CASE REPORT
RAPPORT DE CAS

Clinical progression of a case of toe-tip necrosis syndrome in a feedlot steer

Désirée Gellatly,
Karen S. Schwartzkopf-Genswein,
Wiolene M. Nordi, Tawni Silver,
Murray Jelinski

ABSTRACT

A fractious feedlot steer was diagnosed with hind-limb lameness at 19 d on-feed. Clinical examination (Week 0) revealed white line separation of both lateral digits of both hind feet. Debridement confirmed toe-tip necrosis syndrome and the steer was treated with oxytetracycline (20 mg/kg, SC) and meloxicam (0.10 mg/kg, SC). Radiographs revealed osteolysis of P3 bones of both lateral digits, and by Week 4 the medial claw of the right hind foot was similarly affected. Clinical and radiographic examinations were conducted over a 25-week period. Rectal temperature, body weight (BW), cortisol (salivary and hair), haptoglobin, and complete blood (cell) counts were obtained over a 22-week period. New horn growth covered the debrided sole defects by Week 7, and radiographs confirmed remodelling of the P3 bones. At slaughter (Week 25), both hind feet appeared normal except for the lateral digits being longer than the medial digits. Radiographs indicated permanent changes in the P3 bones. Although this animal had osteolysis in 3 of 4 hind-limb digits, it was successfully treated with antimicrobial therapy and debridement on Week 0 followed by a second debridement in Week 2. Early intervention and debriding are central to preventing other sequelae such as deep digital sepsis.

Key clinical message:

A feedlot steer diagnosed with toe-tip necrosis syndrome, including osteolysis of 3 of 4 hind-limb digits, was successfully treated with debridement and antimicrobial therapy. Radiographic examinations confirmed remodeling of the distal phalanges (P3) and absence of active infection 25 wk after treatment. This case underscores that feedlot animals with osteolysis of multiple P3 bones can be treated successfully using debridement and antimicrobials.

RÉSUMÉ

Évolution clinique d'un cas de syndrome de nécrose de l'extrémité des orteils chez un bœuf de boucherie

Un bœuf de boucherie rebelle a reçu un diagnostic de boiterie des membres postérieurs après 19 jours d'alimentation. L'examen clinique (semaine 0) a révélé une séparation de la ligne blanche des deux doigts latéraux des deux pieds postérieurs. Le débridement a confirmé le syndrome de nécrose de l'extrémité des orteils et le bœuf a été traité avec de l'oxytétracycline (20 mg/kg, SC) et du méloxicam (0,10 mg/kg, SC). Les radiographies ont révélé une ostéolyse des os P3 des deux doigts latéraux et, à la semaine 4, l'onglon médial du pied postérieur droit était également affectée. Des examens cliniques et radiographiques ont

Olds College of Agriculture & Technology, 4500 50 Street, Olds, Alberta T4H 1R6 (Gellatly); Agriculture and Agri-Food Canada, Lethbridge Research Centre, 5403 1 Avenue S, Lethbridge, Alberta T1J 4B1 (Schwartzkopf-Genswein); University of São Paulo, Av. Pádua Dias 11, Piracicaba, São Paulo State 13418-900, Brazil (Nordi); Department of Large Animal Clinical Sciences, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan T2N 4N1 (Silver, Jelinski).

Address all correspondence to Murray Jelinski; email: murray.jelinski@usask.ca.

été réalisés sur une période de 25 semaines. La température rectale, le poids corporel (PC), le cortisol (salivaire et pileux), l'haptoglobine et une numération globulaire complète ont été mesurés sur une période de 22 semaines. La croissance de nouvelle corne recouvrait les défauts de la sole débridée dès la semaine 7, et les radiographies ont confirmé le remodelage des os P3. À l'abattage (semaine 25), les 2 pieds postérieurs semblaient normaux, à l'exception des doigts latéraux, plus longs que les doigts médiaux. Les radiographies ont montré des modifications permanentes des os P3. Bien que cet animal présentait une ostéolyse de 3 des 4 doigts des membres postérieurs, il a été traité avec succès par un traitement antimicrobien et un débridement à la semaine 0, suivi d'un second débridement à la semaine 2. Une intervention et un débridement précoces sont essentiels pour prévenir d'autres séquelles, telles que le sepsis digital profond.

Message clinique clé :

Un bœuf de boucherie chez qui un syndrome de nécrose de l'extrémité des orteils, incluant une ostéolyse de 3 des 4 doigts des membres postérieurs, a été traité avec succès par débridement et traitement antimicrobien. Les examens radiographiques ont confirmé le remodelage des phalanges distales (P3) et l'absence d'infection active 25 semaines après le traitement. Ce cas souligne que les animaux d'élevage présentant une ostéolyse de plusieurs os P3 peuvent être traités avec succès à l'aide d'un débridement et d'antimicrobiens.

(Traduit par Dr Serge Messier)

Can Vet J 2025;66:514–522

The first documented report of toe-tip necrosis syndrome (TTNS) involved an outbreak of "transit-related lameness" in a group of Jersey heifers purchased at a New Zealand auction. Over a 3-week period, animals developed a hind-limb lameness. The primary lesion involved separation of laminae and subsequent necrosis/abscess formation of affected claws. Mechanical trauma and weight-bearing forces placed on the claws during transportation were thought to have resulted in separation of the laminae of the distal phalanx (1). Shortly thereafter, Sick *et al* (2) described a similar clinical scenario involving beef cattle housed in feedyards in Iowa (USA). Depending on the stage of the disease, debridement of the toe ("nipping") revealed a hematoma, an abscess, or black necrotic tissue. The authors speculated that improper handling of hyperexcitable animals resulted in abrasion to the apical sole of the hind claws, resulting in white line separation (WLS).

Although the epidemiology and clinical findings of TTNS have been well described (3–5), there has been confusion regarding the disease's nomenclature, with multiple names suggested: toe abscess, toe ulcer, toe necrosis, toe-tip necrosis, P3 necrosis, and *apicus necrotica*. The International Committee for Animal Recording (ICAR) (6) attempted to address this issue by defining "toe necrosis" as "necrosis of the tip of the toe with affection of bone tissue," a "toe ulcer" as a "circumscribed purulent and necrotizing inflammation of the corium of the claws at the toe," and "white line disease" as "separation of the white line with or without purulent exudation" (6). In practice, feedlot veterinary practitioners see all 3 diseases (toe necrosis, toe ulcers, and WLS) as a continuum of a single disease; hence, we prefer the term "toe-tip necrosis syndrome (TTNS)," because it encompasses these manifestations and myriad sequelae.

Although the etiopathogenesis of TTNS is not fully understood, the "abrasion theory" postulates that excessive wear of the solar horn results in separation along the apical portion of the white line. The excessive wear is assumed to be the inciting cause of the disease (7). Outbreaks cluster by pen, often involve fractious animals, and mostly occur within days to weeks of arrival to the feedlot (7). Wet pasture and pen conditions may result in softer claw horn tissue, leading to increased horn wear (8). Trace mineral deficiencies have also been implicated as a potential risk factor (7). In addition, overcrowding and improper animal handling causes animals to become agitated and push against the animals in front of them. This results in the abrasion of the hind feet, especially on coarse flooring surfaces such as concrete, which often become wet with urine and manure. Abrading the apical portion of the toe damages the white line, allowing bacteria to colonize the claw, with the secretion of hydrolytic enzymes degrading the laminar junctions. Ambulation leads to cycles of loading and unloading, which has been replicated *in vitro* and shown to

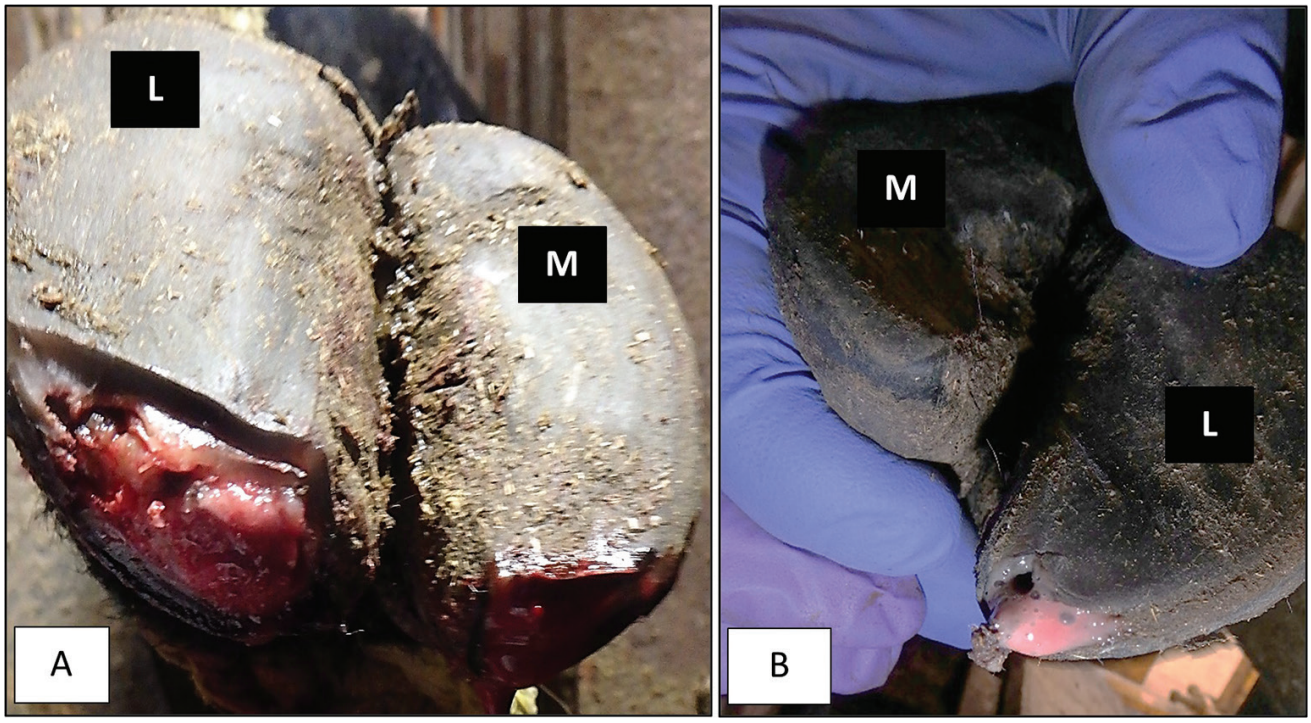

FIGURE 1. Photographic images taken at the initial clinical examination (Week 0) following debridement of the left (A) hind limb and before debridement of the right (B) hind limb of an Angus cross steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta.

L — Lateral claw; M — Medial claw.

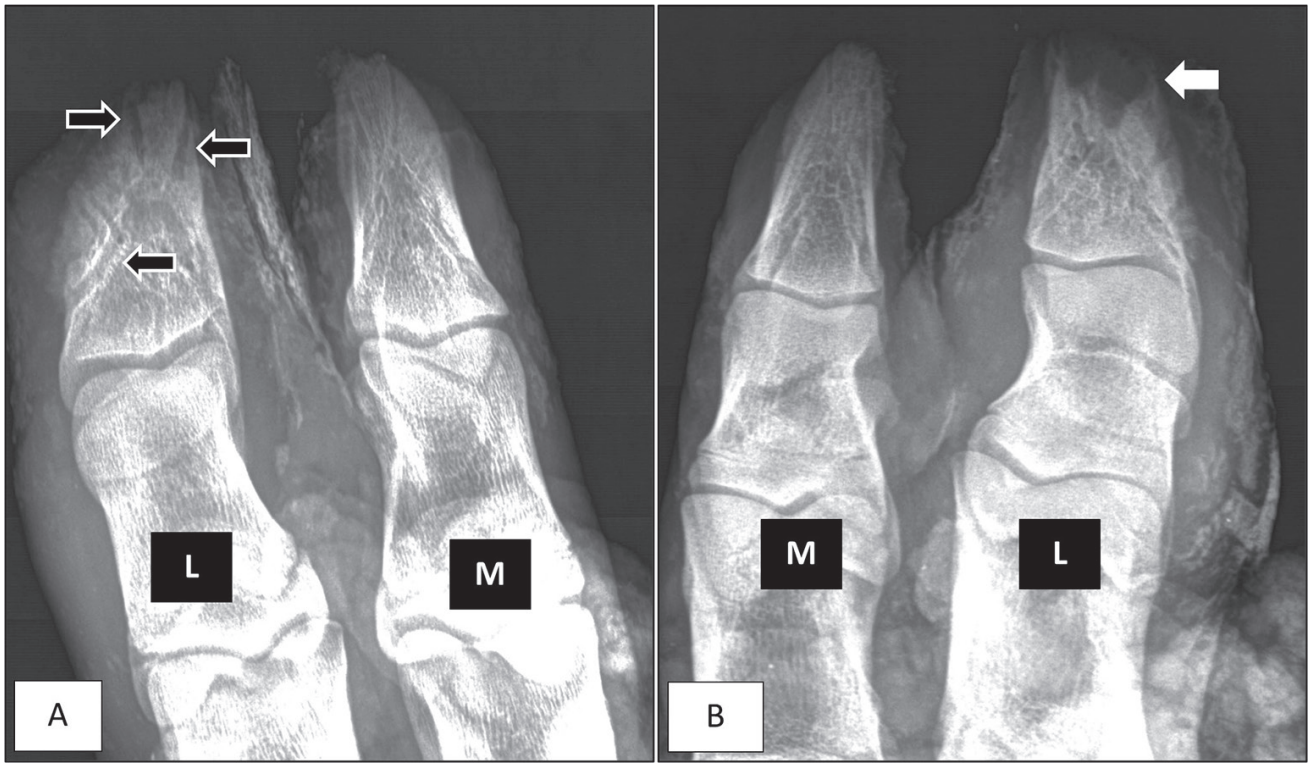

FIGURE 2. Radiographic images taken 1 d after the initial examination (Week 0) following debridement of the left (A) and right (B) hind limbs of an Angus cross steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta. Black arrows indicate the widening of vascular channels, and the white arrow points to the occurrence of osteolysis of the P3 bone.

L — Lateral claw; M — Medial claw.

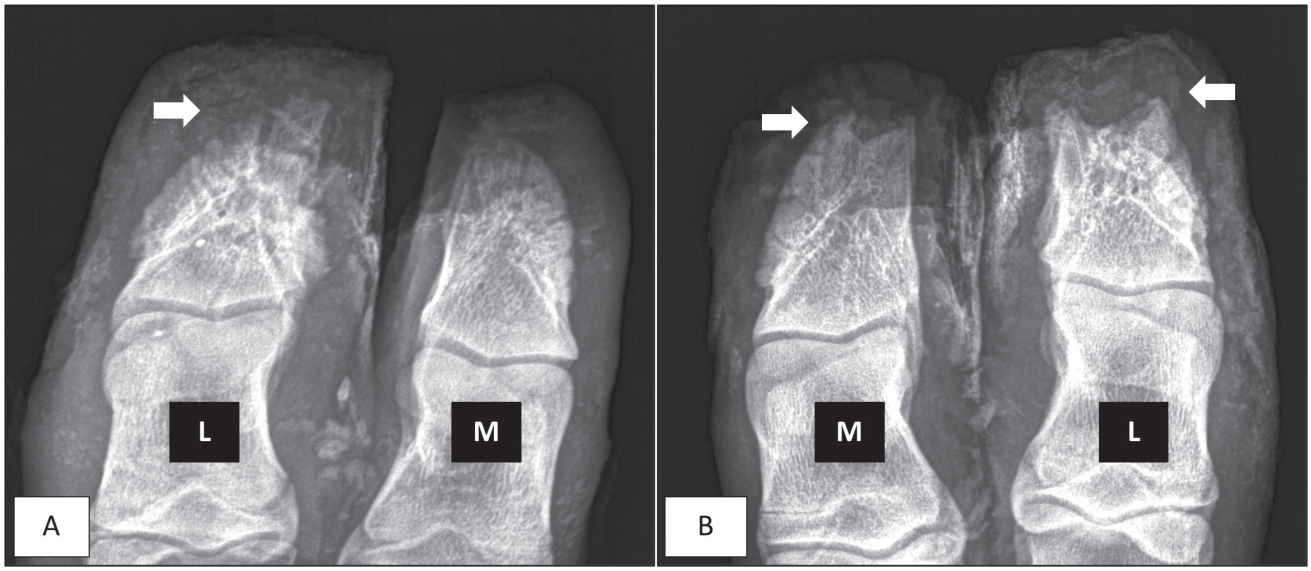

FIGURE 3. Radiographic images taken 4 wk (Week 4) following debridement of the left (A) and right (B) hind limbs of an Angus cross steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta. White arrows point to the occurrence of osteolysis of the P3 bones.
L — Lateral claw; M — Medial claw.

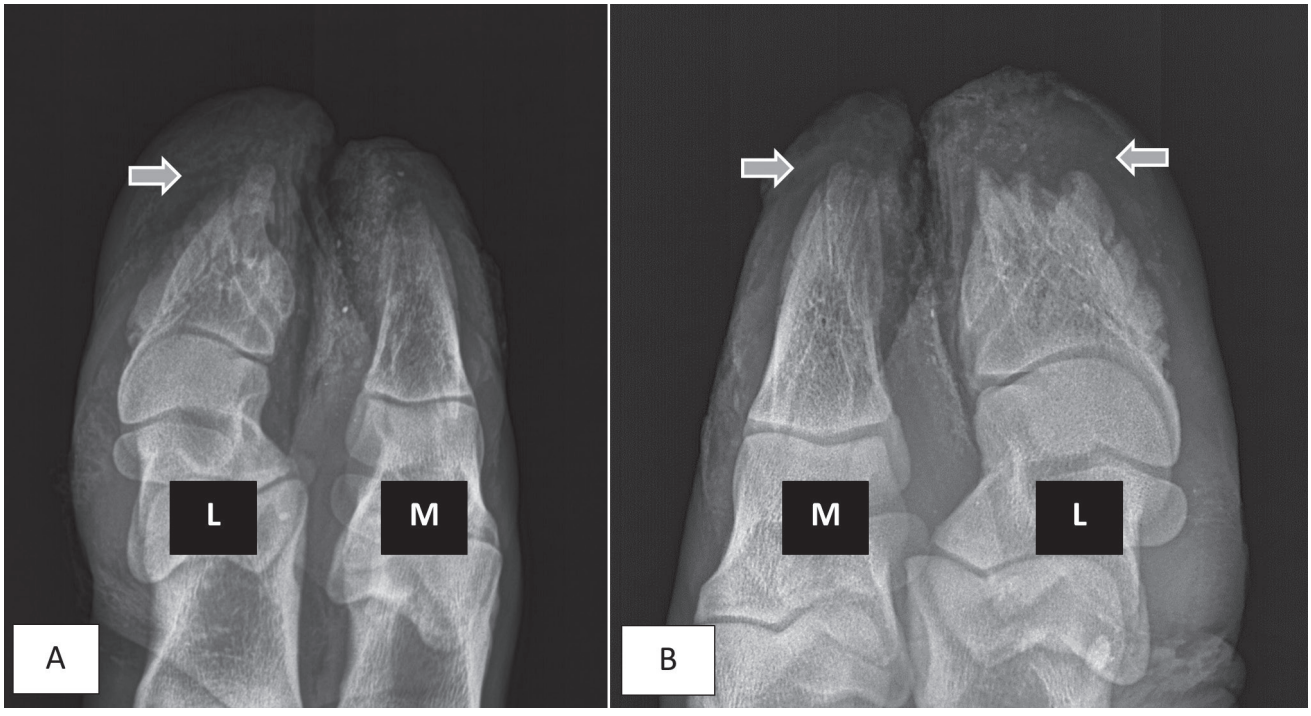

FIGURE 4. Radiographic images taken 7 wk (Week 7) following debridement of the left (A) and right (B) hind limbs of an Angus cross steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta. Gray arrows point to the occurrence of reparative remodeling of the P3 bones.
L — Lateral claw; M — Medial claw.

contribute to degradation of the white line (9). Under loading conditions, the white line gapes open, allowing manure and other organic material to become impacted within the white line; *Trueperella pyogenes* and *Escherichia coli* are commonly recovered from diseased feet (5). The lateral claws are more commonly affected due to the biomechanics of walking, in which most of the load on the hind feet is supported by the lateral claws (10). Clinical lameness likely manifests once the infection penetrates the highly vascularized and innervated corium, resulting in a toe abscess. Disease progression leads to osteolysis (11) of the distal phalanx (toe necrosis), with the infection potentially

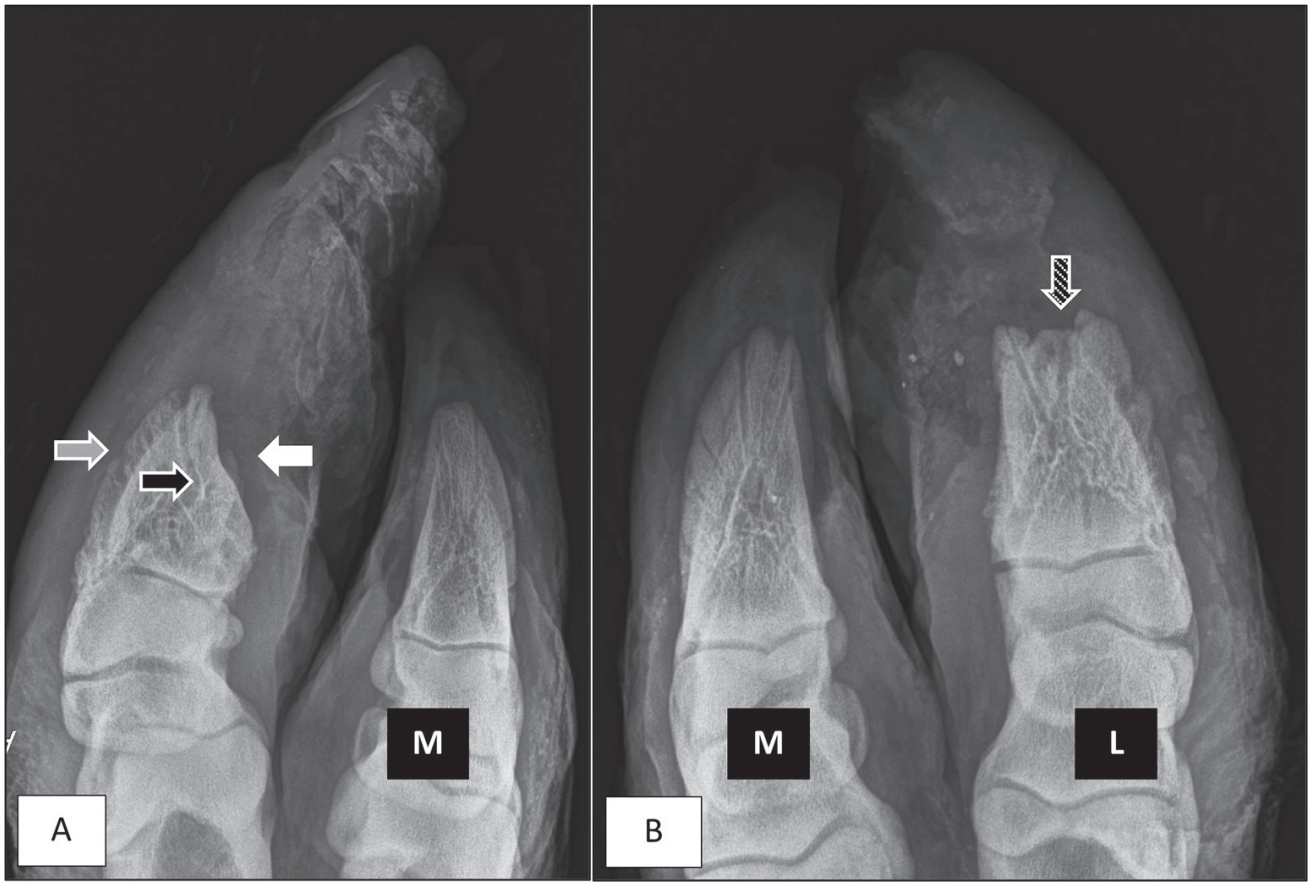

FIGURE 5. Radiographic images, taken post-slaughter, of the left (A) and right (B) hind limbs of an Angus cross steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta. The gray arrow indicates progressive healing of P3 osteitis, the black arrow points to the improved appearance of vascular channels, the white arrow highlights a stable cortical defect in the left lateral claw, and the vertical (diagonal striped) arrow indicates the formation of new bone in the right lateral claw.

L — Lateral claw; M — Medial claw.

spreading to other soft tissues and P2. In some cases, an ascending infection of the hind limb results in tenosynovitis, cellulitis, and myositis. Hematogenous spread of bacteria to more distant organs such as the liver, lungs, and kidneys is not uncommon and results in death (2).

In North American feedlots, the condition may go undetected until the stage of P3 necrosis. Furthermore, logistics and a lack of facilities are impediments to surgical intervention. Cattle given a putative diagnosis of TTNS are typically administered systemic antimicrobials and may have the toe tip debrided ("tipped") to confirm the diagnosis and facilitate drainage. A poor prognosis is often given to cattle with moderate to severe TTNS, resulting in euthanasia or cattle being salvaged for slaughter ("railed").

The study objectives were to describe the clinical progression of TTNS in a feedlot steer and to document lesion healing following debridement and antimicrobial therapy.

CASE DESCRIPTION

A fractious, 181.4-kilogram Angus cross feedlot steer that was 19 d on-feed (DOF) was identified by feedlot pen-checkers as exhibiting a hind-limb lameness with no obvious signs of swelling or injury. These clinical signs were consistent with TTNS. The animal was removed from its home pen and placed in a holding pen for researchers to examine. The apex of each of the 4 hind claws was trimmed using a set of hoof-nippers to determine if the horn tissue was healthy, or conversely to confirm if the claw had toe-tip necrosis. Over a 22-week period, serial clinical and radiographic examinations were conducted, along with blood, saliva, and hair sampling to monitor physiological parameters. Because the animal was hyperexcitable at handling, reliable lameness gait scores could not be obtained. The feet were obtained at slaughter (Week 25) and radiographed at the Western College of Veterinary Medicine (Saskatoon).

TABLE 1. Descriptive summary of body weight and physiological parameters obtained during the initial examination (Week 0) and at subsequent sampling weeks (from 1 to 22) for a crossbred Angus steer diagnosed with toe-tip necrosis syndrome while housed in a commercial feedlot in Alberta.

	Sampling week												
Item	0	1	2	3	4	5	7	9	11	13	16	19	22
Body weight, kg	181.4	190.5	208.7	222.3	231.3	249.5	239.5	271.7	312.5	326.6	371.9	417.3	462.9
Salivary cortisol, μg/dL	0.25	0.44	0.26	0.72	0.25	0.45	0.30	0.16	0.15	0.10	0.28	0.17	0.25
Hair cortisol, pg/mg	6.04	—	9.62	—	6.32	—	—	—	7.30	—	4.17	—	3.07
Rectal temperature, °C	39.4	39.8	40.3	40.1	40.2	40.0	41.0	39.3	39.4	39.6	40.0	39.4	39.5
Haptoglobin, mg/mL	1.74	0.10	0.11	0.07	0.10	1.10	0.42	0.06	0.08	0.07	0.02	0.05	0.09
RBC, 10^6/μL	8.5	8.8	8.4	8.8	8.7	8.6	8.8	7.6	8.5	8.9	8.9	9.6	9.4
WBC, 10^3/μL	13.2	13.7	13.7	14.6	10.3	13.1	11.5	14.0	9.1	8.9	11.4	13.5	11.4
M:L ratio	0.26	0.16	0.23	0.22	0.18	0.19	0.22	0.19	0.17	0.10	0.20	0.25	0.17
Platelets, /μL	558	611	437	403	417	363	362	381	326	336	283	265	250

M:L — Ratio between absolute counts of monocytes and lymphocytes; RBC — Red blood cell count; WBC — White blood cell count.
" — " Indicates no occurrence of sampling.

Hoof-testers did not elicit a pain response in the diseased claws, which was generally the case throughout the study. At the time of initial clinical examination (Week 0), both left and right lateral claws had evidence of WLS. Debriding the claw horn revealed necrosis of the horn tissue of the left lateral claw (Figure 1 A) and a purulent, bloody discharge of the right lateral claw (Figure 1 B). No attempt was made to resect the diseased P3 bones. In addition to undergoing debriding of the necrotic horn tissue, the steer was administered oxytetracycline (20 mg/kg) and meloxicam (0.10 mg/kg) SC. The claws were left unbandaged and the animal was placed in a hospital pen. Radiographs were obtained 1 d after the initial examination (Week 0) and on Weeks 2, 4, 7, 13, 19, 22, and post-slaughter (Week 25). Initial radiographs (Week 0) revealed signs of an aggressive primary bone disease characterized by widened vascular channels on the left lateral P3, suggesting osteitis, though infection was uncertain (Figure 2 A). In addition, osteolysis was observed in the lateral (4th) digit of the right hind limb (Figure 2 B), whereas the medial digits in both hind limbs appeared normal. On Week 2, treatment of the affected digits involved debridement of all necrotic horn tissue. By Week 4, both left (Figure 3 A) and right (Figure 3 B) medial claws had developed WLS, and radiographs revealed osteolysis of the medial P3 bone and progressive lysis of bilateral lateral digits. New sole formation was evident in all 4 claws by Week 7; however, the steer became febrile (41°C) and had lost 10 kg of BW since Week 5. On radiographs obtained on Week 7, affected P3 bones (Figures 4 A, B) showed signs of reparative remodeling. By Week 7, the steer was treated as a presumptive case of bovine respiratory disease (BRD) and administered SC injections of florfenicol (40 mg/kg BW) and flunixin meglumine (1.0 mg/kg BW).

By Week 22, clinical examination revealed that, aside from the lateral claws being longer than the medial claws, the claws appeared grossly normal. At slaughter (Week 25), radiographic findings demonstrated progressive healing of P3 osteitis, bilateral increased horn growth, and improved vascular channel appearance, along with a stable cortical defect of the left lateral claw (Figure 5 A) and new bone of the right lateral claw (Figure 5 B). The right medial digit at this point appeared normal.

This steer was part of a larger research project that involved monitoring rectal (body) temperature and physiological parameters: salivary cortisol, hair cortisol, haptoglobin, and complete blood (cell) counts (CBC) (Table 1). This study was approved by the Lethbridge Research and Development Centre's (Lethbridge, Alberta) Animal Care Committee (ACC #1817). Briefly, for the salivary cortisol, saliva was collected, stored, and analyzed as described by Meléndez *et al* (12). The inter-assay coefficient of variation (CV) was 17.1% and the intra-assay CV was 4.6%. Hair samples for cortisol concentration analysis were collected, processed, and analyzed as described by Moya *et al* (13). The inter-assay CV was 12.4% and the intra-assay CV was 5.9%. Blood samples for haptoglobin were collected, stored, and analyzed as described by Meléndez *et al* (14). The inter-assay CV was 22.0% and the intra-assay CV was calculated at 8.6%. Blood samples for CBC were collected as described by Meléndez *et al* (12). The CBC was done using a HemaTrueHematology Analyzer (Heska, Loveland, Colorado, USA), which provided red and white (WBC) blood (cell) counts, monocyte-to-lymphocyte ratio, and platelet counts.

DISCUSSION

This case was unique in that the animal developed osteolysis or osteomyelitis of the distal phalangeal bone (P3) in 3 of the 4 hind claws, with the 2 lateral claws most severely affected. Previous research indicates that the lateral claws are more commonly affected, as most of the load on the hind feet is supported by the lateral claws (10). Despite the severity of the disease, radiographs confirmed the clinical impression that the animal had fully recovered by the time of slaughter. Although aggressive surgical intervention such as resection of P3 has been highly effective (11), this requires a veterinarian trained to conduct this procedure. Furthermore, if >1 claw is affected, as in this case, claw amputation is not an option. Generally, lame feedlot cattle are administered antimicrobials and monitored for recovery.

The treatments described herein are within the scope of trained personnel, with the main challenge being restraint of the animal during debridement, which in this case was facilitated by securing a rope at the level of the fetlock and extending the leg directly behind the animal. Claw amputations can be done in a similar manner, but a tip-table enables much better restraint. It is important to highlight that, because TTNS frequently affects >1 claw (5,7,11), all claws must be examined before a block is applied or claw amputation contemplated. In our case, aftercare was uncomplicated, no topical antimicrobials or bandages were applied, and the animal was housed in a dirt-floor hospital pen. Segregation in hospital pens reduces competition at the feed bunk and simplifies handling for reassessments.

The use of radiographs to confirm P3 bone necrosis is not novel (7,11). However, to our knowledge, this is the first case report to document the progression of TTNS in a feedlot animal from diagnosis to slaughter, using clinical examinations, serial radiographs, and physiological parameters. The steer in this report had a clinical presentation similar to that in most TTNS cases. The steer developed a lower hind-limb lameness shortly after arrival to the feed yard, and there was no obvious swelling of the affected feet. This is a typical presentation unique to TTNS (2,7). The diagnosis can easily be confirmed by trimming the apex of the claws, with further debridement to remove all necrotic horn tissue. Aggressive debridement to the point of curettage of P3 may be required (11) and would also require the use of regional anesthesia. If clean, dry bedding is unavailable, then application of a bandage should be considered.

This case was consistent with previous reports that visual toe lesions are often poorly correlated to the underlying pathology (7,15). Identifying toe pathology such as WLS, with or without purulent material, is best viewed as the proverbial "tip of the iceberg," with more extensive pathology, such as osteolysis of the P3 bone, often occurring within the hoof (15). This animal was only 19 DOF when diagnosed, yet both lateral claws had developed obvious signs of osteolysis. These findings underscore how rapidly the disease can progress from a mild, early-stage lameness to a recommendation for euthanasia. Previous research demonstrated that 81% of animals diagnosed with TTNS within 5 DOF died within 15 d of treatment, with 65% euthanized due to intractable lameness (7). This highlights the importance of treating all TTNS cases as having potential to develop into deep digital sepsis, which encompasses a range of pathologies, including P3 osteitis, P2 osteomyelitis, sepsis of the distal interphalangeal joints, and tenosynovitis of the flexor tendon sheath (16).

The physiological parameters in the present case were largely unremarkable. Previous studies reported variable associations between stress, inflammatory biomarkers, and clinical outcomes (17–20). However, few studies have documented associations between the biomarkers used in this study and lameness in beef cattle. The steer described in this case was febrile (≥ 40°C) at 6 of the 13 time points when body temperature was measured. This was consistent with a previous study in which feedlot cattle diagnosed with toe necrosis were febrile, whereas those with other forms of lameness (foot rot, digital dermatitis, or laminitis) were afebrile (21). Given the animal's consistently excitable behavior during handling, the elevated body temperatures (aside from Week 7) were likely confounded with the typical increase seen in more excitable animals due to handling-related stress.

Salivary cortisol and haptoglobin concentrations, as well as platelet counts, were correlated with the disease. Hair cortisol and other CBC parameters showed slight variations across the sampling time points. Salivary cortisol increased over the first 3 wk of the study, when the animal was likely experiencing the most claw-related pain, and then tapered to basal levels by the end of the study. Basal serum cortisol concentrations in cattle range between 0.15 and 0.25 μg/dL, with peak concentrations between 0.60 and 2.0 μg/dL, depending on the stressor and animal (19). Haptoglobin concentration was greatest for Week 0 (1.74 mg/mL), suggesting that the steer had a marked inflammatory process occurring at the time of the initial assessment. Elevated blood haptoglobin concentration has been used to detect inflammation and disease in

cattle (17,22). The decrease in haptoglobin concentrations between Weeks 0 and 2 could be attributed to a reduction in inflammation following toe nipping/debridement and antimicrobial administration. Platelet counts were highest in the first 4 wk, likely related to infection and stress due to long-term pain (23). Basal platelet counts in cattle range between 160 and 800/μL of blood (23). Cattle diagnosed with toe-tip necrosis were reported to exhibit greater blood platelet and lymphocyte counts and lower WBC counts than those diagnosed with foot rot or injury (21).

It is noteworthy that the steer in this case developed BRD between Weeks 5 and 7. Haptoglobin increased dramatically at Week 5 and salivary cortisol increased to a lesser extent, whereas WBC and the monocyte-to-lymphocyte ratio were equivocal. Haptoglobin has been positively associated with BRD in calves (17,18). The steer also lost 10 kg BW between Weeks 5 and 7, which was the only period when weight loss was observed over the 22-week study period. Thus, haptoglobin concentrations, BW, and rectal body temperature were all consistent with a BRD diagnosis. Whether the second administration of antimicrobials at Week 7 contributed to the healing of the claws is unknown. However, the regrowth of the solar horn tissue, declining haptoglobin and salivary cortisol concentrations and platelet counts, and radiographic evidence of progressive healing of the P3 bones before the BRD event suggested that the Week 7 antimicrobials may not have contributed to the recovery from TTNS. This steer was finished at 300 DOF with a final BW of 646 kg.

This case highlights that feedlot animals with osteolysis of multiple claws can be treated successfully with low-cost antimicrobials (oxytetracycline) and debridement. In addition, debriding may need to be repeated, but the procedure is relatively simple and can be done by trained feedlot workers. Implementing low-stress handling techniques, particularly acclimating cattle upon feedlot arrival to reduce animals' stress levels on future handling occasions, along with improved flooring in handling facilities, such as sand with proper drainage, is crucial for reducing the incidence of toe-tip necrosis in feedlot cattle. This approach not only enhances animal welfare but also has significant economic benefits for the beef industry.

ACKNOWLEDGMENTS

We thank Wiseman Feeders Ltd. (Lethbridge, Alberta), for participating in the study; Ranch Docs Veterinary Services (Lethbridge, Alberta), for providing the field radiographs; and the Veterinary Medical Centre, Western College of Veterinary Medicine, for providing the terminal radiographs. We are very thankful for the funding provided by Agriculture and Agri-Food Canada, Canadian Beef Check-Off, and the Beef Cattle Research Council through the Canadian Beef Cattle Industry Science Cluster. CVJ

REFERENCES

1. Dewes HF. Transit-related lameness in a group of Jersey heifers. N Z Vet J 1979;27:45.
2. Sick FL, Bleeker CM, Mouw JK, Thompson WS. Toe abscesses in recently shipped feeder cattle. Vet Med Small Anim Clin 1982;77:1385–1387.
3. Miskimins DW. Bovine toe abscesses. Proceedings of the 8th International Symposium on Disorders of the Ruminant Digit 1994:54–57.
4. Penny C, Bradley S, Wilson D. Lameness due to toe-tip necrosis syndrome in beef calves. Vet Rec 2017;180:154.
5. Jelinski M, Marti S, Janzen E, Schwartzkopf-Genswein K. A longitudinal investigation of an outbreak of toe tip necrosis syndrome in western Canadian feedlot cattle. Can Vet J 2018;59:1202–1208.
6. ICAR. The Global Standard for Livestock Data [Internet]. ICAR Central Health Key [updated January 14, 2020]. Available from: http://www.icar.org/index.php/publications-technical-materials/amendments-recording-guidelines/diseases-codes-for-cows/. Last accessed February 27, 2025.
7. Jelinski M, Fenton K, Perrett T, Paetsch C. Epidemiology of toe tip necrosis syndrome (TTNS) of North American feedlot cattle. Can Vet J 2016;57:829–834.
8. Gyan LA, Paetsch CD, Jelinski MD, Allen AL. The lesions of toe tip necrosis in southern Alberta feedlot cattle provide insight into the pathogenesis of the disease. Can Vet J 2015;56:1134–1139.
9. Johnston JD, Eichhorn DJR, Kontulainen SA, Noble SD, Jelinski MD. Investigation of white line separation under load in bovine claws with and without toe-tip necrosis. Am J Vet Res 2019;80:736–742.
10. van der Tol PP, Metz JH, Noordhuizen-Stassen EN, Back W, Braam CR, Weijs WA. The pressure distribution under the bovine claw during square standing on a flat substrate. J Dairy Sci 2002;85:1476–1481.
11. Kofler J. Clinical study of toe ulcer and necrosis of the apex of the distal phalanx in 53 cattle. Vet J 1999;157:139–147.
12. Meléndez D, Marti S, Pajor E, *et al.* Effect of band and knife castration of beef calves on welfare indicators of pain at three relevant industry ages: I. Acute pain. J Anim Sci 2017;95:4352–4366.
13. Moya D, Schwartzkopf-Genswein KS, Veira D. Standardization of a non-invasive methodology to measure cortisol in hair of beef cattle. Livest Sci 2014;158:138–144.
14. Meléndez DM, Marti S, Pajor EA, *et al.* Effect of timing of subcutaneous meloxicam administration on indicators of pain after knife castration of weaned calves. J Anim Sci 2017;95:5218–5229.
15. Kofler J. Pathogenesis and treatment of toe lesions in cattle including "nonhealing" toe lesions. Vet Clin North Am Food Anim Pract 2017;33:301–328.
16. Paetsch C, Fenton K, Perrett T, *et al.* Prospective case control study of toe tip necrosis syndrome (TTNS) in western Canadian feedlot cattle. Can Vet J 2017;58:247–254.
17. Godson DL, Campos M, Attah-Poku SK, *et al.* Serum haptoglobin as an indicator of the acute phase response in bovine respiratory disease. Vet Immunol Immunopathol 1996;51:277–292.

18. Moisá SJ, Aly SS, Lehenbauer TW, *et al*. Association of plasma haptoglobin concentration and other biomarkers with bovine respiratory disease status in pre-weaned dairy calves. J Vet Diagn Invest 2019;31:40–46.
19. Chen Y, Arsenault R, Napper S, Griebel P. Models and methods to investigate acute stress responses in cattle. Animals 2015; 5:1268–1295.
20. Mormède P, Andanson S, Aupérin B, *et al*. Exploration of the hypothalamic-pituitary-adrenal function as a tool to evaluate animal welfare. Physiol Behav 2007;92:317–339.
21. Schwartzkopf-Genswein KS, Janzen E, Jelinski M, *et al*. Occurrence, characterization and risk factors associated with lameness within Alberta feedlots. Interim Report (4 pg) to ALMA Project # 2013R008R ANH.10.11 (December 31, 2014). Ottawa, Ontario: Agriculture and Agri-Food Canada, 2014.
22. Wittum TE, Young CR, Stanker LH, Griffin DD, Perino LJ, Littledike ET. Haptoglobin response to clinical respiratory tract disease in feedlot cattle. Am J Vet Res 1996;57:646–649.
23. Roland L, Drillich M, Iwersen M. Hematology as a diagnostic tool in bovine medicine. J Vet Diagn Invest 2014;26:592–598.