Clostridial Tetani Infection in a Yearling Arabian Colt

(Blacky's Spaz Attack)

Lensey Watson

Mississippi State University

College of Veterinary Medicine

Class of 2021

Clinicopathologic Conference

January 08, 2021

Advisor:

Dr. Cathleen Mochal-King, DVM, MS,DACVS

Introduction:

Tetanus is caused by *Clostridium tetani*, a gram-positive spore-forming bacterium commonly found in the soil. Tetanus was first recognized by Greek physicians more than 20 centuries ago identified as a neurological disease based on the Greek word *tetanos* which means to contract.[1] Of the large animal species, horses are the most sensitive, followed by small ruminants and cattle which are the most resistant to the toxin.[2] Tetanus is characterized by muscular rigidity and spasms near the infected wound.[3] Furthermore, generalized tetanus is characterized by a "sawhorse" stance, with an extended rigid tail and a stiffened gait, hyperesthesia progressing to generalized tonic contractions with opisthotonus even with mild stimulation.[3] Respiratory failure secondary to respiratory muscle spasms can result in death.[3]

History and Presentation:

A 1-year-old Arabian colt presented to Elgin Veterinary Hospital in Elgin, Texas on August 25, 2020 for suspected tetanus. According to the owners, the colt did not experience any cuts or castration event and had be administered one tetanus toxoid vaccination approximately 6 months ago. The owners reported that they noticed him to be stiffer than normal approximately 1 week prior to presentation.

On presentation, the colt was bright, alert, responsive and hyper-reactive. He weighed approximately 506 pounds (230 kilograms) with a body condition score of 4/9 (ideal being 5). Physical exam revealed a heart rate of 66 beats per minute, respiratory rate to 20 breaths per minute and a temperature of 100.4 degrees Fahrenheit. His hydration status was within normal limits with a capillary refill time of less than 2 seconds and pink moist mucous membranes. Normal borborygmi in all four quadrants of his gastrointestinal tract. On presentation packed cell volume of 42% (normal being 30-50%[4]) and a total protein of 8.2 g/dL (normal being 4.6 –

6.9[4]). A complete blood count was performed which had no significant findings present. The serum chemistry revealed hyperkalemia at 5.2 mEq/L, hypochloridemia, increase total bilirubin, increased aspartate aminotransferase (AST), and increased creatine kinase (CK). These values were collected using the Abaxis Vetscan HMii Hematological Analyzer.

Examination revealed that he was very hyper-esthetic especially when stimulated. He had a very stiff/stilted gait, rigid neck and pectoral muscles, prolapsed membrana nictitans especially when stimulated or menace response was tested. He had an erect tail, sardonic grin, erect ears, and excessive drooling/hypersalivation. Mentation was otherwise within normal limits. Based on these findings the colt was hospitalized so he could receive aggressive treatments and be placed in a quiet, dark isolated stall.

Diagnostic approach/Considerations:

Primary diagnosis of tetanus is based on patient history and physical exam findings. Gram stain and culture of *Clostridium tetani* is rarely performed in large animals.[2] This is due to its limited diagnostic value because the sporulated and vegetative form is difficult to differentiate from other anaerobic bacteria.[3] Bloodwork values are often unremarkable unless other secondary conditions are present. If the patient has a wound or aspiration pneumonia, then neutrophilic leukocytosis with a left shift may be observed.[3] If the patient has muscle trauma from sustained contracture and/or prolonged recumbency, then muscle enzymes (CK and AST) may be elevated.[3]

Pathophysiology:

Tetanus typically gains access via deep wounds, lacerations, surgical sites, umbilicus of neonatal foals, postpartum reproductive tracts, or site of unbroken skin or healed location by the

time clinical signs become apparent.[3] Tetanus is caused by *Clostridium* tetani, an anaerobic, gram positive, non-encapsulated spore-forming bacillus that typically takes 5 – 10 days after inoculation to cause clinical signs. Often times tetanus is nonpathogenic when ingested and it remains within the gastrointestinal tract but is highly pathogenic when inoculated in an anaerobic environment.[5] The tetanus toxin gains access to the nervous system via the cytoplasm of distal processes of neurons via endocytosis at viable nerve endings in the tissues near the site of the wound.[6] The toxin is then transported into the central nervous system via retrograde axonal transport and released into the interstitial fluids at the neuronal junctions through the process of exocytosis.[6] Free toxin binds to the cell membrane of inhibitory interneurons of the spinal cord via endocytosis and disrupts the inhibitory neurotransmitters.[6] While the inhibitory neurotransmitters (Gamma-aminobutyric acid and glycin) are being blocked by the tetanus toxin, Acetylcholine which is produced by the excitatory neuron also know as the upper motor neuron is still being produced.[6] This in turn leads to increased muscle contractions/spasms without the inhibitory phase to follow.

Clinical signs of tetanus are due to hyperactivity of voluntary (striated) muscles in the form of rigidity and spasms.[7] Tetanus is often categorized into either generalized or localized depending on the clinical signs present. Clinical signs of localized tetanus are in the form of muscle rigidity and spasms near the infected wound and progress to generalized tetanus.[3] Often times, localized tetanus is not observed and the patient is seen for more generalized clinical signs. Generalized tetanus presents with the more classic clinical signs of tetanus. These are the "sawhorse" stance, extended, rigid tail, stiff gait if ambulatory, enophthalmos, prolapsed membrana nictitans, rigidity of facial muscles resulting in risus sardonicus (sardonic grin), and dorsomedial retraction of the ears.[3] Other common clinical signs with generalized tetanus are

miosis, dysphagia, ptyalism, and laryngeal spasms.[3] Hyperesthesia typically leads to painful reflex muscle spasms that progress to tonic contractions with opisthotonos due to even mild stimulation.[3] Further disease progression leads to marked extensor rigidity of all four limbs making it impossible for voluntary movement causing recumbency.[3] Tetanus left untreated can lead to a variety of complications including decubital ulcers, regurgitation, dysuria, constipation, aspiration pneumonia, and/or death due to respiratory failure.[3]

Treatment, management and prevention options:

Tetanus must be treated aggressively and for a variable amount of time depending on severity and chronicity of the disease. Recovery is typically slow and only occurs once new interneuronal synapses develop to replace those inactivated by the toxin.[3] There are a variety of treatment options used to combat this disease process. Antibiotic therapy is initiated to eradicate the vegetative form of *Clostridium tetani*. Penicillin is the drug of choice and is recommended to use at the highest dosage.[3] Other antibiotics can also be effective for the treatment of tetanus. If there is a deep or contaminated wound, Metronidazole is commonly used because of its ability to penetrate necrotic tissues without losing efficacy.[3] Critical treatment includes the neutralization of any toxin that has not bound to the CNS.[3] This is achieved by the administration of the Tetanus antitoxin. The passively acquired antibodies neutralize unbound toxins circulating in blood and those present in the wound.[3] Although the patient has received the antitoxin, disease will continue to progress because once the toxin is internalized it cannot be neutralized by the antitoxin.[3] Depending on the severity of clinical signs and hyperactivity of the patient, sedation may be necessary to control muscle spasm and rigidity. Typical sedatives include phenothiazines which work at the level of the brainstem to depress descending excitatory input

on the lower motor neuron within the spinal cord.[3] Another consideration would be benzodiazepines, which are muscle relaxants that act as GABA antagonists.[3] Successful tetanus treatment depends on the quality of supportive care provided to the patient. Tetanus patients need to be placed in a dark, quiet environment, with minimal stimulation and handling.[3] Extended attention must be given to those patients that are recumbent, have hypertonic urethral and anal sphincters, lack effective abdominal press, have dysphagia, laryngeal spasm, or respiratory obstruction.[3] Often times the patients that experience generalized muscle contractions end up having hyperthermia that must be controlled.[3] Since, tetanus toxin concentrations do not generate protective immunity, horses must be immunized with tetanus toxoid at the beginning of the treatment process to initiate a protective antibody response.[3]

Based on the history provided and clinical signs noted on presentation, the colt was presumptively diagnosed with Tetanus. No wounds or deep tissue infections were identified as the cause of the infection. Initial treatment consisted of: 1) a tetanus toxoid intramuscular injection in the right pectoral muscle, 2) 10,500 units (7 vials) intramuscularly and 4,500 units (3 vials) subcutaneously of the tetanus antitoxin, 3) 40,000 IU/kg (~15mls) of Procaine Penicillin G intramuscularly in the right side of the neck, and 4) 6.6 mg/kg (~15mls) of Gentamicin intravenously. The colt was placed in a quiet, dark isolation stall with no access to water, hay, or grain. An intravenous catheter was placed and the colt was maintained on Sodium Chloride fluids with added dextrose at a rate of 40ml/kg/day to combat the hyperkalemia and provide adequate energy. These fluids were discontinued on day 3 of hospitalization due to possible phlebitis and recovery of clinical signs. On day 1 through 3 of hospitalization, the colt was treated with 15mls of Procaine Penicillin G intramuscularly twice daily, 15mls of Gentamicin intravenously once daily, and 22mg/kg (12 tablets) Metronidazole per rectum twice daily.

Tetanus antitoxin was administered throughout his stay at the hospital as well. On day 1 in hospital, he was administered 4,500 units (3 vials) subcutaneously. On day 2 in hospital, he was administered 3,000 units (2 vials) of tetanus antitoxin subcutaneously. On day 3 in hospital, he was administered 7,500 units (5 vials) of tetanus antitoxin subcutaneously.

The disease process could have ultimately been prevented with implementation of a proper vaccination protocol. Oftentimes, this includes initial vaccination of tetanus toxoid at 6 months of age and a boost at 1 year of age followed by annual boosters from this point forward. Unvaccinated horses that are diagnosed with tetanus should be administered both the tetanus toxoid and antitoxin. It is important to have clear client communication prior to administration of the antitoxin due to its rare, but documented side effect. That side effect includes idiopathic acute hepatic disease, also known as *Theiler's* disease, serum hepatitis, serum sickness, and post-vaccinal hepatitis.[3] Signs range from hepatic enzyme elevation to hepatic encephalopathy depending on severity.[3] If a horse contracts *Theiler's* disease, the prognosis becomes grave to poor.[3]

Case outcome:

The colt had an impressive, rapid recovery process while in the hospital. After 2 days of treatment, the colt was observed chewing and swallowing, moving around his stall, moving his ears toward the direction of sound, and had decreased salivation. At this point, the colt was offered a Senior mash and alfalfa leaves in small portions throughout the day. On the third day of hospitalization, the colt was bright, alert, responsive, eating well, and while stiff was walking around his stall well. During physical exam on day 3, it was noted that the colt had a temperature of 103.3 degrees Fahrenheit. At this time, a thoracic ultrasound was performed to

check for aspiration pneumonia. Fortunately, he did not have any acute signs of aspiration pneumonia such as consolidation or commit tails. His elevated temperature was attributed to potential phlebitis, so his fluids were discontinued and intravenous catheter was removed.

Unfortunately, due to financial constraints of the owner the colt was sent home after only 3 days of hospitalization. Prognosis was fair because of his rapid progress to eating and drinking well. His owner was also informed of the risk factors such as aspiration pneumonia and *Theiler's* disease that were still possible. He was informed that the benefits of using the tetanus antitoxin outweighed the potential, rare risk and to monitor for signs of liver disease such as inappetence and colic. The owner was instructed to provide a quiet, dark, well ventilated stall with a fan where he would not be startled by loud noises and light for at least 1 month. A second tetanus vaccination approximately 1 month post-presentation was recommended. The owner was also provided with strict feeding instructions that included feeding a Senior mash (few handfuls of Purina Equine senior added to water) and several handfuls of alfalfa leaves with added water fed as small frequent meals throughout the day and night. It is imperative for them to monitor closely for signs of aspiration pneumonia such as coughing, nasal discharge, inappetence, and an increased temperature at greater than or equal to 102 degrees Fahrenheit. The owner was informed that the stiffness is likely to take months to resolve and the importance of monitoring for worsening or inability to eat and swallow (drooling typically first sign observed) because of the risk of aspiration pneumonia. Unfortunately, the colt's condition at this time is unknown due to lack of communication between the client and the practice.

References:

1. Humeau Y, Doussau F, Grant NJ, et al: How botulism and tetanus neurotoxins block neurotransmitter release, *Biochimie* 82: 427-446, 2000.
2. MacKay J. Tetanus. In: Smith BP. Large Animal Internal Medicine. 5th ed. St. Louis. Saunders Elsevier, 2015; 996-998.
3. Morresey PR. Tetanus. In: Reed SM, et al. Equine Internal Medicine. 3rd ed. St. Louis. Saunders Elsevier, 2010; 637-641.
4. Southwood LL. Normal Ranges for Hematology and Plasma Chemistry and Conversion Table for Units. In: Southwood LL. Practical Guide to Equine Colic. 1st ed. John Whiley & Sons, Inc., 2013; 339-342
5. Linnenbrink T, McMichael M. Tetanus: pathophysiology, clinical signs, diagnosis, and update on new treatment modalities. J Vet Emergency and Critical Care 2006; 16 (3): 199-207
6. Zachary JF. Disorders of Domestic Animals Botulism and Tetanus. In: Zachary JF. Pathologic Basis of Veterinary Disease. 6th ed. St. Louis. Saunders Elsevier, 2017; 186-189
7. Hassel, B. Tetanus: pathophysiology, treatment, and the possibility of using botulinum toxin against tetanus-induced rigidity and spasms. Toxins 2013; 5, 73-83