PEER REVIEWED

Case Report – Tail Tip Necrosis in a Confined Cattle Feeding Operation

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Abstract

Tail tip necrosis was reported in cattle fed in a confinement feedlot in Nebraska. One-time capacity of the feedlot was 80,000 head, and reported prevalence of tail tip necrosis was 1%. Cattle were typically on feed for 120-150 days before signs of the disease were noted. Thirty percent of affected cattle failed to respond to therapy, and died or were marketed prematurely because of chronic disease. Abnormalities of the tail, joints, and lungs were found during post-mortem examination. *Arcanobacterium pyogenes* was isolated from all sites that were sampled. Tail tip necrosis is common in confinement feedlot facilities where cattle are housed on slatted floors, and can lead to bacteremia and death.

Keywords: bovine, tail tip necrosis, feedlot

Résumé

La nécrose du bout de la queue a été rapportée chez des bovins nourris dans un parc d'engraissement confiné du Nebraska. Le nombre de têtes à tout moment s'élevait à 80 000 et la prévalence de la nécrose du bout de la queue était de 1%. Les bovins étaient typiquement nourris de 120 à 150 jours avant que les signes de la maladie n'apparaissent. Près de 30% des bovins affligés ne répondaient pas au traitement et sont morts ou ont été envoyés au marché prématurément en raison de maladie chronique. L'examen post-mortem a révélé des anormalités de la queue, des articulations et des poumons. La bactérie Arcanobacterium pyogenes a été isolée de tous les sites échantillonnés. La nécrose du bout de la queue est fréquente dans les parcs d'engraissement confinés où les bovins sont gardés sur les planchers lattés et peut entraîner la bactériémie et la mort.

Introduction

Producers in the upper midwestern United States continue to build confinement cattle feeding facilities within proximity to ethanol plants to utilize by-product feeds for finishing cattle. Tail tip necrosis is a recognized problem in confined feeding operations.[2,3,8,9] *Arcanobacterium pyogenes* (formerly *Corynebacterium pyogenes)* and *Bacteriodes melaninogenicus* have been isolated from lesions in bulls with tail necrosis.[7]

A mail survey of 255 feedlots in southern Ontario was conducted to establish prevalence of the problem.[3] Of those surveyed, only 71 feedlots penned cattle on slatted floors, but 96% of those facilities reported a problem with tail tip necrosis.[3] In a report from Switzerland, tail tip necrosis was more prevalent in cattle finishing units with slatted floors compared to confinement facilities that deep-bed cattle with straw. Prevalence increased as stocking density increased, but prophylactic tail docking decreased prevalence of the lesion in cattle housed on slatted floors.[8]

Confinement feeding operations discussed in this case report differ from traditional open-air cattle feedlots. These confinement feeding facilities provide cattle with 20-30 square feet of pen space per animal. The pen floor is typically either concrete or metal, and slatted or sloped for waste management. They are also covered with a roof to provide protection from the environment and to limit the amount of manure and urine runoff. Cattle in traditional open-air feedlot operations are typically provided 150-400 square feet of pen space per head, and housed outdoors in pens with dirt floors.

An increase in construction of confinement feeding operations and more frequent reports of tail tip necrosis led to the design of a field and diagnostic investigation of the disease. The purpose of this study was to further de-

fine the pathogenesis, to identify the etiological agent(s), describe case outcomes, and develop management options for controlling tail tip necrosis, including welfare of the animals and the economics of the disease.

History

In November 2005, the manager of an 80,000-head capacity confinement cattle feeding operation in central Nebraska reported tail tip necrosis and abscessation affecting approximately 1% of cattle in inventory. Lesions were not seen in newly arrived cattle, and cattle were in good health prior to the development of tail lesions. Cattle with tail lesions were typically on feed for 120 to 150 days and were near market weight. The disease progressed rapidly over approximately 10 days, resulting in severe lameness, depression, and death.

Cattle were routinely vaccinated during arrival-processing with infectious bovine rhinotracheitits (IBR) virus, bovine viral diarrhea virus (types 1 and 2), parainfluenza-3 virus, and bovine respiratory syncytical virus vaccine,[a] and re-vaccinated 14 days later with IBR vaccine.[b] All cattle were treated metaphylactically with tilmicosin[c] (4.54 mg/lb [10 mg/kg] body weight) on arrival. Cattle also received an injectable endectocide[d] (1 mL/110 lb [50 kg] body weight), given an oral dose of fenbendazole[e] (5 grams of paste/220 lb [100 kg] body weight), and a numbered ear tag for identification. Cattle received a growth-promoting implant[f] at the time of revaccination, and were reimplanted with a terminal steroid implant[g] approximately 100 days prior to harvest. Cattle with horns were dehorned, and bulls were castrated at arrival-processing. The switch hair was removed from the end of the tail at the time of processing with a mechanized device.

Cattle were housed in a confinement feeding barn which had a side curtain on the north side and was open on the south side. There was concrete slatted flooring with 105 to 200 cattle per pen, stocked at an average of 23 ft^2 per head. All cattle had *ad libitum* access to feed and water.

Pen checkers pulled cattle when they saw a bloody tail in early cases, or minor knuckling of one rear limb in more progressed cases. Cattle with tail abscess lesions were treated with ceftiofur sodium[h] (0.5 mg/lb [1.1 mg/kg] of body weight) for three consecutive days, and the tail was amputated proximal to the lesion utilizing a high-tension rubber band.[i] Banding-amputation was done in an attempt to localize the lesion and minimize the spread of infection to other body systems. The consulting veterinarian reported 30% of treated cattle either died or were marketed early because of poor response to treatment. Once cattle exhibited multi-limb lameness, nearly 100% either died or were marketed prematurely. The high treatment failure rate, concern over animal welfare issues, and continued economic losses were reasons cited for seeking further diagnostic investigation.

Clinical and Laboratory Findings

Two steers severely lame in the hind limbs were presented to the Kansas State University Veterinary Diagnostic Laboratory (KSU VDL) for examination. Both steers were humanely euthanized. Postmortem examination showed both steers were in good body condition with adequate body fat stores. The tails had been amputated, with 1/2 to 1/3 of the proximal tail remaining.

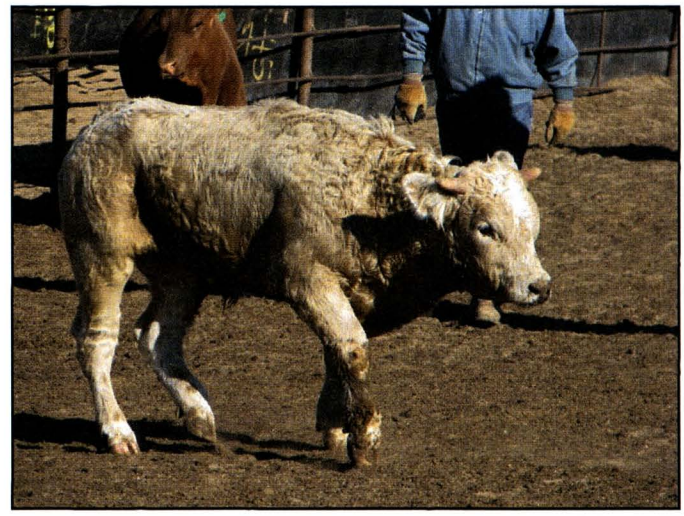

Figure 1. Severely lame Charolais steer that had been on feed in a Nebraska confinement feedlot. The steer was presented for necropsy.

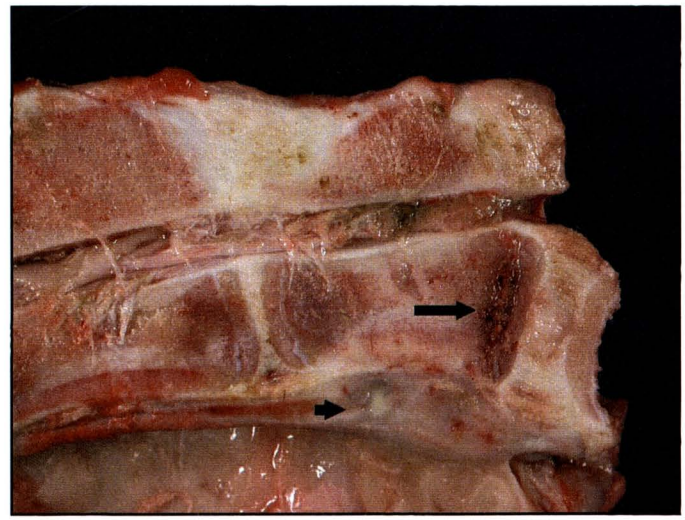

Figure 2. Sixth sacral vertebra caudal physis (long arrow) and ventral tail abscess (short arrow) in a Charolais feedlot steer.

Gross Pathology

The Charolais-cross steer (Figure 1) had multiple abscesses along the ventral surface of the sacral vertebrae (Figure 2), suggesting an ascending bacterial infection along the ventral surface of the tail, perhaps within the vasculature (although there was no definitive evidence that the abscesses were within the vasculature). The caudal physis of the 6th sacral vertebra was dark red, thickened and necrotic with adjacent bone lysis indicating physitis (Figure 2). Numerous 2-3 cm-sized, dark red to black, discrete foci consistent with an embolic pneumonia were distributed throughout all lung lobes (Figures 3 and 4). The interlobular septae were diffusely widened with clear fluid.

The black steer had dry, gangrenous necrosis in the distal end of the remaining tail (Figure 5). The right stifle was swollen, and the joint contained large amounts of yellow-green purulent fluid consistent with fibrinosuppurative arthritis (Figure 6). Three draining tracts from the joint connected to large periarticular abscesses in the muscles and soft tissues distal, cranial, and caudal to the joint.

Histopathology

Histologic evaluation of lesions from both calves was conducted at the KSU VDL. The 6th sacral vertebra had physitis characterized by necrosis and neutrophilic inflammation of the subchondral trabecular bone and the

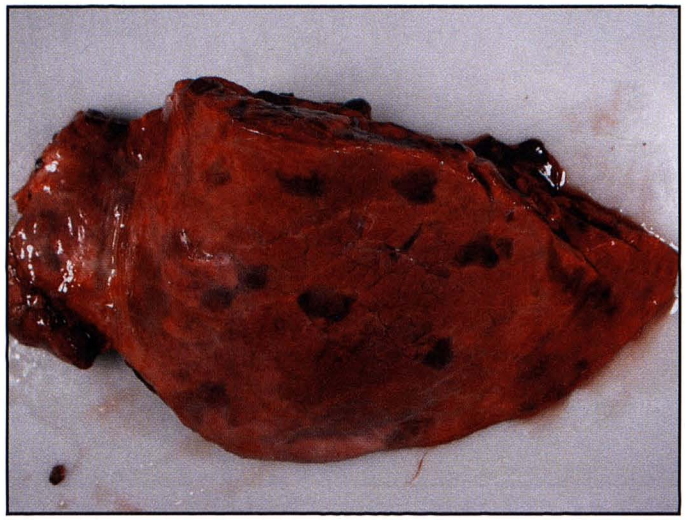

Figure 3. Embolic pneumonia in a Charolais feedlot steer due to *A. pyogenes*.

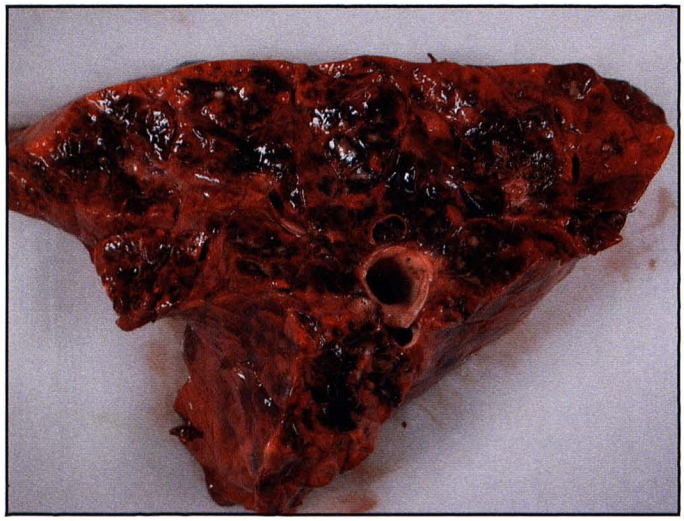

Figure 4. Cross-section of lung with embolic pneumonia due to *A. pyogenes* bacteremia in a Charolais feedlot steer.

Figure 5. Gross lesions associated with tail tip necrosis in a black feedlot steer.

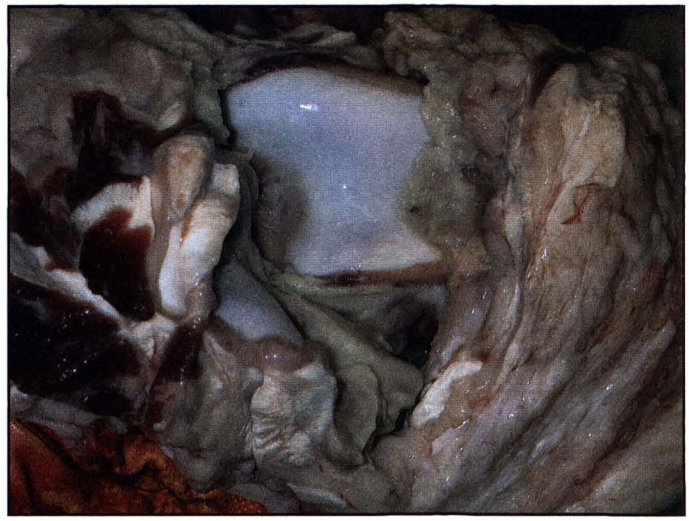

Figure 6. Arthritis caused by *A. pyogenes* in the right stifle joint of a black feedlot steer.

physis. The ventral tail abscesses were microscopically characterized by areas of connective tissue and skeletal muscle necrosis with neutrophilic inflammation further surrounded by granulation tissue. Also, there were numerous intact and degenerate neutrophils admixed with abundant fibrin within the stifle joint capsule. The periarticular abscesses were characterized by necrosis and large numbers of degenerate neutrophils admixed with abundant fibrin surrounded by granulation tissue.

Microscopically, the grossly affected areas of the lungs contained fibrin, edema, and occasionally neutrophils in the alveolar spaces, and the interlobular septae were widened by abundant fibrin. Occasionally, arterioles and alveolar capillaries contained fibrin thrombi. The microscopic lesions were consistent with an embolic pneumonia.

Bacteriology

Culture of the tail abscesses and lung lesions yielded abundant growth of *A. pyogenes* in pure culture. Bacterial cultures of the material from the periarticular abscesses and joint fluid also yielded abundant growth of *A. pyogenes* in pure culture.

Discussion

Tail tip necrosis has been documented in cattle from European and Canadian confinement finishing operations.[2,3,8,9] The current literature has described this syndrome through slaughterhouse inspection of tails, mail-in surveys of producers, and inspection of cattle at smaller feeding operations (less than 500 head).[3,8] This case report is unique because it describes the problem in a US confinement cattle feeding operation with a capacity of 80,000 head.

Prevalence of this syndrome increases when cattle are fed in confinement on slatted floors.[3,8] The authors of this case report speculate that the amount of allotted space leads to animals stepping on the ends of tails.

A. pyogenes was isolated from each lesion site cultured from the steers presented for examination. It is a common opportunistic pathogen that causes a variety of suppurative infections involving the skin, joints, and organs.[4] *A. pyogenes* is commonly found in mixed infections; however, it is capable of acting as a primary pathogen and can cause systemic infection, often following trauma to a mucous membrane. Pyolysin, a potent extracellular toxin that is a haemolysin, is the primary virulence factor for *A. pyogenes*. It is cytolytic for a number of immune cell types, such as macrophages. Pyolysin has also been shown to be dermonecrotic[6] and lethal to laboratory animals via intravenous and intraperitoneal routes.[5] In addition to pyolysin, a pore-forming, cholesterol binding toxin, *A. pyogenes* expresses a number of virulence factors which help it to bind host epithelial cells.[1] This microbiological description of *A. pyogenes* allows us to logically envision its natural role in tail tip necrosis cases, and can lend insight to how therapies could be improved for treatment of these cases.

Recommended treatment for tail tip necrosis is systemic antimicrobial therapy and banding of the tail proximal to the necrotic area to amputate the affected area.[3,8,9] Case outcomes or expected case fatality rates with these treatment protocols were previously unknown. The consulting veterinarians for this feedlot reported a 70% treatment success rate; however, nearly 100% of cattle died or were marketed prematurely if the disease had progressed to swelling and lameness of the hind limbs. Treatment was unrewarding when it reached this stage.

Antimicrobial susceptibility patterns from *A. pyogenes* isolates received at the KSU VDL received during the time this case was investigated are presented in Table 1. More than 90% of the organisms were susceptible to 11 of the 16 antimicrobials tested. Use of neomycin and gentamicin is strongly discouraged in food producing animals, and clindamycin and tiamulin are not approved for use in cattle. Susceptibility to penicillin and ceftiofur, which was used to treat the condition in this case, was not reported. Along with susceptibility, the progression of this disease may dictate that antimicrobials with good tissue penetration and the ability to control sepsis should be considered. Increased duration of therapy may be helpful, but was not evaluated

Table 1. Percentage of *Arcanobacterium pyogenes* isolates from cattle susceptible to various antimicrobials.[a]

Antimicrobials	Percent susceptible[b]
Ampicillin	100
Chlortetracycline	100
Clindamycin	91
Enrofloxacin	55
Erythromycin	100
Florfenicol	100
Gentamicin	100
Neomycin	91
Oxytetracycline	64
Spectinomycin	91
Sulphachloropyridazine	64
Sulphadimethoxine	82
Sulphathiazole	0
Tiamulin	100
Tilmicosin	100
Trimethoprim/sulphamethoxazole	100

[a]Data courtesy of the Bacteriology Laboratory, College of Veterinary Medicine, Kansas State University.
[b]Eleven isolates were cultured between August 7 and October 18, 2005.

in this case. As noted, most cattle affected in this case and in the published literature were generally close to harvest, which dictates the need for careful treatment strategies to meet mandated pre-harvest withdrawal requirements.

Prevalence of tail tip necrosis was 1% of the population in the feedlot described here. In contrast, Swiss investigators reported that the prevalence of a tail tip necrosis approached 40% of cattle housed on slatted floors.[8] This facility allowed animals 23 ft^2 per head, which is similar to pen space allotments published in European literature. The Swiss report showed that the prevalence of tail lesions decreased[8] in 880 to 1,100 lb (400 to 500 kg) cattle as pen space was increased from 22 ft^2 to 37 ft^2. The difference in the number of cattle on feed in the Nebraska facility and the European facilities may partially explain differences in the lesion frequency that was reported. Also, Swiss investigators were looking for any change in tail pathology, while the Nebraska feedyard personnel were only treating cattle that had lesions requiring therapy. This raises the question of possible differences in case definition.

Cattle in this case report developed tail tip necrosis late in the feeding period, similar to other reports.[3,8] As the animals increase in size and weight, there is less space per pound of body weight,[8] essentially increasing risk of injury because of crowding. One management technique that might decrease the prevalence of tail tip necrosis is to increase the pen space allotment as animals increase in body weight, or avoid filling the pens as densely at the beginning of the feeding period. Because the problem does not typically occur until later in the feeding period, more intense surveillance in longer-day cattle and expanded training on early signs of the disease could result in earlier intervention and improved response to treatment.

A lot of unanswered questions about tail tip necrosis in feeder cattle remain. Prevalence of tail tip lesions in different types of confinement feeding operations with different space allotments has not been elucidated in the US. Also, drug usage, duration of therapy, and the usefulness of tail amputation have not been defined. A clinical study on the pathogenesis of the disease could lead to novel therapeutic and management intervention strategies.

Conclusions

Tail necrosis and abscessation was diagnosed in 1% of cattle on feed in an 80,000 head confinement feedlot facility in Nebraska. Lesions typically were seen in cattle on feed for 120 to 150 days or more, and approaching slaughter weight. Progression of tail lesions can lead to hematogenous infections of lungs and joints, and the disease can progress from lameness to depression to death in a 10-day period. Treatment of cattle with lameness and joint swelling has been unrewarding. More research to investigate this syndrome in US feeder cattle is needed.

Endnotes

[a]Bovishield Gold 5, Pfizer Animal Health, New York, NY
[b]Bovishield IBR, Pfizer Animal Health, New York, NY
[c]Micotil, Elanco Animal Health, Greenfield, IN
[d]Ivermectin 1% Injection, Durvet Animal Health Products, Blue Springs, MO
[e]Safeguard, Intervet/Schering-Plough Animal Health, Millsboro, DE
[f]Component IS, Vetlife, West Des Moines, IA
[g]Component TES with Tylan, Vetlife, West Des Moines, IA
[h]Naxcel Sterile Powder, Pfizer Animal Health, New York, NY
[i]Callicrate Bander, No-Bull Enterprises, St. Francis, KS

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