severe respiratory distress due to smoke inhalation
- 50% or more of the body's surface area is affected
- the eyes, the muzzle or mouth are burnt, preventing vision, impairing breathing, or ingestion
- burns to the hooves and lower limbs
- burns to the udder or penis and scrotum
- burns of the anus and vulva

Burnt livestock that are alert, ambulatory and able to drink and eat should be provided with veterinary care if feed, labor, and facilities are available, and the owner is physically and emotionally capable. Those with more than 15 to 20% of the body's surface area affected require intensive veterinary care at considerable expense (10,12).

Recovery

Property and livestock losses are consequences of both barn fires and wildfires. In both situations, the support of the community, farming and financial sectors is critical. As related by Dr. McLeod, psychosocial trauma and compromised mental well-being are part of the journey, and have only recently been recognized as an essential part of response management during the recovery phase in incidents involving animals (4). Compromised mental health and illness encompass a range of disorders from sleeplessness to post-traumatic stress disorder (Riley *et al.* unpublished data). Therefore, it is strongly recommended that following fire incidents that involve the death or injury of livestock veterinarians assist their clients in identifying appropriate support resources such as those provided by the National Farmer Mental Health Alliance (https://nfmha.ca/). Veterinarians should seek opportunities for training in mental health awareness, response, and support for themselves and their employees. The Canadian Veterinary Medical Association provides a sponsored course called the Working Mind from the Mental Health Commission of Canada (https://openingminds.org/training/twm/).

REFERENCES

1. Tremblay R. Food Animal Matters: Fire. Can Vet J 2024;65:1318–1320.
2. Barn fires in Ontario. 2024. Ontario Ministry of Agriculture, Food and Agribusiness and Ministry of Rural Affairs. Available

from: https://www.ontario.ca/page/barn-fires-ontario. Last accessed February 10, 2025.
3. Enhancing Emergency Management in Canada's Livestock & Poultry Industry. 2023. Animal Health Canada. Available from: https://animalhealth.ca/. Last accessed February 10, 2025.
4. Vroegindewey G. Veterinary behavioural health issues associated with disaster response. Am J Emerg Med 2021;10.47389/36: 78–84.
5. Barn fire prevention tool. 2024. Equine Guelph — The Horse Portal. Available form: https://thehorseportal.ca/healthcare-tools/barn-fire-prevention-tool/. Last accessed February 10, 2025.
6. Robertson A, McDonald D, Ward D, eds. Reducing the Risk of Fire on Your Farm. Ministry of Agriculture, Food and Rural Affairs, Toronto, 2011. Available from: https://www.ontario.ca/page/publication-837-reducing-risk-fire-your-farm. Last accessed February 10, 2025.
7. Kim DH, Yoo S-O, Kim SC, Hwang DK. Analysis of thermal characteristics of electrical wiring for load groups in cattle barns. Ind Health 2015;53:505–510.
8. Species Evacuation and Transport Guide. 2014. National Alliance of State Animal and Agricultural Emergency Programs. Available from: https://www.thenasaaep.com/_files/ugd/8b3e05_4757683ce32a4a4ab2c404820bebbe4f.pdf. Last accessed February 10, 2025.
9. Current Best Practices in Animal Emergency Management Mass Care and Sheltering. 2023. National Alliance of State Animal and Agricultural Emergency Programs. Available from: https://www.thenasaaep.com/_files/ugd/01f302_2308991f4c2848708b340258db187c1f.pdf. Last accessed February 10, 2025.
10. Duckett WM. Acute care of burn victims. J Eq Vet Sci 1995;15: 157–159.
11. Assessing bush fire burns in livestock. 2018 NSW Government Department of Primary Industries. Available from: https://www.dpi.nsw.gov.au/__data/assets/pdf_file/0007/96811/Assessing-bushfire-burns-in-livestock.pdf. Last accessed February 10, 2025.
12. Herbert EW. Findings and strategies for treating horses injured in open range fires. Eq Vet Educ 2018;30:177–186.

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Context, connections, and care: Towards animal healthcare nested within our shared planetary home

Margot W. Parkes

When faced with daunting, open-ended questions, my inclination is to return to what I refer to as "first principles," or what are perhaps better described as patterns of ideas that have consistently offered a useful point of reference in the past. This is my response to the important, but challenging question of "how to encourage animal health professionals to make useful contributions when faced with complex challenges that span health, ecosystems, and society?"

My offering here distills ideas developed in relation to "Working Together for WHOLE Systems," in which the WHOLE refers to approaching 'Well-being and Health Oriented to Living systems and Equity,' as part of a larger text focused on Animals, Health and Society (see Reference 1, especially Box 5.2). For this contribution, I focus especially on the 3 interrelated imperatives of *context, connections,* and *care*.

CONTEXT

In the work I am involved with, "context matters." I view this to be the case for any coherent health practice, whether considering the health of humans, animals, places, or the planet. The idea of context is not only important for the animals (and humans and places), you may be interacting with or serve, but also you and what you bring, as a health professional. I encourage any health professional to consider their positionality as part of a reciprocal contextual relationship that also determines how you will approach the connections and care that will enrich your work. Context also offers a necessary step toward a WHOLE Systems perspective, strengthening your own capacity to focus on well-being and health while (also) orienting to the wider context of living systems and equity.

Accordingly, my context(s) as I write this piece matters. I am writing as a woman whose heritage is of English, Irish, and Scottish descent, born in Aotearoa/New Zealand in the east coast town of Timaru in the territory of Kāi Tahu Iwi (iwi is tribe in Maori). A foundation for my orientation to health is my training and work as a medical doctor in Aotearoa/NZ and subsequent work and experience across the fields of human ecology, public health, and ecosystem approaches to health (ecohealth), with ongoing alignments to approaches to One Health and planetary health. Alongside this, my orientation to health is occurring within, and deeply related to the living systems (and ecosystems) we depend on, and especially informed by my orientation to rivers and watersheds (2). My ongoing work and learning are influenced by over 5 decades of (re)orienting to, visiting, and occasionally living at Taieri Mouth in the southeast of Aotearoa/NZ (where the Taieri River meets the Pacific Ocean). In addition, for the past decade, I have also been privileged to mostly live, work, learn, play, love, mourn, and be filled with wonder, within the unceded territory of the Lheidli T'enneh which, in Dakelh language, describes 'the people of the confluence of the two rivers' (Lheidli T'enneh First Nation 2020), referring to the Nechako and the Lhtakoh (or Fraser) rivers that flow together Prince George, British Columbia, Canada.

Introducing myself in this way, reveals my inclination to consider rivers as a form of eco-social 'elder,' in ways that continue to have profound influence on my work, including how I understand health-in-context. For nearly 3 decades my approach to health, has been tightly coupled with how I understand ecosystems and equity dynamics within the context, or setting, of watersheds (3). The watershed context invites a combined attention to both ecosystems

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and social systems, drawing attention to equity and power dynamics (including distribution of decision-making power, authority and influence), in ways that is closely related to the theme of connections.

I encourage all animal health professionals to consider how your work is influenced by your own positionality, your own wider context, and how these in turn influence how you see the contexts within which the health is emerging, for the animals you are interacting with, and the systems that health is embedded in.

CONNECTIONS

An intentional appreciation of context raises questions about various axes of connections that influence health. As noted earlier, these connections are especially important for an understanding of well-being and health that is oriented to living systems and equity. For some, the full scope of relationships and interactions involved with 'systems connections' can be a little disorienting in their complexity. To avoid feeling overwhelmed, and to help me navigate the enrichened perspectives I have gained from focusing on connections, I have found it useful and deeply informative, to return to **3 axes of connections** that each deepen my understanding of health. Some may see resonance with familiar patterns in the clinical method. Others may see these connections as just a way to understand context. I find asking questions about these connections re-assuring, opening ways to find patterns that can deepen approaches to health from whatever perspective you are bringing. Connections can also be seen as part of a wider effort to strengthen capacity for transformative, inclusive, and integrative approaches to learning (and re-learning), that are especially relevant given the threats to our shared planetary home (4).

Nested connections. What parts of the health system am I interested in? If I am caring for a particular animal, do I have what I need to grasp health of this animal as part of a nested system (4,5), with relevant insights to be gained from the scale below (the diseased organ?) *and* the scale above (the herd?). Am I willing to go beyond this, further 'upstream' in the ecological hierarchy? Am I able to imagine health within a herd, nested within a farm, within a watershed, within a bioregion, within our planetary home? At what point do I lose focus? What happens when I identify blind spots? For example, when a focus on planetary shifts or global climate change, shifts attention away from specific health dynamics within watersheds, and the global impacts on the living systems of lands and waters that all animals depend on. How do these nested connections deepen my appreciation of health?

Temporal connections *(past, present, future)*. What timescales am I paying attention to, when seeking to understand the health of the animal for which I am caring? What of the past for this animal, and its predecessors? Or of the lands, waters, living systems they depend on? What are the implications of my decisions beyond the immediate timeframe for this animal and the nested systems it exists within. What are the lines of connections and pathways of influence among the past, present, and future health of this animal, and the possible future health of other animals, humans, species? What are the points of connections for this animal and my own future?

Iterative, reflective connections. How much does your practice reflect the reality that understanding health and well-being is iterative? Does your practice reflect the ways that future learning will create opportunities to deepen understanding of matters missed earlier? Are you open to celebrating various, connected phases of work that recognize the new insights possible across short-, medium-, and long-term practices? How do you maintain and retain curiosity about these iterative connections and your own emergence as a learner, practitioner, and healthcare provider?

I encourage all animal health professionals to remain curious about the patterns of connections in their work, and how greater attention to these might enhance their healthcare practice, and/or the ways they care about the ways health is nested within our shared planetary home.

CARE

Being curious about context and connections is a pathway to invigorate your healthcare practice, as well as the ways in which you ***care about health.*** Caring about health is, of course, different than healthcare. Asking questions about context and connections can deepen the care you have about your own health, alongside the health of those you are charged with caring for and, in addition, your care about the state of the (animal) healthcare you are engaged in. This work isn't easy and, reiterating the comments above, a key way to ensure to express your commitment

to care and be positive is to ***commit to asking good questions*** (6,7). The quality and scope of the questions you ask is likely to determine the extent to which your work and practice will be informed by context, connections, and your capacity to change in ways that will improve present and future (health)care.

Asking good questions about 'Well-being and Health Oriented to Living Systems and Equity' often takes us beyond the familiar, and into new terrains of knowledge, engagement, action, and change (4). This is when attention to WHOLE systems also requires attention to 'Who and How are we Open to Listen, and Engage/exchange?' Care about WHOLE Systems, is often determined by the extent we are willing to **be open to refer or collaborate or both.** Being prepared for and open to teamwork is an expression of care and raises a further set of questions. As a particular type of healthcare provider, what determines your willingness to embrace working with others, across specialties, disciplines, sectors or partners (perhaps even species) to improve the quality of care? Does your care for health, and those you are caring for, lead you to recognize, respect, and learn to work with, and bridge across, other knowledge and approaches?

I encourage all animal health professionals to devise and revise their own list of questions that will create opportunities to deepen their attention to interrelationships among context, connections, and health, in ways that will enhance their own health practice, and to enrich how they care about health, for themselves and other species, both now and into our shared future.

REFERENCES

1. Parkes MW. Working together for WHOLE systems: Approaching Well-being and Health, while Oriented to Living-systems and Equity (Chapter 5). In: Stephen C, ed., Animals, Health and Society: Health Promotion, Harm Reduction, and Health Equity in a One Health World. Boca Raton, Florida: CRC Press, 2021:71–94.
2. Parkes MW. River conversations: A confluence of lessons and emergence from the Taieri River and the Nechako River. River Res Appl 2022;38:443–452.
3. Jenkins A, Capon A, Negin J, *et al*. Watersheds in planetary health research and action. Lancet Planet Health 2018;2: e510–e511.
4. Redvers N, Faerron Guzmán CA, Parkes MW. Towards an educational praxis for planetary health: A call for transformative, inclusive, and integrative approaches for learning and relearning in the Anthropocene. Lancet Planet Health 2023;7:e77–e85.
5. Parkes MW, Horwitz P. Ecology and ecosystems as foundational for health. Chapter 2. In: Frumkin H, ed., Environmental Health: From Global to Local (3rd ed.). Hoboken, New Jersey: Jossey-Bass, 2016.
6. Wahl DC. Designing Regenerative Cultures. Triarchy Press, 2016. Available from: https://www.triarchypress.net/drc.html#. Last accessed February 12, 2025.
7. Kania J, Kramer J, Senge P. The Water of Systems Change. 2028. Available from: https://www.fsg.org/resource/water_of_systems_change/. Last accessed February 12, 2025.

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