115's Wild Rodeo

Haley Brazell

Mississippi State University

College of Veterinary Medicine

Class of 2021

Clinicopathologic Conference

December 4th, 2020

Advisor:

Amelia Woolums, DVM, MS, PhD

Introduction

Lameness in cattle is highly prevalent causing a substantial financial impact for producers, three fourths of the animals sent to slaughter at an early age are the result of lameness.[3,4,6] In cattle weight distribution and hoof conformation play a key role. The forelimbs carry approximately 53% of the load and the hindlimbs around 47% of the load.[2] The weight distribution paired with the dynamics of the bovine claw leads to the majority of lameness being located in the foot, specifically the hind feet.[2] Common causes in beef cattle include interdigital necrobacillosis also known as foot-rot, trauma, and toe abscess.[2,4,10] Toe abscesses have also been referred to as toe ulcer, apical white line disease, toe necrosis and others.[3,4,6,9] Recently a study published in the Canadian Veterinary Journal, developed the umbrella term "Toe Tip Necrosis Syndrome".[3,6,9] Toe Tip Necrosis Syndrome (TTNS) encompasses the pathology seen with the previous terms plus disease sequela.[3,4,6,9] The following is a case presentation and discussion of TTNS in a beef heifer.

History & Presentation

115 is an approximately 10-month-old Brangus heifer that presented to Food Animal Emergency Services on November 10th, 2019 for complaints of lameness. 115 was previously seen on November 2nd by the ambulatory service, after injuring both lateral claws of the hindlimbs. There were abrasions noted at the white line, presumed to be due to trauma from the bottom grate of the chute when the heifer was processed at arrival 2 days prior. A dose of flunixin meglumine (Banamine), 250 mg (1.1 mg/kg) was administered intravenously. On November 6th, the patient had bilateral nasal discharge. It was suspected that she developed Bovine Respiratory Disease (BRD) so a dose of ceftiofur crystalline free acid (Excede) 1,371 mg (6.6 mg/kg) was administer in the fat pad behind the ears. On November 7th, during processing at South Farm, 115 became agitated in the chute and was noted to flip over backwards. The patient was noticeably lame

afterwards and swelling was noted over the right gluteal muscles. Another dose of Banamine 458 mg (2.2. mg/kg) was administered that day and she received a dose prior to presentation on the 10^{th}.

On presentation 115 was bright, alert, responsive, she was excitable and aggressive. 115 weighed 457 pounds (208 kilograms) with a body condition score of 3/9 (5/9 being ideal). Physical exam revealed a heart rate of 88 beats per minute, respiratory rate of 88 breaths per minute and a temperature of 103.2 degrees Fahrenheit. Her hydration status was normal, and her mucous membranes were pink and moist. Bruxism, which is a sign of pain in cattle was heard throughout the entire physical examination. There was bilateral serous nasal discharge, there was no ocular drainage noted. Cardiopulmonary auscultation was within normal limits and rumen contractions were normal with 1 per minute.

Examination of 115's extremities showed mild swelling in the right hindlimb over the superficial gluteal muscle. The distal extremities had abrasions on all four limbs. There was a small amount of blood present in the interdigital space of the right hindlimb. Mild swelling was present bilaterally at the coronary band in the rear limbs with the right being worse than the left. A drainage tract was also noted, over the medial claw of the right hindlimb. During the examination of 115's extremities she became excited, turned around and kicked the head gate causing a non-weight bearing intermittent lameness in the right hindlimb and another superficial abrasion over the dorsal aspect of the left carpus. Based on these findings and gate analysis 115 was given a lameness score of 4/5 and was hospitalized so she could be further evaluated on the following Monday.

Diagnostics & Considerations

Diagnosis of TTNS usually involves a multimodal approach consisting of assessment of clinical signs, a foot table exam, potentially radiographs, and fluid cytology of any purulent material from the claws.[5] Definitive diagnosis is made by identifying necrosis of the toe and P3, after nipping and debriding the lesion or during necropsy.[4,5] Histopathology of the structure can also be considered.[4,5] Clinical diagnostic signs primarily consist of lameness without effusion present at the coronary band or heal bulbs.[4,7,9] On foot table exam, identification of white line separation at the toe is considered highly suggestive for the development of TTNS.[4] In one particular study diagnosis of TTNS, "was considered if there was a lesion of the foot involving inflammation or necrosis of the corium with or without any combination of inflammation, necrosis or lysis of P3".[3] Toes will have a rounded appearance and wear.[7,9] Hoof testers can be used to localize pain.[8,9] The use of a probe to identify and follow tracts, help to reveal pseudo-soles and extent of infection to P3.[5,8,9]

Aerobic and Anaerobic culture swab of the toe apex, P3 or cut surface at necropsy can be performed on blood and MacConkey agar, typical bacteria cultured are *Escherichia coli* and *Trueperella pyogenes*.[5,9,10] Immunohistochemistry can be performed on an ear notch or other tissue if BVDV persistent infection is suspected to be a contributing cause.[5,10] Radiographs can be diagnostic to identify the involvement of P3, such as osteomyelitis, the presence of sequestrums and pathological fractures.[8,9] Dorsopalmar/plantar and one oblique view provide the most information.[8,9] "Histopathology can be performed using hematoxylin and eosin stain, to identify inflammation, thrombi, necrosis, bacteria and to evaluate the epidermal lamellae. Periodic Acid-Schiff reaction, and Gram's stain can also be considered when evaluating and identifying the lamellae's basement membrane and presence of bacteria".[3,5] Gross necropsy will show a pseudo-sole, osteolytic changes and involvement of the corium.[8]

Diagnose of TTNS in 115 was made using findings from the foot table exam and her history leading up to the development of disease.

Pathophysiology

The normal bovine hoof has around 7mm of sole depth, followed by 1 mm of sole corium and 3 mm of a subcutaneous layer at the toe before finding P3's solar surface.[9] This only accounts for approximately 11 millimeters or roughly 1 centimeter before bone is exposed. With respect to any disease process it only takes a few days before infection will reach P3.[9] In addition, the bovine claw tends to have a higher moisture content due to the environment.[9] These wet conditions will allow the keratin that makes up the hoof wall to become soft and prone to damage from weight bearing, or hard surfaces.[9] Vascular anatomy also plays a role in the development of toe lesions.[9] The palmar/plantar digital artery and vein form an arch within P3, branching vessels extend outward to the laminae.[9] Any disruption to this network of vasculature could result in pathology to the hoof and spread to the deeper tissues and skeletal structures.[9]

The anatomy described in the previous paragraph has allowed for a few theories to develop on how TTNS occurs in cattle, those include mechanical trauma, vascular disruption, and laminitis.[9] We will briefly talk about the last two theories before going into depth about mechanical trauma. Mild cases of laminitis due to a metabolic imbalance can cause sole hemorrhages, soft or thin soles, softer claw horn and changes to the apex of P3 which can lead to the development of TTNS.[9] Vascular disruption usually due to hypostasis of blood from standing during transport is thought to lead to the development of toe tip necrosis.[3,9] The distal phalanx and associated soft tissue structures rely on constant blood circulation which is facilitated by walking.[9] When cattle cannot walk the blood becomes stagnant leading to decreased circulation, this decrease in circulation can cause hypoxia, ischemia and necrosis of the surrounding tissues

and apex of P3.[9] Bovine Viral Diarrhea Virus (BVDV), has been implicated in causing vascular disruption in the distal extremities as well.[3,9] It is thought that because BVDV causes a vasculitis it will decrease perfusion to the tissues, allowing for toe lesions to occur.[9]

In recent studies, researchers have come to a consensus that the development of TTNS stems from mechanical trauma. The "abrasion theory" as it is termed, revolves around the idea that during processing and transport the claws of cattle are exposed to abrasive surfaces which can cause lesions at the toe.[4,5,7,9] The area of the white line is the most susceptible to developing these lesions.[3,5,7] It is composed of an outer, middle and inner sections, which are all developed from different laminae.[10] This difference in each section means the white line is not a solid tissue structure and is thought to be the weak link in the hoof anatomy.[3,7,10] This weakness is why researchers believe the white line (junction of the hoof wall and sole) is prone to damage when abrasions occur at the apex of the toe.[3,6,7,10]

When damaged, micro-fissures develop in the outer section, repetitive load from walking causes the fissures to become deeper and spread outward leading to separation from the middle section.[5,10] When load is applied the defect in the white line expands allowing bacteria and debris to enter the apex of the toe.[5,7,10] When load is removed from the hoof, bacteria become trapped in the fissures.[5,7,10] Bacteria colonize at the apex of the toe, then migrate to the corium followed by P3 and the surrounding soft tissues.[5,7,10] The most common bacteria cultured from this area are E. *coli* and *Truperella pyogenes.*[5,10] Infection will progress causing necrosis of the corium, apex of P3 and soft tissue structures.[5,10] The end stage of the disease process results in infection spreading proximal up the extremity and/or bacteremia which can result in death of the animal.[4,5]

In 115's case, the initial insult was thought to come from being processed in the chute system. 115 was excitable and therefore scuffed the plantar surface of her hooves causing the

abrasions at the white line. From there, the development of TTNS followed the "abrasion theory" as previously described. The disease process was caught in time before the infection spread proximally and bacteremia developed.

Treatment & Management

Successful treatment of TTNS depends on catching the disease process early, the age of the animal, how many claws are affected, pregnancy status and if any co-morbidities are present.[9] One of the first steps is to perform a thorough foot table exam and foot trim.[9] The foot trim usually consists of removing "1-2 mm of sole and hoof wall to create a clean surface" in all four extremities.[9] Once this is complete the claws can be examined for any lesions.[9] Removal of affected hoof is aided with the use of a hoof knife, and hoof trimmers.[5,9] In cases where only one claw is affected, the adjacent claw is examined to see if it can withstand placement of a wooden block to relieve pressure on the affected claw.[8,9] If both claws are affected, it is recommended that a wooden block not be placed.[9]

In advanced cases, involving the deeper structures, thorough debridement of the underrun sole, necrotic tissue and bone are required. Intravenous regional limb perfusion of 10-15 mls of lidocaine can provide local anesthesia to aid in this process.[5,8,9] Debridement should be continued "2-3 mm past healthy tissue and bone".[9] A bone curette, probe to find tracts of necrotic tissue and Forstner drill may be needed in these cases.[5,8,9] If P3 is involved the apex may need to be resected, with the aid of bone curette, obstetrical wire, surgery using an angle grinder or amputation of the claw.[5,8,9] It is recommended to sedate these animals with xylazine or detomidine if surgery is to be performed.[9]

“Angle grinders with a steel disc, disinfected with 70% ethanol solution can be used to remove the tip of the claw, infected tissue (horn, corium) and bone.[9] Surgical amputation of the claw is considered in non-healing cases and if infection has reached the distal interphalangeal joint space.[9] Once foot trim, thorough debridement or surgery are complete, the area should be flushed with sterile saline, dried and a tetracycline or Betadine spray applied.[5,8,9] The claw should have a sterile dressing applied and a pressure bandage placed that extends to the metatarsus or metacarpus.[9]

Mild cases of TTNS usually heal around 30 days, but in severe cases healing can take around 5-8 weeks.[5,8] If bone resection, surgery or amputation were performed, animals should be started on a broad spectrum systemic antibiotic with good Gram negative and anaerobe coverage, along with administration of a non-steroidal anti-inflammatory drug such a meloxicam.[5,8,9] Animals should be separated from the herd and stalled on soft surfaces.[5,9] Bandage changes should occur every 3-5 days until the site is covered with a healthy bed of granulation tissue, and re-epithelization is observed at wound edges.[8,9] There is a report of, “81.5 % success rate following surgical techniques if only one claw is involved”.[9] If more than one claw is involved the success rate decreases significantly and most animals are culled.[8,9]

Prevention of TTNS consists largely of minimizing stress and contact with abrasive surfaces.[4,5,9] Management strategies include limiting exposure to concrete surfaces or covering concrete and steel surfaces with rubber flooring.[5,9,11] Other options include turning cattle out on a dry pasture or housing in a heavily bedded stall, free of moisture.[9,11] It is also recommended to have regular foot trims performed 2-3 times a year and animals should be evaluated routinely for lameness to catch early disease development or at-risk animals.[9]

Treatment of 115 was initiated on November 11th, with an oral bolus of electrolytes to correct mild dehydration. A subcutaneous injection of florfenicol (Nuflor) 8,400 mgs (40mg/kg) was given every 96 hours and the heifer was started on oral meloxicam 15 mg (1mg/kg) once daily for 4 consecutive days. Initially the top differentials were interdigital dermatitis (foot rot) and trauma. On November 15th, a foot table exam and foot trim were performed. 115 was sedated with an intramuscular injection of acepromazine 10mgs (0.1mg/kg) to reduce stress and aid examination of her extremities. A combination of small hoof trimmers, a hoof knife, bone curette and probe were used. Upon examination of the plantar surfaces white line separation was noticed at the apex of the medial and lateral claws in both hindlimbs .(1) Hoof trimmers were used to remove the apex of each toe with each toe revealing a tract.(1) A probe was used to follow each tract along the sole of the respective toe.(1) The soles of all 4 rear claws were removed due to the formation of pseudo-soles (false soles) and the underlying necrotic tissue was removed. Necrotic hoof wall was removed from the medial and dorsal surfaces in all 4 claws. Following debridement, the distal phalanx was exposed in all 4 rear claws and a sequestrum was removed from P3 of the lateral claw of the right hind limb. The claws were cleaned with chlorhexidine scrub, rinsed with distilled water, and dried with 4X4 gauze. Silver sulfadiazine cream was applied to the plantar surfaces of each foot with 4X4 gauze. Soft padded bandages were applied using brown gauze, Vetwrap, Elastikon and duct tape applied to the bottom of the foot. The patient was administered another dose of Nuflor as previously described.

On November 19th treatment consisted of additional debridement of the right rear hoof and lateral claw of the left rear hoof. Additional sequestrums were removed from P3 of the medial claw on the right hindlimb and the lateral claw of the left hindlimb. Soft padded bandages were changed as previously described. Administration of Nuflor and continued meloxicam at every other day

dosing. Additional cleaning and bandage changes took place on November 21st and November 22nd due to patient removing the bandages. 115 was also administered Nuflor every 4 days and continued meloxicam every other day until discharge from the Animal Health Center.

On November 25th, a swelling with subcutaneous emphysema was noted on the left side of the heifer's neck. The area was ultrasounded, revealing a fluid filled space within a capsule. The area was clipped, prepped with Betadine scrub, and lanced with a number 10 scalpel blade, after which purulent material was drained from the area. The abscess was flushed with Betadine solution and packed with Betadine soaked brown gauze. The bandages on her rear limbs were changed as previously described. On November 27th, the bandages were removed so her hooves could be debrided and cleaned. The Betadine packing from the abscess was also removed. A sequestrum was removed from P3 of the lateral claw of the right hindlimb and excessive granulation tissue was removed. A soft padded bandage was re-applied to the right hind foot. A cast was placed on the left rear hoof. The cast was placed to give a more permanent bandage solution due to 115 removing the soft padded bandages, as the left foot was judged to no longer need regular debridement. At this time, an abscess had developed over the medial claw of right hind limb, the abscess was lanced, flushed with Betadine, and left open to drain.

The cast on the left hindlimb was removed on November 30th and replaced with a soft padded bandage along with a bandage change of the right hind limb. The bandage change was repeated on December 1st. On December 2nd, both soft padded bandages were removed, and her hooves were found to be healing appropriately. The abscess on her neck and over the medial claw of the right hindlimb were also healing appropriately. The left hoof had a healthy bed of granulation tissue so a soft padded bandage was re-applied before discharge. The right hoof was placed in a cast, due to a small amount of P3 still being exposed. Nuflor was discontinued at this

time. 115 was discharged on December 3rd with instructions to monitor her lameness, and have the bandage and cast replaced if they were removed. A recheck visit was scheduled for 2 weeks after discharge, so the hooves and abscesses could be re-evaluated, and the cast removed from the right hindlimb.

Case Outcome

115 returned for her 2 week recheck and cast change on December 16th. Her lameness had improved since her last visit and she was given a 1/5 lameness score. The abscess on the left side of her neck was still present but appeared to be healing appropriately. Her right hindlimb cast was still intact along with the left hindlimb bandage. Foot table exam revealed appropriate healing in all four claws, with significant improvement in the left hindlimb and new sole growth. P3 was no longer visible in the right hindlimb claws and a healthy bed of granulation tissue was present. The bandage on the left hindlimb was not replaced, while a splint was placed on the right hindlimb to provide support until new sole growth appeared. 115 was scheduled for a 2 week recheck to reassess the abscess on the left side of her neck and to re-evaluate the right hind limb.

On January 2nd, 2020 115's lameness was worse, she was noticeably lame at the walk and given a score of 2/5. The splint on the right hindlimb was worn at the bottom along with a significant amount of manure packed at the fetlock. This finding was thought to be the cause of her lameness. The abscess on the left side of her neck had not improved and two Penrose drains had been placed on December 31st. A foot table exam showed that all four claws were still healing appropriately with the claws of the left hindlimb nearly completely healed. The right foot had continued to heal well since the last visit with new sole growth present, as a result a bandage was not replaced. 115 was hospitalized for approximately 2 weeks so the abscess on her neck could be treated. While in hospital she was also diagnosed with dermatophytosis and treated. Following her

discharge, 115's skin lesions and abscesses resolved, she was no longer lame, and all four claws completely healed. Following her recovery 115 was sent to market.

References

1. Cowley, J. (2020). *Toe Tip Necrosis*. Department of Population Medicine and Pathobiology Presentations. Mississippi State University, Starkville.
2. Fleming, S. A. (2018). *Bovine Lameness*. Food Animal Medicine and Surgery. Mississippi State University, Starkville.
3. Gyan, L. A., Paetsch, C. D., Jelinski, M. D., & Allen, A. L. The lesions of toe tip necrosis in southern Alberta feedlot cattle provide insight into the pathogenesis of the disease. Canadian Veterinary Journal 2015; 56:1134-1139.
4. Jelinski, M., Fenton, K., Perrett, T., & Paetsch, C. Epidemiology of toe tip necrosis syndrome (TTNS) of North American feedlot cattle. Canadian Veterinary Journal 2016; 57:829-834.
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. Canadian Veterinary Journal 2018; 59:1202-1208.
6. Jelinski, M., Waldner, C., & Penner, G. Case-control study of mineral concentrations of hoof horn tissue derived from feedlot cattle with toe tip necrosis syndrome (toe necrosis). Canadian Veterinary Journal 2018; 59:254-260.
7. Johnston, J. D., Eichhorn, D. J., Kontulainen, S. A., Noble, S. D., & Jelinski, M. D. Investigation of white line separation under load in bovine claws with and without toe-tip necrosis. American Journal of Veterinary Research 2019; 80:736-742.
8. Kofler, J. Clinical Study of Toe Ulcer and Necrosis of the Apex of the Distal Phalanx in 53 Cattle. The Veterinary Journal 1999; 157:139-147.

9. Kofler, J. Pathogenesis and Treatment of Toe Lesions in Cattle Including “Nonhealing” Toe Lesions. Veterinary Clinics of North America: Food Animal Practice 2017; 33:301-328.
10. Paetsch, C., Fenton, K., Perrett, T., Janzen, E., Clark, T., Shearer, J., & Jelinski, M. Prospective case-control study of toe tip necrosis syndrome (TTNS) in western Canadian feedlot cattle. Canadian Veterinary Journal 2017; 58:247-254.
11. Penny, C., Bradley, S., & Wilson, D. Lameness due to toe-tip necrosis syndrome in beef calves. http://veterinaryrecord.bmj.com. Accessed July 11, 2020.
12. Shearer, J. K., Plummer, P., & Schleining, J. Perspectives on the treatment of claw lesions in cattle. Veterinary Medicine: Research and Reports 2015; 6:273-292.