DOWNER COW SYNDROME: DIAGNOSIS AND TREATMENT

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Senior Seminar Paper
Cornell University College of Veterinary Medicine
September 27, 2006

Abstract

A first lactation, two month fresh Holstein presented to the Cornell University Hospital for Animals for inability to rise. On physical exam she was bright and alert. She was placed in a floatation tank for presumptive neuromuscular injury. Every third day she was walked out of the float tank and into a stall, to assess whether she could rise on her own. If unable to rise, she was raised with hip lifters and then walked into the tank. Once up she was able to remain standing and walk without any obvious signs of lameness.

The primary causes of downer cows are numerous. All recumbent cows, regardless of the inciting cause are subject to pressure damage. This is termed secondary recumbency and is the unifying factor in downer cow syndrome. Treatment of downer cows involves first treating the initial cause of recumbency, if possible, and then attempting to decrease pressure damage. Treatment of downer cows is labor intensive, thus diagnosis of the cause of recumbency is important so labor expenditures can be focused on those animals with a reasonable prognosis of survival.

Case History and Signalment

"Enchant", a 2 year-old female, 2 month-fresh, first lactation Holstein, presented to Cornell University Hospital for Animals for inability to rise. Two days prior to arrival at Cornell Enchant became stuck underneath the neck rail of her tie stall. She was down for about 12 hours before rising and walking to a box stall. She remained in the box stall for the next day, getting up and down without difficulty. She displayed no signs of lameness while in the box stall. The following day she was found down in the barn gutter, after apparently escaping from the box stall. She was unable to rise throughout the day and remained down until presentation at Cornell University Hospital for Animals that evening.

Clinical Findings

On presentation, Enchant was bright, alert, and unable to rise. She was able to creep forward using her forelimbs. Her vital parameters were normal. A California Mastitis Test (CMT) and strip plate were negative in all four quarters. No crepitus or swellings were palpable in any joint. Several superficial skin abrasions were present over her spine. No swellings were noted over the vertebrae and no pain response was elicited upon palpation. Rectal exam revealed no hematomas or asymmetry of the pelvis. Enchant was treated with 20mg of dexamethasone, one 500ml bottle of calcium gluconate and intravenous fluids. She ate well overnight.

The next morning, Enchant was bright and alert, yet still unable to rise. She was found lying frog-legged in her stall. She was hobbled, rolled onto a mat and pulled into the float tank. Once the tank was full with water, Enchant was able to stand. For the first few hours in the tank, she was unwilling to bear weight continuously on her left hindlimb. As the day progressed she bore more weight on her left hindlimb and by evening she was standing equally on both hindlimbs. Once standing in the float tank she resumed eating. Enchant remained in the float tank for two days. She was treated with Spectramast LC, oxytetracycline intravenously, dexamethasone and intravenous fluids.

On day four the tank was drained. Enchant was backed out of the tank and into a stall. She remained standing for 5 minutes, before lying down. Superficial skin abrasions, most likely from the hobbles rubbing on her water-softened skin, were noted bilaterally on the cranial aspect of her fetlock and pastern joints. These abrasions were gently cleaned and allowed to air dry. The dexamethasone was discontinued and she was started on fluxinin meglumine. Enchant was unable to rise unaided. She was successfully raised with hip lifters in the early afternoon. Once up Enchant was able to walk without obvious signs of lameness. However, when standing she

arched her back. She remained standing and walking around her stall for 1.5 hours before laying down. She was again raised with hip lifters in the early evening and remained standing for two hours. Intravenous fluids were discontinued overnight.

On the morning of day five, Enchant was raised with hip lifters, led into the float tank and floated. Once she was in the tank, the hobbles were removed to help prevent further skin abrasions.

On day seven the tank was drained and Enchant walked out of the tank without any signs of lameness into a sand-bedded stall. She remained standing in her stall for 45 minutes and appeared reluctant to lie down. She no longer stood with an arched back. Once down she was unable to rise on her own. In the afternoon, she was raised with hip lifters and remained standing for a few hours. Later that evening she was able to rise with someone tailing her.

On day eight, the decision was made to float Enchant for another two days. Although Enchant was able to rise with assistance, it was not without difficulty. Returning her to the tank for another two days decreased the risk of potential injury as she struggled to rise. On day ten Enchant was walked out of the float tank and into a sand-bedded stall.

Throughout her time in the floatation tank Enchant remained bright and alert. After the first day of floatation, the tank was drained twice daily for milkings and then refilled with clean water. During milkings Enchant was able to stand unaided. She was frequently tachycardic when standing without floatation. Once the tank was full, her heart rate returned to a normal range (60-80 beats per minute). She was continually CMT negative in all four quarters, produced 50-60lbs of milk per day and ate very well. No asymmetry, swelling or crepitus was palpable in her hindlimbs.

Enchant remained in the hospital for another nine days after leaving the floatation tank. She continued to improve in her ability to rise; however if her hobbles were removed she would struggle to rise. On day 13, two soft swellings were noticed over her right shoulder and left stifle. These were diagnosed by ultrasound as hematomas, and were presumed to have occurred as she struggled to rise. Enchant was discharged to the care of her owners on day 19.

Diagnostic Tests

Enchant's hematocrit and total serum protein were mildly elevated on the day of presentation, indicating dehydration. An electrolyte panel was run at presentation and revealed no abnormalities. A CBC and chemistry panel, run on day 3 (the first day the lab was open), showed an elevated CK (12,225 U/L) and AST (1789 U/L). Elevations in CK and AST are indicative of skeletal muscle damage. A chemistry panel and CBC were repeated on day 8; the CK (1856 U/L) and AST (556 U/L) were elevated, but decreased in comparison to the previous results.

ISTAT panels were run periodically and revealed no abnormalities. Serum potassium was within normal limits. During her stay at the hospital, Enchant was negative for urine ketones.

Differential Diagnoses

The list of differential diagnoses can be divided into infectious, traumatic and metabolic categories. Infectious causes include; mastitis, metritis, massive sepsis, vertebral abscesses, claw diseases and lymphosarcoma. Traumatic causes include rupture of gastrocnemius tendon, coxofemoral luxation or fracture, myopathies, neuropathies, spinal cord trauma, calving paralysis and a wide variety of fractures. Hypocalcemia, hypokalemia, hypomagnesemnia and nervous

ketosis are metabolic causes of down cows. Massive hemorrhage can also result in a cow that is unable to stand.[2]

Gram-negative bacteria and *Staphylococcus aureus* are the most common causes of toxic mastitis, although *Arcanobacterium pyogenes* and environmental *Streptococcus* species are possible culprits.[2] Enchant's udder was normal on palpation and she was CMT and strip plate negative in all four quarters. Rectal exam revealed a small uterus with very little to no fluid. Thus, mastitis and metritis were ruled out as possible causes.

Metabolic derangements (hypocalcemia, hypomagnesemia, hypophosphatemia) resulting in downer cows typically occur within a few days of calving to one month post-calving. These animals usually present with abnormal vital signs and/or an altered state of consciousness.[3] Hypokalemia occurs most commonly within the first 45 days of lactation. Animals with hypokalemia are typically dull and depressed.[4] Enchant was bright, alert, and eating with normal vital parameters at presentation. Additionally, she was two months fresh making hypocalcemia less likely. Thus, metabolic problems resulting in recumbency were low on the differential list. An ISTAT panel to assess calcium level revealed no abnormalities and a separate chemistry panel revealed no electrolyte abnormalities. Thus, hypomagnesemia, hypocalcemia, hypokalemia and hypophosphatemia were ruled out as possible causes. Massive sepsis was ruled out on the basis of her normal vital parameters (temperature, heart rate and respiratory rate).

Based on the history, the most likely cause of Enchant's inability to rise was some type of traumatic injury, presumably due to slipping and falling into the barn gutter. A precise muscular/neurologic disease could not be determined on initial physical exam. Calving paralysis was ruled out because she was 2 months post-calving when signs occurred. Laminitis and severe claw injury were unlikely as her feet were not painful or abnormal. Possible differential

diagnosis included coxofemoral luxation, fracture of the femoral head, fracture of the pelvis, vertebral fracture or subluxation, vertebral abscess, rupture of the gastrocnemius tendon, swelling or damage to a nerve or muscle or compression of the spinal column or a spinal nerve.[5]

Enchant continued to improve throughout her stay in the floatation tank and was eventually able to rise on her own. Rupture of the gastrocnemius muscle typically results in a severely dropped hock with the point of the hock resting on the ground. The prognosis for these animals to return to function is generally hopeless.[5] Based on her continual improvement and her ability to stand normally, gastrocnemius rupture was ruled out. Lymphosarcoma of the spinal cord was unlikely given Enchant's age (2 year old) and her continual improvement. Coxofemoral luxation and fractures of the hip joint result in a cow that is reluctant to bear weight on the affected limb, short strided and advances the limb with an outward motion.[5] On the basis of these clinical signs hip dislocations or fractures were ruled out. Vertebral fractures, abscesses and lymphosarcoma were ruled out as Enchant continued to improve and the prognosis for animals with these injuries is grave.[5]

Muscular and nerve damage due to trauma and resultant swelling are the most likely cause of Enchant's inability to rise because these types of injuries will resolve with time. Additionally, Enchant's history fits with these potential diagnoses. As she had difficulty rising without hobbles on, it is likely that she damaged her adductor muscles by splaying when she fell into the barn gutter.

Discussion

Cox proposed that a downer cow be defined as "any cow that is down in sternal recumbency for more than 24 hours with no evidence of systemic disease". This definition intentionally excludes those cows in lateral recumbency, which are believed to be in the process

of dying, rather then a treatable case. More recently, Smith proposed that downer cows be defined as any cow unable to rise to a standing position.[6] The term "creeper" is often used to describe an animal that moves considerable distances using its forelimbs, but is unable to stand.[7] By these definitions, Enchant would be considered a downer cow and a creeper. To explain the pathogenesis of downer cow syndrome Cox divided it into three stages, primary, secondary and terminal recumbency.

Primary Recumbency

Primary recumbency results from a wide variety of conditions. Metabolic derangements, severe ketosis, toxic mastitis or metritis and the stress of parturition are systemic causes of recumbency. Non-systemic causes include lymphosarcoma, vertebral fracture, vertebral abscess and a variety of fractures, luxations, dislocations, myopathies and neuropathies resulting from trauma.[8] An important clue to the cause of primary recumbency is when the cow went down in relation to calving. Hypocalcemia is the most common cause of primary recumbency in dairy cattle, occurring most frequently within a day of calving.[9] Calving paralysis, a common local cause of recumbency, results from damage to the obturator nerve and the lumbar root (L6) of the sciatic nerve as the calf passes through the birth canal. [10]

After milk production peaks, metabolic problems are less likely to be the cause of primary recumbency, and non-systemic causes should be suspected. Animals suffering from non-systemic primary causes of recumbency are typically bright, alert and eating, whereas those suffering from systemic causes are typically non-alert. This generalization however, does not always hold true and an animal can be suffering from multiple causes resulting in an inability to rise.[10]

Secondary Recumbency

Regardless of the primary cause of recumbency, once down all animals are subject to tissue compression, leading to ischemia of the muscles and nerves. Secondary recumbency, a result of pressure damage to the muscles and nerves, is the unifying factor in all downer cow cases. Cox observed that many downer cows could support weight on their forelimbs if assisted to stand with hip lifters or slings, while their hindlimbs remain non-functional. This observation prompted him to investigate the importance of pressure damage, as it seemed unlikely that a systemic problem would preferentially affect only the hindlimbs. [3,9]

To investigate the effects of pressure damage in down cows, Cox anesthetized 15 non-pregnant and 1 early pregnant Holstein cows with Halothane anesthesia for 3, 6, or 12 hours. The animals were placed in sternal recumbency with the right pelvic limb positioned underneath the body on a 14mm thick rubber commercial cow mat. Eight of these animals stood within 3 hours of the termination of anesthesia. The other eight remained recumbent and either died or were euthanized. Interestingly, there was no relationship or correlation between the duration of anesthesia and the ability to stand. At the end of anesthesia the right pelvic limb was severely swollen and in rigid extension.[11]

CK was highest in the ambulatory group (those that were able to rise post-anesthesia) at 24 hours post-anesthesia and highest in the downer group, 48 hours post-anesthesia. There were no significant differences between the CK values of downer and ambulatory animals at 0, 3, 6, 12 or 24 hours. At days 2 and 4 the downer group had significantly higher CK values, however by day 6 no difference was seen between the two groups. Based on the similar CK values, Cox concluded that the degree of muscle damage between the two groups was

comparable. Thus, the probability of becoming a downer could not be explained solely on duration of recumbency or muscle damage.[11]

The downer group was euthanized and necropsied 10 to 14 days after the initial experiment. Necropsy revealed necrosis of the caudal thigh muscles, with the semitendinosis muscle the most markedly affected. The muscles were pale, necrotic and foul smelling. The semitendinosis muscle had noticeably thicker fascial boundaries then the semimembranosis or biceps muscle, making it more likely to be affected by compartment syndrome. Inflammation of the sciatic nerve caudal to the proximal end of the femur was a consistent finding. The nerve was extremely discolored with extensive proliferation of collagenous tissue around the nerve. Peroneal nerve damage was observed in nine animals. The study concluded that muscle and nerve damage is very important in the pathogenesis of downer cow syndrome and that subtle differences in positioning resulting in nerve damage was a major determinate in whether a cow became a downer.[11]

. In a separate study of anesthetized cows in sternal recumbency, pressure readings in the hamstring muscles were obtained. When the pelvis was tilted about 25 degrees off vertical, maximum hamstring pressures were seen. As the animal was rolled laterally, the pressures dropped to almost zero. Thus, subtle changes in body position can result in considerable pressure differences.[8] The greatest soft tissue damage occurs in areas where soft tissue is compressed between the supporting surface and bone. The peroneal nerve as it crosses the lateral side of the stifle and the sciatic nerve as it crosses the proximal end of the femur are very vulnerable to pressure damage.[9]

Cox's original observation that the forelimbs are spared during sternal recumbency can be explained on the basis of pressure damage. The bovine sternum is well developed to bear

weight of the cranial body during sternal recumbency, thus little weight is placed on the forelimbs. During sternal recumbency, the forelimbs are not located underneath the body and are thus protected from compression damage. In contrast one pelvic limb is always located underneath the body during sternal recumbency.[8] When a cow is in lateral recumbency the forelimbs are no longer protected from compression damage and paralysis of the forelimbs is observed, due to pressure damage to the nerves of the brachial plexus. [10]

Terminal Recumbency

Struggling to rise often results in rupture of muscle and ligaments and terminal recumbency. Common injuries seen include rupture of the gastrocnemius tendon, tearing of adductor muscles and hip luxation. The prognosis for terminal recumbency is grave.[12]

Prevalence

In a study of dairy culling patterns in the northeastern and upper midwestern United States, injury was the most common reason for culling dairy cattle, according to DHIA records.[13] A separate study on downer cow occurrence in Minnesota dairy herds revealed that the incidence of downer cows was 21.4/1000 cow-years at risk. Overall, 33 percent of the downers recovered, 23 percent were slaughtered and 44 percent died. Fifty-eight percent of downer cases occurred within one day of parturition and 95 percent occurred in the first 100 days of lactation.[1] A cumulative postpartum incidence rate for downer cow syndrome in a study of New York State Holstein dairies was 1.1%.[14]

Treatment

Treatments for downer cows should first address the cause of primary recumbency. Steroids, including dexamethasone are frequently used to decrease swelling, inflammation and pain associated with traumatic injuries. Non-steroidal anti-inflammatory drugs (NSAIDs) are

frequently used to keep the animal comfortable and eating.[12] Orogastric tubing with water and electrolytes when the animal is initially down may be helpful to aid in rehydration. Any animal displaying signs of hindlimb weakness should be hobbled to help prevent falling in abduction. Ample food and water should always be available to the downer cow. This can be difficult with creepers who often knock over water buckets placed near them.[3]

At a minimum all down cows should be moved off concrete surfaces to a heavily bedded stall. A dedicated sand bedded stall is ideal, however a bedded pack, grass or loose dirt area is also sufficient.[3] Sand is the best bedding for down cows because it conforms to the cow, spreading the supporting surface over a greater area and creating fewer pressure points. It provides excellent footing and good hygiene, as manure can be easily removed and urine percolates through. The downfall of sand is that it can be abrasive and it is difficult to handle.[15] Any down cow should be maintained in sternal recumbency and rolled from one side to another every couple of hours to allow circulation to return to the down limb.[3]

Devices to raise cattle are many and include hip lifters, slings, inflatable bags and water floatation. Lifting devices aid in diagnosis and prognosis as those animals that are able to support their own weight once up and then remain standing have the best prognosis.[8] These devices do not treat the primary cause of recumbency.[16]

Hip lifters are placed around the tuber coxae and the animal is raised using a winch. Hip lifters can cause severe muscle damage because the entire weight of the animal is born by the soft tissues surrounding the tuber coxae. Hip lifters should always be well padded and attached for the minimum amount of time necessary to assess whether the cow can support her own weight. Although hip lifters are easy to use and useful in helping a cow to rise, they have the potential to cause great harm and should be used with caution.[7]

Lifting air bags are placed underneath a down cow and then rapidly inflated. The abdomen of the cow supports most of the weight of the cow and abdominal contents are pushed cranially against the diaphragm, making respiration difficult.[9] Additionally, the cow will often roll off the bag and maintaining her on the bag requires numerous dedicated people. Bellybands present a similar problem because they push the abdominal contents against the diaphragm and tend to result in an unresponsive cow.[16]

The livestock wheel chair is a metal cart fitted with slings. The theory behind this device is that a cow can walk and bear some of her weight while being supported.[15] This device though helpful, is difficult to use and requires a dedicated area on the farm for the animal to walk around in its wheelchair. A full body sling has been developed, which acts to spread the weight of the cow more evenly across her body, including the sternum and abdomen. The slings are less traumatic then hip lifters, however they are more labor intensive and the cow often struggles. If a cow is to be slung for a period of time, padding is necessary to help decrease pressure sores.[3]

Floatation with water is the most effective method of lifting as water distributes the weight evenly over a large surface, thus creating very few, if any, pressure points. A commercially available floatation tank, "Aqua Cow®" is available, although some farms have constructed their own devices. The tank is rapidly filled with warm water. Rapid filling is necessary so that as the cow attempts to stand, the water level rises quickly enough to support her.[16] The advantage of floatation is that cows can be floated for days in a tank, greatly decreasing pressure damage to the muscles and nerves and allowing the animal time to heal from the original insult. Most cows do not panic or try to get out of the tank. The warm water may be potentially beneficial in increasing circulation. Smith, et al performed a retrospective, non-controlled study of down cows that presented to the Veterinary College at the University of

California at Davis and were floated in an Aqua Cow® tank. Out of the 70 cases that were floated, 32 had successful outcomes (46%), when success was defined as ability to rise, stand and walk unassisted. The greatest success was seen with calving paralysis cases; out of 18 cases, 14 had successful outcomes (78%). The candidates for the float tank were selected, and any animal with a diagnosed fracture, severe spinal cord compression or severe systemic illness was not floated.[6]

Conclusions

Treatment of downer cows is labor intensive, thus diagnosis of the cause of recumbency is important so that labor expenditures can be focused on those animals with a reasonable prognosis of survival. Treatment should first focus on the primary cause of recumbency and then on preventing secondary pressure damage to muscles and nerves. Floatation tanks offer the best means of preventing pressure damage and secondary recumbency.

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