Down to the Wire

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Introduction

Bovine traumatic reticuloperitonitis, also known as hardware disease, is a common disease of dairy cattle. [3,5,10] It sometimes affects beef cattle as well, but it rarely affects other ruminants. Bovine traumatic reticuloperitonitis occurs when a piece of metal is ingested by cattle and settles in the reticulum. [3,5,10] Through reticular contractions, the ingested piece of metal penetrates the wall of the reticulum and can eventually penetrate the diaphragm and pericardial sac. [3,5,10] This leads to inflammation in the cranial abdomen, ventral thorax, and pericardium. Hardware disease used to be much more prevalent but through the development of preventive measures and methods, far fewer cases are seen today. However, when prevention is lacking, it still occurs and requires accurate diagnosis and treatment.

History and Presentation

18 B is a five-year old Holstein cow that presented to Mississippi State University Food Animal Services on October 3rd, 2019, for a six-day history of ADR. She had been lethargic and depressed for six days and off feed for five days. She was given ceftiofur crystalline free acid (excede) and dinoprost tromethamine (lutalyse) the morning of October 2nd, but they did not seem to help her condition. She was also 12 days postpartum from an uncomplicated delivery.

On presentation, 18 B was depressed but alert and responsive. She had an increased respiratory effort and rate of 80 breaths per minute and a heart rate of 60 beats per minute. Her rectal temperature was also increased at 107.1°F. She weighed 511 kgs and had a body condition score of 2/5. She had edema in her brisket region, distended jugular pulses, distended milk veins, and decreased rumen contractions. She was reluctant to move, stood with abducted elbows, and would not ventroflex when her withers were pinched. On auscultation of the

thoracic cavity, there were muffled heart sounds bilaterally and decreased lung sounds/crackles cranioventrally. The remainder of her lung sounds were normal. On rectal palpation, the rumen was small and empty, and the dorsal sac was full of gas. A thoracic ultrasound was performed, revealing evidence of cardiac tamponade, pericardial effusion, and pleural effusion. On abdominal ultrasound, the abomasum was also empty and had thickened walls.

Differential Diagnosis and Diagnostic Approach

Based on the history and clinical presentation, our differential diagnosis list included bovine traumatic reticuloperitonitis, lymphosarcoma, abomasal ulcers, vagal indigestion, intestinal obstruction, and valvular endocarditis. [1,2,3,9,10] With the use of ultrasound, we refined our list of differentials down to bovine traumatic reticuloperitonitis or lymphosarcoma. [3] Next, a pericardiocentesis was performed and we obtained 15 milliliters of yellow tinged, malodorous fluid. [3] We also collected a blood sample and ran a PCV (27%) and TP (7.2 g/dL). Based on the characteristics of the pericardial fluid, we suspected bovine traumatic reticuloperitonitis. [1,3] Both bovine traumatic reticuloperitonitis and lymphosarcoma have a grave prognosis; therefore, 18 B's owner elected humane euthanasia. [1,3]

Another common clinical sign seen in cases of bovine traumatic reticuloperitonitis is tachycardia. [1,2,3,10] In this case, tachycardia was not present; however, thoracic auscultation revealed muffled heart sounds consistent with splashing and gurgling, making it hard to obtain an accurate heart rate.[3,10] The 60 beats per minute was obtained from the pulse of the tail vein. This may not be an accurate representation of 18 B's heart rate as cardiac output may be decreased due to pathology even if heart rate was increased. [1]

Other diagnostics that can be performed in future cases of hardware disease include routine bloodwork, analysis of the pericardial fluid, a coagulation panel, and a glutaraldehyde coagulation test. [1,3,5,9] Routine bloodwork would most likely reveal a neutrophilia with a left shift, and plasma fibrinogen, globulin, and total protein would be markedly increased. [5,9] Bloodwork may also reveal an increased gamma-glutamyl transferase (GGT), aspartate-aminotransferase (AST), and bilirubin due to hepatic congestion. [1,3,5] In chronic cases, some cattle have revealed a prolonged prothrombin, thrombin, and activated partial thromboplastin time due to disruption in hepatobiliary circulation, or liver inflammation and necrosis. [5] Cattle will also have a shortened glutaraldehyde coagulation time due to the increased presence of fibrin and globulins in cases of bovine traumatic reticuloperitonitis. [3,5] If the case is chronic enough to cause ileus, then metabolic alkalosis can occur with hypochloremia and hypokalemia; similar to bloodwork seen in cases of displaced abomasum or vagal indigestion. [5] Radiographs and metal detectors can also be used to determine if a piece of metal is present in or beyond the reticulum. [3,4,5,10] An ultrasound can be used to determine if fluid accumulation is present in the form of abscesses, abdominal effusion, pleural effusion, or pericardial effusion, as seen in the case of 18 B. [3]

Pathophysiology

Bovine traumatic reticuloperitonitis occurs when metal objects penetrate through the reticulum, past the diaphragm, and into the pericardial sac. [3,5,10] These metal objects can include pieces of wire, nails, or other small pieces of metal ingested by cattle. [3,5,10] These pieces of metal are either mixed with their hay during harvesting, accidentally added to their grain ration, or inadvertently mixed with their total mixed ration (TMR). [5,10] The metal pieces usually settle in the reticulum due to the location of the ruminoreticular fold. [5,10] Reticular

contractions cause the metal objects to penetrate through the wall of the reticulum. [5,10] A common time for this to occur is during late pregnancy and parturition due to the compression of the rumen and the reticulum, leading to an increased likelihood of penetration. [5,7,10]

Once the reticular wall is perforated, gastrointestinal contents including ingesta and bacteria leak into the abdominal cavity surrounding the reticulum. [1,5,10] This results in a local peritonitis as well as abscesses and adhesions. [1,5,10] In some cases, diffuse peritonitis can result. These adhesions and abscesses can lead to vagal indigestion, specifically type 2, failure of omasal transport, or type 3, failure of abomasal outflow. [6] Vagal indigestion results in decreased appetite, decreased milk production, scant feces, ileus, a papple shaped abdomen, and bloat, which are all clinical signs consistent with bovine traumatic reticuloperitonitis. [6]

If the metal piece continues to penetrate further, it will eventually pierce the diaphragm and pericardial sac. [1,5,8,10] If the diaphragm is pierced, it can result in pleuritis and pulmonary abscesses. [5,10] The introduction of gastrointestinal contents and bacteria into the pericardial sac causes pericarditis and eventually myocarditis. [5,8,10] Other organs that can be affected by these metal pieces include the spleen and the liver. [1,5] When this occurs, abscesses commonly form to wall off the infection. However, widespread infections can lead to septicemia. [3,5,8,10]

Treatment and Management Options

In acute cases of bovine traumatic reticuloperitonitis, there are a few treatment options available to include surgical intervention and medical management. [4,5] For surgical intervention, a rumenotomy approach is used to remove the metal object(s) from the reticulum. [4,5] At this time, any abscesses that are adhered to the outside of the reticulum are also drained through the reticulum to limit any further peritonitis. [4,5] Antimicrobials are also given

intraoperatively and continued postoperatively. [4,5] For medical management, antimicrobials such as oxytetracycline or procaine penicillin G are used to limit the progression of the peritonitis. [4,5] A magnet is also placed in the reticulum to prevent reoccurrence. [4,5] Serial radiographs can be taken to evaluate the starting point and migration of the metal object(s). [4] If the metal foreign body becomes completely attached to the magnet after administration, surgical intervention is not needed. [4] However, if the metal foreign body is still penetrating the reticulum, 1-3 days post administration of the magnet, surgical intervention is indicated. [4] All affected cattle should be confined for a couple of weeks. [4,5] Stress and movement should be limited to prevent further penetration of the metal object. Oral or IV fluids may be necessary in some cases of severe dehydration, as well as transfaunation of rumen fluid for cattle with prolonged gastrointestinal stasis. [1,5]

Magnets used to be the treatment of choice, but now they are the mainstay of prevention. [2,4] Usually these bar magnets are administered orally at one year of age at the time of pre-breeding vaccinations. These magnets help contain the metal pieces in the reticulum and prevent them from penetrating into the abdomen or thorax. [2,4] Other simple prevention methods include discontinuing the use of bailing wire, keeping cattle away from construction areas, properly maintaining fences and buildings, and passing cattle feed over magnets. [5] Any sensible strategy to prevent the introduction and possible ingestion of metal objects is effective.

Expected Outcome and Prognosis

In uncomplicated cases of bovine traumatic reticuloperitonitis, the chance of short-term survival is fair, while the chance of long-term survival is poor. [3,5] In complicated cases, the prognosis for recovery drops dramatically. [3,5,10] In these cases, the economic value of the cow should be evaluated. If the cow has increased value or is actively pregnant, medical management

and surgical intervention can be attempted until parturition is reached. [3,5] This can include treatment options discussed above as well as repeated pericardiocentesis to drain the excess pericardial fluid, or even pericardiotomy. [1,3,5] If the cow however has a limited value to the owner or poor overall prognosis, humane slaughter should be considered. [3,5]

Case Outcome

After discussion with the owner about the poor prognosis of bovine traumatic reticuloperitonitis; humane euthanasia was elected. Sedation was given prior to euthanasia with 0.2 mg/kg of xylazine intravenously. A captive bolt was used to euthanize 18 B, followed by intravenous potassium chloride (KCl). Necropsy of 18 B revealed multiple lesions consistent with the suspected diagnosis of bovine traumatic reticuloperitonitis.

On necropsy of the abdomen there was yellow tinged fluid consistent with ascites, as well as multiple abscesses found around the reticulum. There were fibrinous attachments to the abdominal wall and on the omentum, ventral to the liver. There was also evidence of local peritonitis around the duodenum and outflow tract of the abomasum seen as a mat of fibrin. There were also a few areas of fibrin seen on the liver.

When the thoracic cavity was opened, there were fibrous attachments from the pleural surface of the lungs to the thoracic wall as well as to the pericardial sac. The lungs were poorly collapsed and on cut surface, the airways were extremely dilated. The pericardial sac was markedly enlarged and thickened. The pericardium was 5-6 mm thick and contained almost a liter of yellow tinged, malodorous fluid. The surface of the epicardium was covered in yellow tinged fibrin and fibrous connective tissue characteristic of the classic “bread and butter” lesion associated with fibrinous pericarditis. [1,3] On cut surface, there was fibrosis of the epicardium

that was approximately 5-6 mm thick. There was evidence of myocardial hypertrophy throughout the heart and the size of the left ventricular lumen was greatly decreased.

On necropsy, a metal detector was used to try to find a piece of metal in the heart. Unfortunately, no metal pieces were detected in the heart; however, there was a small piece of metal found in the reticulum. This is not an unfamiliar finding in cases of bovine traumatic reticuloperitonitis. By the time clinical signs and pathology are this severe, the causal piece of metal may have migrated to another part of the body. [(3)] Originally our hypothesis was that the initial incident occurred 12 days prior during the parturition process; however, based on pathological findings, the introduction of the piece of metal into the pericardium probably occurred earlier than that. Many of the pathological findings were more consistent with a chronic condition rather than acute.

During necropsy there were aerobic cultures taken from the peritonitis in the abdomen as well as from the pericardial sac. Both cultures revealed growth of *Trueperella pyogenes*, further revealing that the causative agent of these infections was from something in the abdomen that penetrated into the thorax and pericardial sac. This is consistent with our diagnosis of bovine traumatic reticuloperitonitis.

Based on the cause of the ADR and eventual death for 18 B being bovine traumatic reticuloperitonitis, we recommended putting a magnet in the reticulum of the remaining cattle in the herd. This prevents metal pieces that are ingested by these cattle from penetrating through their reticulum, diaphragm, and eventually pericardial sac. This magnet will keep all the ingested metal collected in the ventral portion of the reticulum and keep it from migrating through the rest of the gastrointestinal tract. We also recommended that magnets be placed in the mixing wagon to decrease the likelihood of pieces of metal getting into the feed.

Conclusion

In conclusion, bovine traumatic reticuloperitonitis is a disease that is very easy to prevent but difficult to treat. Once the clinical signs are severe and secondary complications are present, the prognosis for recovery is grave. Therefore, it is vital to invest in the upfront costs of preventative measures.

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