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# Tetanus in Cattle: Review and Case Description of Clinical Tetanus in a Holstein Heifer

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## Abstract

Tetanus (*Clostridium tetani* infection) is a potentially life-threatening neurologic disease affecting cattle. Clinical signs of tetanus are frequently unrecognized until advanced stages of the disease, when treatment and management of affected animals are difficult and prognosis is relatively poor. This case report demonstrates the successful early diagnosis and subsequent management of wound tetanus in a 13-month-old Holstein heifer. Primary clinical signs that led to an accurate diagnosis and early treatment included stiff gait, erect tail, and prolapsed nictitans. Intensive medical management yielded a relatively rapid improvement in clinical signs. The heifer calved and entered the lactating herd. Practitioners that observe the previously mentioned clinical signs in cattle should consider tetanus as a primary differential diagnosis and initiate aggressive medical management.

**Keywords:** clostridium, tetanus, wound, bovine, diagnosis, treatment

## Résumé

Le tétanos (infection au *Clostridium tetani*) est une maladie neurologique potentiellemenet mortelle qui affecte les bovins. Les signes cliniques du tétanos passent souvent inaperçus avant que la maladie n'atteigne un stade avancé. Or, à ce moment, le traitement et la gestion des animaux affectés sont difficiles et le pronostic défavorable. Le cas présenté ici fait état d'un diagnostic effectué suffisamment tôt et du traitement réussi d'une blessure infectée au tétanos sur une génisse Holstein de 13 mois. Les premiers signes cliniques qui ont permis un diagnostic exact et un traitement rapide étaient : une démarche raide, une queue dressée et le prolapsus de la membrane nictitante. Le traitement médical intensif qui fut appliqué a permis l'amélioration rapide des signes cliniques. La génisse a vêlé et fut intégrée aux autres vaches en lactation du troupeau. Les vétérinaires qui observent sur des bovins les signes cliniques mentionnés ci-haut devraient considérer le tétanos en premier lieu pour le diagnostic différentiel et amorcer un traitement médical approprié des plus énergiques.

## Introduction

Within the bovine veterinary community, bacterial pathogens are dealt with routinely; therefore, it is easy to be complacent regarding infections that appear to be under control or are relative clinical rarities. Infection with *Clostridium tetani*, resulting in tetanus, has largely been controlled by adoption of husbandry practices that reduce risk of infection. Since *C. tetani* spores are prevalent in the environment, the potential for wound contamination will always be present, and astute recognition of tetanus clinical signs is important. Diagnosis of tetanus in early stages by the practitioner is based entirely on detecting relatively subtle clinical signs. This case report provides a review of tetanus diagnosis and treatment, then describes the diagnosis and subsequent case management of a 13-month-old Holstein dairy heifer with wound-induced tetanus.

## Review of *Clostridium tetani* Infection

*Etiology and Pathogenesis*

The neuroparalytic syndrome of tetanus is caused by exotoxins produced by the gram-positive bacillus, *Clostridium tetani*.[2,17] It is a motile, spore-forming obligate anaerobe.[2,16] *C. tetani* is ubiquitous in soil worldwide, but may also be isolated from the feces of domestic animals.[2,16] Under aerobic conditions, *C. tetani* produces hardy spores that may survive in soil for years.[3] These spores are resistant to many standard disinfection procedures, including steam heat at 212°F (100°C) for 20 minutes.[10] Germination of *C. tetani* spores in anaerobic environments, such as devitalized tissue, results in rapid growth during the vegetative stage. During rapid growth, the bacteria produce several exotoxins, including tetanospasmin, the tetanus neurotoxin (TeNT) responsible for causing clinical disease.[2,10] TeNT is a 150-kd protein with three active domains that each contribute to

the overall effect on the patient.[16] *C. tetani* also produces tetanolysin, an exotoxin that has a necrotizing effect on tissues, and may serve to further decrease tissue oxygenation to allow further proliferation of the bacteria.[16]

The most common infection sites in cattle for *C. tetani* include deep, necrotic wounds, either traumatic or surgical, necrotic lesions of the vulva or vagina following dystocia, and severe postpartum metritis.[3] Surgical wounds that may predispose to germination of *C. tetani* spores include those following dehorning, castration, nose ring placement, tail docking, and ear tag placement.[3,18,20] The site of infection may never be identified in some cases.[3] The incubation period, or time from injury to first clinical signs, averages seven to 10 days but may be as long as 60 days.[2]

Tetanus is usually a disease affecting individual animals,[18] but two older case reports describe outbreak situations involving *C. tetani*.[6,15] The first report describes an outbreak affecting an entire group of 48 yearling Angus-Jersey cross heifers and steers grazing on a pasture of lush clover and dry millet stalks. The second case report describes two separate instances of Shorthorn heifers grazing sorghum stubble pastures and subsequently demonstrating classic clinical signs of tetanus. Both case reports demonstrate the potential for gastrointestinal absorption of TeNT following overgrowth of *C. tetani* in the stomachs of affected cattle. The most recent published case describes tetanus in a group of feeder calves following dehorning or band castration.[9] A group of 140 calves were processed, and three weeks later 11 of the calves exhibited tetanus signs ranging from prolapse of the nictitans to stiff gait and erect tail.[9]

The target of TeNT is inhibition of neurotransmission, which is the same target of toxins produced by *Clostridium botulinum*.[17,19] From the site of production or absorption, TeNT diffuses in body fluids until it reaches the presynaptic membrane of cholinergic nerve terminals, where it binds irreversibly.[17] Then, TeNT is internalized and transported retroaxonally to spinal cord inhibitory interneurons.[17] Transport to interneurons occurs first in motor neurons and later in sensory and autonomic nerves.[2] Inhibition of neurotransmitter release, or neuroexocytosis, in these interneurons is caused by specific cleavage of a group of proteins called SNARE proteins (soluble n-ethylmaleimide sensitive factor attachment protein receptor), which are integral to the exocytosis process.[19] Within spinal inhibitory interneurons, cleavage of these SNARE proteins by TeNT blocks the release of vesicular contents to the extracellular environment,[19] thus preventing the release of neurotransmitters.[16] The resulting spastic paralysis, demonstrated by stiff gait, prolapsed nictitans, and other clinical signs, is due to a loss of spinal inhibitory control of motor neuron activity.[17] The binding of TeNT to nerve tissue is considered irreversible, and recovery of the patient only occurs with the growth of new nerve terminals.[17] This process may take days to weeks, and explains the variable duration of clinical signs in affected animals.[16]

*Clinical Presentation*

Clinical signs of tetanus in cattle are varied and range from mild to severe on presentation.[3] The first detectable clinical signs of tetanus in cattle are usually generalized stiffness and reluctance to move.[1] As the disease progresses, a change in gait is detected; a stiff, stilted walk is classic for tetanus in cattle.[3,16] A "sawhorse" stance is typical in affected cattle due to extensor muscle rigidity.[3] On further physical examination, the practitioner will frequently note while taking a rectal temperature that the animal's tail is stiff and raised away from the perineum.[3] This "pump-handle" tail is frequently seen in cattle due to involvement of the coccygeal muscles.[16] Affected cattle may display an anxious expression with head extended, nostrils flared, erect ears pulled toward the poll, and eyelids held widely open.[3,16] Prolapse of the nictitans, due to spasm of the retractor oculi muscles pulling the globe into the bony orbit and allowing passive prolapse of the nictitans, is frequently noted.[3] The muscles of mastication may be involved so that attempts to open the mouth are met with extreme rigidity of the jaws.[3] Affected cattle usually lose the ability to eat and may also lose the ability to drink, resulting in progressive dehydration.[3] Rumen contractions are usually weak or absent as the disease progresses. Cattle frequently will bloat due to failure of eructation.[3]

Subtle muscle tremors may be detected early, but these become more pronounced in later stages of tetanus.[1] Tetanic convulsions may be triggered by external stimuli, such as loud noises or handling of the animal, but in advanced cases these convulsions may occur spontaneously.[1] As the disease progresses, animals may become recumbent and unable to rise due to extension of their rear legs and the inability to pull their legs underneath them to rise.[16] If left untreated, animals may become severely dehydrated and fall into a self-destructive cycle of lateral recumbency, extensor rigidity, pain, panic, and exertion.[3] Death in cattle with tetanus may result from exertion and respiratory failure or from bloat-induced aspiration pneumonia. Furthermore, musculoskeletal injuries, such as long bone fracture or hip luxation, may necessitate euthanasia.[3]

*Diagnosis*

The distinctive clinical signs of tetanus detected during thorough physical examination of cattle are usually sufficient to make a diagnosis.[3,16] There are no reliable clinicopathologic tests to confirm a diagnosis of tetanus,[18] and no significant effort has been made to

develop an immunodiagnostic test.[1,16] An ELISA test was developed to detect TeNT at a level of 1.2 ng/ml, but TeNT is so powerful that detection at the picogram level would be necessary to be clinically useful.[16] Differential diagnoses include hypomagnesemia, polioencephalomalacia (PEM), bacterial meningitis, nervous coccidiosis, and lead encephalopathy. Clinical hypomagnesemia and PEM in cattle may result in tetany and convulsions, but prolapse of the nictitans or rumen tympany do not occur.[1] Bacterial meningitis in cattle usually manifests clinically by fever, cutaneous hyperesthesia, and rigidity of the muscles. This disease is more common in neonatal calves, but primary meningitis is extremely rare in mature cattle except in cases associated with *Listeria monocytogenes* or *Histophilus somni*.[13] While the nervous form of coccidiosis may cause muscular tremors, hyperesthesia, and convulsions, a fecal examination for detection of oocysts may be diagnostic.[12] Lead encephalopathy in ruminants leads to staggering, head pressing, muscle tremors, champing of the jaws, and convulsions, but elevated lead levels in blood, urine, or milk are diagnostic. On physical examination, lead encephalopathy patients are frequently blind and are unlikely to display prolapsed nictitans, stiff gait, or trismus that are characteristic of tetanus.[11]

*Treatments*

Favorable outcomes in tetanus cases depend on early detection of the classic clinical signs and institution of treatment.[16] Cattle appear to respond better to aggressive treatment than horses and sheep.[1] Advanced cases are unlikely to respond to therapy and euthanasia should be considered.[1,3] Basic principles for medical management of tetanus cases in cattle are: provide muscular relaxation, neutralize the unbound TeNT toxin, establish active antitoxic immunity, eliminate the *C. tetani* infection, maintain hydration and nutritional status, and provide good footing with deep bedding.[16,18]

Excitement, tetanic episodes, and the associated pain must be minimized in the tetanus patient.[3] The animal should be placed in a dark, quiet stall, and interaction with the patient should be kept to a minimum.[16] Tranquilization is very helpful in managing tetanus patients during administration of treatments.[3] Acepromazine (0.023 to 0.045 mg/lb or 0.05 to 0.1 mg/kg IV/IM q 4-12 h) is useful for this purpose.[3,16,18] Diazepam (0.0045 to 0.18 mg/lb or 0.01 to 0.4 mg/kg IV q 3-12 h) may effectively reduce muscle spasms, but prolonged administration to cattle is expensive.[18] Guaifenesin (5% solution) may lessen muscular spasm by blocking nerve transmission at the level of the spinal cord interneurons, and should be administered intravenously to effect.[16]

Tetanus antitoxin cannot counteract the effects of toxin bound to receptors, but may bind any circulating or unbound toxin.[3] The dose for tetanus antitoxin has a widely reported range from 1,500-100,000 U per animal per day.[16] As a standard treatment for adult cattle, one text recommends 15,000 U once or twice as initial therapy.[3] If the site of the infection has been identified, then local injection of the antitoxin may be of value.[1] It is recommended that antitoxin be administered locally prior to debridement and irrigation of the wound to avoid facilitating further absorption and binding of TeNT.[10] The concentration of TeNT required to cause neurologic signs may or may not stimulate an active immunologic response.[3] Affected animals should be immunized with tetanus toxoid at the time of initial treatment and one to two months later.[18] Simultaneous injections of tetanus toxoid and antitoxin should be made at different sites, and must not be mixed prior to administration.[18]

The infection site that resulted in vegetative growth of *C. tetani* must be addressed. If the wound or site of infection is readily identified, then it must be cleaned and debrided to remove necrotic tissue.[3] Opening the site to oxygen or irrigating the wound with hydrogen peroxide will reduce toxin production by eliminating bacteria in the stage of vegetative growth.[10,16] Systemic antibiotics that have a gram-positive spectrum should be administered at high doses.[16] Primarily, penicillin (10,000-20,000 IU/lb or 22,000-44,000 IU/kg IM q 12 h) is used for treatment of bovine tetanus patients and should be administered for at least seven days.[1,8,18]

Good nursing care is very important during medical management of tetanus patients. The animal must be placed in a stall with deep bedding and good footing to prevent decubital sores and difficulty rising.[18] The animal's hydration status should be monitored closely, and animals may require intravenous fluid support. Recumbent cattle should be maintained in a sternal position to prevent rumen tympany.[16] If the patient develops free gas bloat requiring stomach tubing, then a rumen fistula should be placed to relieve rumen gas until the patient regains the ability to eructate.[3] A fistula can also provide a portal for the delivery of water and feed to the rumen.[3]

The mortality rate may reach 50% in cattle, but animals that survive longer than seven days have a fair-to-good chance of complete recovery.[18] Mild cases usually respond to treatment within one week, but it is important to realize that many patients may stabilize and subsequently develop unforeseen complications that result in death or necessitate euthanasia.[3] Regaining the ability to drink is one of the most encouraging signs of recovery.[3] Cattle that degenerate to a state of lateral recumbency usually require euthanasia.[3]

*Prevention and Control*

While cattle are reported to be less susceptible to tetanus than other farm animals, it is important to reduce the risk of cattle developing wounds or necrotic

sites where *C. tetani* spores may germinate. Elective procedures that could lead to open wounds include dehorning, band castration, nose ring placement, tail docking, and ear tag placement.[3,18,20] It is important that surgeons maintain clean surgical conditions and use sterile instruments for each procedure. Cattle are not routinely vaccinated against tetanus,[18] and most multi-clostridial vaccines for cattle do not contain specific protection against *C. tetani*.[9] On farms or in geographical areas where tetanus is a problem, cattle can be vaccinated easily and inexpensively with tetanus toxoid.[3] Protective antibody levels usually occur about two weeks following the booster injection of the primary series.[7] Tetanus antitoxin is recommended for immediate, emergency, passive treatment of exposed animals when clinical tetanus is expected.[7]

## Case Report

*History*

A 13-month-old Holstein dairy heifer was originally examined on February 25, 2009, after the farm manager noticed that the heifer had been behaving abnormally and appeared lame. Signs were of two days' duration. At the time of initial evaluation, the heifer was nearing breeding age and was due for enrollment in a prostaglandin reproductive synchronization program in early March. The farm's records revealed that the heifer had received routine vaccinations for bovine respiratory disease, leptospirosis, and clostridial pathogens about six months prior to this initial evaluation. No treatments had yet been administered by the farm staff.

*Clinical Findings*

While moving the heifer into a chute for restraint, it was apparent that the tail was rigidly extended and the heifer's gait was markedly stiff, especially in the hind limbs where the stride was visibly shortened. No lameness was apparent. Also, the heifer's abdomen was contracted, causing a tucked appearance. Using a Holstein-specific weight tape, the heifer's weight was estimated at 770 lb (350 kg). Physical examination revealed a quiet, alert heifer with a relatively low body condition score (2.75/5). Vital signs were within normal limits, and the heifer was mildly dehydrated. Its abdomen was not distended, and one moderate rumen contraction per minute was auscultated. There was a deep, 3.2 inch (7 cm) laceration over the medial aspect of the distal left metatarsus (MT III) with associated soft tissue swelling extending distally. The wound appeared old, and a moderate amount of malodorous, grey purulent material discharged from the wound after the scab was peeled back. Examination of the heifer's head revealed an anxious expression, erect ears, bilateral prolapse of the nictitans, and moderate trismus. The heifer had packed a significant amount of cud material in its mouth due to limited ability to masticate and swallow.

The initial problem list included an old, contaminated wound over the distal left MT III and clinical signs of *C. tetani* infection, including distinctive signs of muscle spasms and hyperesthesia. Patient signalment, physical examination findings consistent with *C. tetani* infection, and the presence of a contaminated wound left a short differential diagnosis list: tetanus, hypomagnesemic tetany, bacterial meningitis, nervous coccidiosis, and unusual presentation of PEM or lead toxicity. While hypomagnesemic tetany is a common occurrence in ruminants,[14] the heifer was kept in closed housing and fed a balanced total mixed ration (TMR), thus making hypomagnesemia doubtful. Since bacterial meningitis is extremely rare in mature cattle, except in cases associated with *Listeria monocytogenes* or *Histophilus somni*,[13] this disease was ruled out. The nervous form of coccidiosis was unlikely because the heifer's TMR diet included an ionophore coccidiostat. While PEM and lead encephalopathy may cause muscle tremors, trismus, and stiff gait, the heifer's consistent TMR diet and housing precluded these differentials from the list.

Infection with C. *tetani* was the most likely cause of the following clinical signs: stiff gait, tucked-up abdomen, prolapsed nictitans, erect tail, and moderate trismus. Aggressive medical management, evaluation of blood chemistry and hematology values, and radiographic evaluation of the left hind fetlock were recommended to the farm manager; at that time it was not possible to provide an accurate prognosis, even with an aggressive diagnostic and treatment plan. Due to the uncertainty of the situation, the farm manager consented to medical management for tetanus, but elected not to pursue radiographs or diagnostic blood work until the heifer showed improvement.

*Therapeutic Management*

An aggressive medical management treatment plan was initiated on February 25, 2009. First, the heifer was tranquilized with 7 mg acepromazine intramuscularly to provide muscle relaxation and to control hyperesthesia. This treatment reduced the heifer's response to external stimuli and made treatments less traumatic. Next, 15,000 U of tetanus antitoxin was administered. Half the dose was administered by single subcutaneous injection in the left neck and the other half was administered subcutaneously in the tissues proximal to the medial laceration of distal left MT III. It was considered important to administer the antitoxin prior to cleaning and flushing the wound. To initiate active antitoxic immunity, a dose of tetanus toxoid was administered intramuscularly.

The distal MT III wound was clipped and flushed using dilute povidone iodine solution. A teat cannula flushing system allowed exploration of the extent of the

wound; it was found to be at least 1.4 inches (3 cm) deep and down to bone. In order to oxygenate the deeper tissues in the wound, it was irrigated with 3% hydrogen peroxide. The wound was left with no bandage or cover to allow further oxygenation and drainage. To provide broad-spectrum antimicrobial coverage, the heifer was treated with systemic procaine penicillin (15,900 IU/lb or 35,000 IU/kg IM q 24 h) for seven days and a single injection of long-acting ceftiofur[a] (3 mg/lb or 6.6 mg/kg SQ). Flunixin meglumine (0.68 mg/lb or 1.5 mg/kg IV q 24 h) was administered for three days to provide pain relief. The packed cud material was flushed from the heifer's mouth. During this procedure, the heifer was observed to willingly and successfully swallow a good portion of the flush water during each flushing event. Lastly, the heifer was returned to a box stall deeply bedded with chopped straw. The farm staff were instructed to consult the veterinarian if the heifer's clinical signs deteriorated or if other problems arose, such as bloat or inability to rise and ambulate.

The next day, the farm staff tranquilized the heifer with 7 mg acepromazine intramuscularly to make follow-up treatment easier. Following sedation, second doses of tetanus antitoxin (15,000 U), systemic procaine penicillin, and flunixin meglumine were administered. The heifer's mouth was flushed with water and a significant amount of cud material was removed. The heifer again drank a good portion of flush water during each flushing event. It had no interest in TMR or hay at this time and was returned to a deeply bedded box stall.

While the farm staff continued systemic procaine penicillin treatments, periodic re-examinations were performed to monitor the heifer's progress and observe for such complications as rumen bloat, aspiration pneumonia, or musculoskeletal injury. Table 1 summarizes the physical examination findings and progression of the heifer's clinical signs during the first week of treatment. The most notable changes in the heifer's status were a reduction in the rigidity of the jaws and subsequent lack of cud material packed into its mouth. At the same time, the heifer was showing increased interest in TMR and hay. Improvement in the nictitans, less elevation and stiffness of the tail, and an improvement in gait were also noted. At the end of the first week, the heifer had a mild fever (102.7°F; 39.3°C), likely related to the leg wound.

As a result of the improvements, the farm manager consented to radiographs of the left hind fetlock and limited blood work. On March 4, the heifer's serum fibrinogen was moderately elevated (1,060 mg/dl; Table 2), but consistent with the deep, penetrating fetlock wound.

Digital radiographs of the fetlock were taken on the same date and demonstrated a focal osteomyelitis of the medial aspect of distal MT III with associated soft tissue swelling, dystrophic mineralization in the overlying soft tissue, and a soft-tissue defect consistent with the history of focal trauma. The radiographs provided no evidence of osseous sequestration, foreign body, fractures, or other problems.

Next, the wound was scrubbed and flushed with dilute povidone iodine solution. At this time, the wound did not appear as deep and granulation tissue was present; therefore, it was no longer possible to penetrate to bone with the teat cannula flushing system. The wound was again irrigated with hydrogen peroxide and left uncovered for drainage. In order to maintain broad-spectrum antimicrobial coverage, a second dose

**Table 1.** Progression of heifer's clinical signs at each re-examination in the first week following diagnosis of tetanus. An increased number of check marks (✓) indicate a higher degree or more severe manifestation of the clinical sign.

| Clinical signs | Day 0 (2/25/09) | Day 2 (2/27/09) | Day 3 (2/28/09) | Day 5 (3/2/09) |
|---|---|---|---|---|
| Fever | | ✓ (102.5°F) | | |
| Respiratory rate (breaths/min) | 28 | 32 | 30 | 30 |
| Rumen contraction | 1/min Moderate | 1/min Weak | 1/min Weak | 1/min Moderate |
| Rigidity of the jaws | ✓✓✓ | ✓✓ | ✓ | ✓ |
| Cud material packed in mouth | ✓✓ | ✓ | Resolved | Resolved |
| Eating TMR | | | ✓ | ✓ |
| Stiff gait | ✓✓ | ✓✓ | ✓✓ | ✓ |
| Prolapsed nictitans | ✓✓ | ✓✓ | ✓✓ | ✓ |
| Elevated tail head | ✓✓ | ✓✓ | ✓ | ✓ |

**Table 2.** Progression of heifer's clinical signs at each re-examination in the weeks following diagnosis of tetanus, including day 0. An increased number of check marks (✓) indicate a higher degree or more severe manifestation of the clinical sign.

| Clinical signs | Day 0 (2/25/09) | Day 7 (3/4/09) | Day 9 (3/6/09) | Day 13 (3/10/09) | Day 20 (3/17/09) |
|---|---|---|---|---|---|
| Fever | | ✓ (102.7°F) | | | |
| Respiratory rate (breaths/min) | 28 | 32 | 28 | 30 | 30 |
| Rumen contraction | 1/min Moderate | 2/min Moderate | 2/min Moderate | 2/min Moderate | 2/min Strong |
| Rigidity of the jaws | ✓✓✓ | ✓ | ✓ | Resolved | Resolved |
| Cud material packed in mouth | ✓✓ | Resolved | Resolved | Resolved | Resolved |
| Eating TMR | | ✓✓ | ✓✓ | ✓✓ | ✓✓✓ |
| Stiff gait | ✓✓ | ✓ | ✓ | Resolved | Resolved |
| Prolapsed nictitans | ✓✓ | ✓ | ✓ | Resolved | Resolved |
| Elevated tail head | ✓✓ | ✓ | ✓ | Resolved | Resolved |
| Serum fibrinogen (mg/dl) | | 1,060 | | | 802 |

of long-acting ceftiofur (3.0 mg/lb or 6.6 mg/kg SQ q 7 d) was administered, and staff were instructed to continue systemic procaine penicillin treatments for seven more days. Because of the heifer's low-grade fever and the wound management procedures performed, another dose of flunixin meglumine was administered (0.68 mg/lb or 1.5 mg/kg) IV. When released from restraint, the heifer ate TMR for 10 to 15 minutes.

While the farm staff continued penicillin treatments, periodic re-examinations were performed to monitor the heifer's progress and assess for complications. Table 2 shows the progression of clinical signs at each re-examination in the weeks following diagnosis, including day 0. The most notable changes in the heifer's status included improved appetite and resolution of jaw rigidity, gait stiffness, prolapse of the nictitans, and elevation and stiffness of the tail. By the end of the second week of treatments, the heifer had resolved all clinical signs of tetanus and penicillin treatments were discontinued. However, the leg wound continued to drain purulent material and was only marginally contracted, compared to the prior week. Therefore, a week-long course of systemic florfenicol was initiated (9.1 mg/lb or 20 mg/kg IM q 48 h).

By March 17, three weeks after initial presentation, no clinical signs of tetanus were observed. The heifer's rectal temperature was normal, and appetite was excellent. The wound appeared to be healing well, with significant contraction of the wound margins and no further discharge noted. Further radiographic and serum fibrinogen evaluations were conducted to plan future antimicrobial treatments. The heifer had mild hyperfibrinogenemia (802 mg/dl), which was considerably improved from the previous evaluation (Table 2).

Radiographs of the heifer's left hind fetlock demonstrated progressive healing of the focal osteomyelitis. Based on its clinical signs, progressive wound healing, decreased serum fibrinogen, and improved radiographic findings, systemic antimicrobial treatments were discontinued.

The heifer was moved out of a box stall and returned to the breeding group. A booster dose of tetanus toxoid was administered five weeks after the initial dose. No further clinical signs of tetanus were observed in this animal, and it entered the farm's routine breeding management program. The heifer subsequently carried a calf to term and entered the lactating herd.

## Discussion

This case represents the typical early clinical presentation of tetanus in cattle. Clinical diagnosis of *Clostridium tetani* infection was made early, and the heifer was able to return to normal function following medical management. A case report by McGuirk provides a similar history, and is another good example of the importance of early diagnosis of tetanus.[8] A two-year-old Holstein heifer in that report was sent to a referral hospital following a week-long treatment of rumen tympany. Two treatments of oral poloxalene were unsuccessful at relieving the bloat, and the heifer was reported to walk stiffly to the trailer for transport to the hospital. On ar-

rival the heifer was recumbent, and examination of the limbs revealed a healing laceration of the right forelimb with persistent swelling around the wound. Despite aggressive medical management, the heifer died due to respiratory failure within 24 hours of arrival at the hospital. It is possible that earlier identification of the wound or more thorough examination of this animal in the field to identify additional signs of tetanus beyond ruminal tympany may have yielded a more positive outcome.

One text reports it is not uncommon for tetanus cases to be misdiagnosed by veterinarians.[3] As with McGuirk's case report, a common feature of misdiagnosis is rumen tympany. To avoid overlooking tetanus in the early stages, close observation of the animal's gait and head is recommended.[3] Early cases of tetanus in cattle frequently demonstrate a stiff gait or sawhorse stance. It is important to release confined cattle to make this evaluation. Examination of the head may reveal passive prolapse of the nictitans and trismus. In the case reported here, these signs were all present and led to rapid diagnosis and initiation of medical management. Early detection and aggressive treatment of this tetanus case, where there was diminished swallowing and rumen motility, contributed to the favorable outcome.

Identifying the source of *C. tetani* infection in animals with clinical signs of tetanus is important so that wounds can be cleaned and debrided. While the site of infection may never be identified in a significant proportion of cases, the most common infection sites in cattle are generally deep, penetrating wounds, either iatrogenic or traumatic.[3] Necrotic lesions of the vulva or vagina and severe metritis in the postpartum period are common infection sites in cattle.[3] The distal MT III wound in this case was the obvious source of *C. tetani* infection, and the wound was subsequently debrided and irrigated to kill *C. tetani* in the vegetative growth stage. Identifying the wound site allowed for enhanced use of tetanus antitoxin proximal to the wound prior to debridement and irrigation. The heifer returned to normal function, as noted in Table 2.

Medical management of this case followed accepted medical principles. Tetanus antitoxin was administered subcutaneously and at the site of the wound prior to debridement, and the animal was treated with procaine penicillin. Because the heifer never bloated or became recumbent, the decision was made to manage the case on site. This heifer never lost the ability to drink and was able to maintain hydration during the recovery period. If the heifer had become bloated or severely dehydrated, the farm manager indicated she would consent to placement of a rumen fistula. Clinical signs of tetanus began to resolve within one week of treatment initiation, and clinical signs were fully resolved with two weeks of treatment. This represents typical case progression in cattle diagnosed early and treated aggressively.

Following radiographic evaluation of the leg wound, a diagnosis of focal osteomyelitis of the medial aspect of distal MT III was made. Following two weeks of systemic penicillin and ceftiofur treatment, the wound was still draining purulent material; therefore, treatment was changed to systemic florfenicol, which represents an extra-label drug use. Currently there is no labeled antimicrobial for treatment of osteomyelitis lesions in cattle. Florfenicol has been reported as a suitable antimicrobial for treatment of vertebral osteomyelitis in cattle,[4] and a recent study demonstrated the pharmacokinetics of florfenicol in synovial fluid after regional intravenous perfusion of the hind limb of adult cows.[5] The authors suggested that florfenicol delivered by regional intravenous perfusion may be a suitable treatment for deep digital sepsis and similar lesions in cattle.[5] Following florfenicol treatment in this case, the limb wound contracted and purulent discharge ceased.

When procaine penicillin is used at an extra-label dose for an extended period, such as in this case, it is imperative that the veterinarian recommend an extended pre-harvest withdrawal time before the animal can be harvested. The withdrawal time was not an issue in this case because the heifer regained full function and later entered the milking herd.

## Conclusions

The Holstein heifer in this case report demonstrated typical early signs of tetanus in cattle and subsequently responded to aggressive medical management. While cattle are relatively resistant to tetanus, it is important that veterinarians perform a complete physical examination of cattle presenting with relatively nonspecific signs, such as stiff gait or rumen tympany, to avoid misdiagnosing tetanus in the early stages. If bovine tetanus cases are diagnosed early and an aggressive treatment plan is initiated, cattle may make a full recovery. The heifer in this case had an uneventful pregnancy, transition period, and early lactation, peaking at 85 lb/day (38.6 kg/day) on a three-times-a-day milking schedule.

## Endnote

[a]Excede, Pfizer Animal Health, New York, NY


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