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Case Report

A Case of Traumatic Reticulopericarditis in a 6-Year-Old Sanga Cow

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Abstract

The current report describes a typical case of traumatic reticulopericarditis (TRP) in a six (6)-year-old Sanga breed cow that was presented to Ondangwa state veterinary clinic with a history of extended neck, loss of weight, lagging behind and reluctance to stand. On physical examination the cow was observed to have a swollen brisket, abducted forelimbs, an arched back and tachycardia with a characteristic muffled heart sound, a hyperkinetic pulse rate and tachypnea. Based on the history, physical examination and diagnostic tests (such as wither's test, pole's test, slope test and sharp turn test) a tentative diagnosis of traumatic reticulopericarditis was made. This was the basis for a poor diagnosis. Due to the poor prognosis, the cow was recommended for slaughter. Unfortunately, it died before slaughter a day after presentation. An on farm postmortem was conducted which confirmed the diagnosis as TRP due to a sharp wire that perforated the pericardium from the reticulum.

Keywords: Cow; Slaughter; Postmortem; Foreign Body; Traumatic Reticulopericarditis

Introduction

Traumatic reticulopericarditis (TRP) also known as 'hardware disease' is a sporadic disease of ruminants characterized by inflammation of the pericardium [17]. It is caused by perforation of the reticulum and pericardium by ingested foreign body (FB). TRP is usually observed in cattle where it is mostly caused by long, thin sharp foreign bodies such as wires, needles and nails that penetrate through the reticulum, diaphragm and pericardial sac resulting in traumatic pericarditis [21]. If Pericarditis is left untreated it can lead to hematogenous spread of disease such as Salmonellosis, Colibacteriosis, Pasteurellosis and anaerobic infections. Once the disease is spread hematogenously, the cattle begin exhibiting signs of septicaemia which can mask the disease [7]. Interestingly, idiopathic pericarditis which is seen in humans, dogs and horses is rarely seen in cattle [13].

Etiology

Cattle are commonly affected because they do not discriminate when they feed and do not completely masticate feed before they swallow therefore they end up ingesting foreign metallic objects [17].

The disease is common when silage, hay and green chop are made from fields that have old fences that are rusty and falling apart, baling wire or sites of newly constructed, burned down or torn down buildings. Wire is the most common material used for fencing in the northern area of Namibia [8].

Geographical distribution

This disease is distributed worldwide, in areas that use wire as a fencing material, in areas that collects old tires, new building sites and old, burned torn down buildings [12]. The disease is also seen in farms that practice free ranging especially in industrial areas [3].

Pathogenesis

Cattle use their tongues to graze and do not completely masticate because the later regurgitate the food, this allows the animals to feed on non-food objects such as wires or nails [17].

The reticulum is the second stomach of the four stomachs of a ruminant animal. When ruminants swallow feed, it enters the rumen. As the wire enters the rumen it passes through due to the ruminal movements and enters the reticulum were it sinks and settles

Exotic chicken production system is characterized by intensive management with adequate provision of feed resulting to high production of number of eggs and meat than the indigenous chicken breeds but not generally adapted to adverse environmental conditions compared to that of indigenous chicken [7]. Exotic chicken is characterized by high mortality rate due to disease, poor management and coupled with nutrition amongst them [16]. Thus, infectious diseases are one of the major factors constraining the sector.

Newcastle diseases according to [3] is a viral disease of birds which belongs to paramyxovirus type I (APMV-I) serotype of the genus *Avulavirus* and the subfamily Paramyxovirinae and family Paramyxoviridae known for causing Newcastle disease virus (NDV). Newcastle disease (ND) is regarded as a very highly contagious viral infection of birds which remains a serious health issue with significant economic loss due to high mortality and costs of disease control [5]. This disease is recognized as one of the most important limiting factor in chicken farming making it a serious threat to intensively reared chickens [6]. Newcastle disease has been recognized globally as one of the most economically important disease of chicken and other birds due to the fact that it is easily transmitted through direct contact of healthy birds with their droplets and body fluids from infected birds and also through contact with contaminated feed, water and equipment [13]. Newcastle strain can lead to high mortality rate of chicken without showing any clinical signs prior to it. [9] also posited Newcastle disease (ND) as an infectious and one of the most devastating diseases which can lead to huge productivity losses in poultry.

Haematological observation provides valuable information about health of human and animals. According to [2] changes in haematological parameters are often used to determine health status of the body and to know the degree of environmental, nutritional and pathological stresses.

Newcastle disease impact is most notable in domestic poultry due to the fact that this disease is of serious economic threat to the poultry industry resulting in increased morbidity and mortality rates and loss of eggs for both breeding and human consumption (Alexander, 2003).

Exotic birds are routinely vaccinated against prevailing disease and prominent among this disease is the Newcastle Disease which has been regarded and viewed as one of the most serious fatal poultry disease of economic importance [5]. The attack of this disease has led to hindrance to asses' cheap source of protein and provision of income in poultry production. [1] posited that this disease is endemic in nature due to economic loses to farmers coupled with hampering the growth of poultry industries that has been recorded due to their attack. The continuous reoccurrence of Newcastle disease even in vaccinated flock's haematology have been reported.

in the pocket due to the anatomical positioning of the rumen [20]. The reticulum is anatomically lower than the rumen therefore the objects flows easily from the rumen into the reticulum and because the abomasum is slightly anatomically higher than the reticulum, a pocket is formed in the reticulum, where heavy foreign bodies sink and settle. The objects become further trapped when it lodges in the honey comb wall structure of the reticulum [20].

The FB however only perforates the reticulum wall when there is increased contraction in the GIT or pressure as the case is during parturition. Once the FB perforates through reticulum wall it can cause bacteria to move into the peritoneal area causing a traumatic reticuloperitonitis. However the FB sometimes proceeds and perforates the diaphragm and heart, with the wire carrying bacteria from the stomach to the pericardium leading to pericarditis. Pericarditis can later spread the bacteria hematogenously causing septicaemia. Because of the position of the reticulum, the foreign body usually perforates through the left side of the heart [6]. The perforation and inflammation of the pericardial area causes accumulation of fluid and fibrinous accumulate in the pericardial area known as pericardial effusion which affects the function of the heart and leads to the clinical signs observed [6].

Clinical Presentations

The first clinical sign observed in cattle with 'hardware disease' is tachycardia. The tachycardia is brought on by compression of the heart by the pericardial effusion which accumulates in the pericardial sac and prevents normal pumping of the heart, which results in tachycardia as a compensatory mechanism [18]. In some cases, the heart rate is only mildly elevated from 80 to 100 beats per minute (bpm). However, typically the heart rate is severely increased with rates as high as 130bpm. The degree of tachycardia depends on the amount of pericardial effusion [18]. The other clinical sign observed in cattle with 'hardware disease' is muffled heart sounds, caused by pericardial effusion and fibrinous changes in the pericardial sac [18]. Asynchronous abnormal heart beat can also be heard together with the muffled heart sounds. With predominance of fluid there are splashing or gurgling sounds, which vary continuously in pitch, loudness, duration and point of maximum intensity [9]. The jugular vein becomes extended with the degree of distension varying depending on the degree of cardiac tamponade [5]. Other signs that can be observed include edema of the submandibular region, brisket and ventral abdomen. The animals tend to stand with abducted elbows in order to try and aid cardiac function or as a result of pain. Other indicators of pain that maybe observed in the animal may include bruxism and grunting. When there is septicaemia the animal can have a fever of 40°C which results in an abnormal demeanor and loss of appetite [11]. However, the fever can be absent and in rare cases temperature can even be below normal. In cases where septicaemia is absent, this does not rule out

TRP. Due to the cardiac insufficiency or direct involvement of the lungs, the respiratory rate is also usually elevated and shallow [15].

Pathological signs

In the TRP case sometimes there are extensive fibrinous adhesions between the cranioventral aspects of the reticulum, the ventral abdominal wall, and the adhesions and multiple abscess may be observed on either side of the reticulum [10]. Large quantities of turbid, foul-smelling peritoneal fluid that contains fibrinous clots may be present [10].

Diagnosis

After a thorough history is taken and a good physical examination has been done, a series of confirmatory diagnostic tests can be done to confirm a suspected 'hardware disease' case, such as

Ultrasonography

This is the best method for diagnosis of suspected TRP case because it characterizes pericardial effusion. It is performed on a standing cow using 5.0 MHz sector or convex transducer from the third to fifth intercostal space on both sides of the thorax [6]. The ultrasonography of a cow with TRP appears to have a large amount of hypoechogenic fluid seen [1] in the thorax, sometimes containing strands or free clots of fibrin may be seen floating around in the fluid between the pericardium and epicardium. The lungs appear compressed and displaced medially and dorsally by the present pleural effusion. The heart may be obscured at times by the effusion. In severe cases, ultrasound of the abdomen shows signs of changes in the reticulum typically in traumatic reticuloperitonitis [5].

Haematological and biochemical test

A thin blood smear is the easiest and quickest method of examination, however, not accurate. In an animal with TRP, the blood smear would show signs of neutrophilia with a left shift, which could also be observed in any chronic bacterial infection because of the present septicaenia. A glutaraldehyde test is one of the tests that can be done. However, it is a non-specific indicator of inflammation that indicates elevation of fibrinogen and globulin, which decreases the clotting time of the blood in more than 90% of affected cattle [5]. This test can be used to differentiate between inflammatory and non-inflammatory right side cardiac insufficiency. In a haematological test, the other things that can be observed in cattle with TRP are Leukocytosis and hyperfibrinogenaemia, which all indicate inflammation. There may also be a detection of activities of Y-glutamyltransferase, aspartate aminotransferase and increased bilirubin, which would all indicate hepatic congestion that usually occur in cattle with right cardiac failure [2]. In a recent study, it was demonstrated that cardiac troponin proteins are usually presents in amounts above the normal concentration in cattle with pericarditis. Cardiac troponin proteins are usually present in the blood either at a very low concentration or below the limit of detection of most tests [14].

Other tests

Other tests that can be done in a live animal include the wither's test and the pole test. These tests can be together with other tests mentioned above.

A pole test is done by placing a pole at the thoracic area of the animal and slightly lifting the pole, the test is positive when the animal pushes against the pole in response to the pain being exerted by the wire as it moves up with the pole. The wither's test is done by gently exerting pressure on the withers of the cattle in which normally the animal moves down away from the hand. If the animal fails to move down it is positive for TRP [4].

Radiology

A laterolateral radiographic view is taken of the caudoventral thorax and reticulum while the cow is in a standing position. A positive TRP radiography shows a FB perforating the cranial reticular wall and diaphragm. In most affected cattle, the cardiophrenic angle is obscured and the cardiac silhouette and ventral diaphragm completely obliterated from view because of extensive fibrinopurulent lesions. A gas-fluid interface is commonly seen in the caudoventral thorax which is a characteristic of abscess formation, indicating bacterial infection. The metallic FB which is usually a piece of wire or nail usually appears radio-dense [7].

Examination of the pericardial fluid

The pericardial fluid can be collected by aspiration and bacteriological examinations and inflammatory factors test can be done. The fluid is however collected carefully to prevent spreading of the disease [15].

Differential diagnoses

Clinical signs such as distended jugular vein and tachycardia, right-sided heart failure attributable to valvular endocarditis, cardiac leucosis, cardiomyopathy or other causes may be considered [9]. Distension of the jugular vein without any sign of right side cardiac insufficiency may be due to compression or obstruction of the cranial vena cava by a thrombus or a thoracic mass. Pleural effusion may be a result of many causes that are not heart disease [15].

Treatment

There are a few therapeutic options available to treat TRP. First one being broad spectrum antibiotics such as Amoxicillin 10mg/kg IM/SC or Tetracycline7mg/kg IM/SC. This treatment is done for duration of 1 to 2 weeks depending on the severity of the infection, which can be determined by clinical signs observed [5]. Treating

with antibiotic however only helps treat septicaemia but the wire remains lodged in the tissues, therefore certain clinical signs remain such as those caused by right heart failure [15].

The second and advised method of treatment is a rumenotomy. This is a routine surgical procedure where the rumen is surgically incised via the left abdominal wall. Rumenotomy is performed for a number of reasons, but in this case it would be performed in order to retrieve the wire that has perforated through the reticulum, diaphragm and pericardium [3].

Postoperative care

The animal is given a course of antibiotics for 5-7 days, anti-inflammatory drugs for 2-3 days, dressing alternate days for 7-10 days, fluid therapy is required [16]. The animals should be given mild osmotic laxative, to assist in prompting gut motility, a light diet for a few days post-surgery. Removal of sutures should be done on the 8th to 10th day post operation. A percardiocentesis is usually done as well to reduce the amount of fluid in the pericardial cavity to improve cardiac function [3].

Prognosis

The prognosis in these cases is usually poor in cattle with TRP because the pericardial effusion is usually fibrinopurulent.

Clinical Case Report

A 6 year old black and white intact Sanga cow was presented to Ondangwa state veterinary clinic on the 17th November, 2017 with a history of lagging behind the rest of the herd, reluctant to stand, had an extended neck with lack of appetite and was losing weight. It was also revealed that the cow had calved five months prior to presentation but unfortunately the calf died. The calving was characterized by excessive straining and the cow had become recumbent for a few days post parturition. The clinical signs were initially observed immediately post parturition, however the animal then appeared normal but manifested the similar clinical signs a week prior to presentation. A herd size of 27 cattle was managed under a traditional extensive method without any supplementations. Physical examination findings revealed the following results (Table 1).

However the abnormal findings observed were tachypnea, brisket edema, abducted forelimbs, arched back and ectoparasites (Boophilus species). On cardiac auscultation, muffled heart sounds with a concurrent tachycardia were detected. Left flank abdominal auscultation revealed ruminal stasis. On rectal palpation, pasty scanty feces were observed with almost empty intestines.

Parameters	Observed at Examination	Normal Ranges
Temperature	38. 7°C	38.0°C - 39.0°C
Heart Rate	118 beats/minute (Muffled)	60 - 80 beats/ minute
Pulse Rate	114 pulses/minute (Weak and thready)	60 - 80 pulses/ minute
Respiration Rate	45 breaths/minute (Rapid shallow breathing)	15 - 30 breath/ minute
Capillary Refill Time	<2 seconds	<2 seconds
Mucous Membranes	Pink and moist	Pink and moist
Lymph Nodes	Non-reactive	Non-reactive
Ecto-Parasites	Ticks	No parasites
Rectal Examination	Pasty scanty faeces	Soft and even consistency
Ruminal Movement	Ruminal stasis	1-3 times per minute
Others	Brisket edema, abducted forelimbs and arched back	

Table 1: Shows the results of the physical examination findings.

A problem list of reluctance to stand, lagging behind, extended neck, arched back, weight loss, abducted forelimbs, tachycardia, tachypnea, weak and thread pulse, rapid shallow breathing ruminal stasis, pasty scanty feces, brisket edema and ectoparasites (Boophilus spp) was generated. The tentative diagnosis was traumatic reticulopericarditis (hardware disease). The differential diagnoses were traumatic reticuloperitonitis and tick bone diseases (TBDs) such as East coast fever or anaplasmosis.

To confirm the diagnosis, a withers test was conducted that revealed a negative dorsi-reflex. The pole's test was also carried out at the level of the xiphosternum in which pain was elicited with the cow arching it back. When auscultating over the trachea while carrying out the withers or pole test, a grunt sound was heard. The slope test showed resistant of movement down the slope by the cow and sharp turn test also reviewed resistance to turn by the cow.

A definitive diagnosis of TRP was reached. Due to the severe clinical signs and cows poor health, the prognosis was considered grave. Therefore, slaughter was recommended and no treatment plan was instituted. Unfortunately, while awaiting slaughter the cow died a day after being examined.

On farm standard necropsy was conducted first by wetting the carcass with water taking note not to create any lesions. The cow was then placed in left lateral recumbency and post mortem was conducted with the use of a sharp postmortem knife, making an incision on the ventral midline starting from between the jaws to the perineal region, avoiding the mammary glands. The skin was reflected to check for lesions on muscle and fascia. The right leg was then detached from the body and an incision was made on the medial side of the leg, connecting it to the midline incision. The skin was then reflected and the muscles of the leg were reflected and the hip joint was disarticulated.

The abdominal cavity was then opened by incising abdominal muscles, of which the incision extended from the sternum to the pelvis. Upon opening of the cavity it was revealed that there were adhesions of the reticulum, spleen and liver to the diaphragm and abdominal cavity. The thoracic cavity systematically opened by cutting through the ribs at the junction with sternum, costo-chondral and vertebra.

Necropsy revealed the following

- Enviromental examination of the grazing area and the cattle kraal revealed lose wires in the environment.
- An external examination demonstrated froth from the nostrils, rigor mortis as well as abdominal distension (Figure 2).
- The heart and lungs had formed adhesions to the thoracic wall and the lungs had adhered to the diaphragm. The lungs revealed an increased interlobular space figure 3 (B-C)
- When the trachea was cut open, there was evidence of froth.
- A wire was found perforating through the reticulum, the diaphragm and pericardium after the anterior surface of the reticulum was examined figure 3 (D).
- The pericardium revealed fibrinous adhesions attaching the pericardium to the cardiac muscles with tan-coloured fluid in the pericardial sac figure 3 (E).

Figure 1: Showing the physical examination of the cow.

Figure 2: Showing the presence of froth from the nostrils at necropsy.

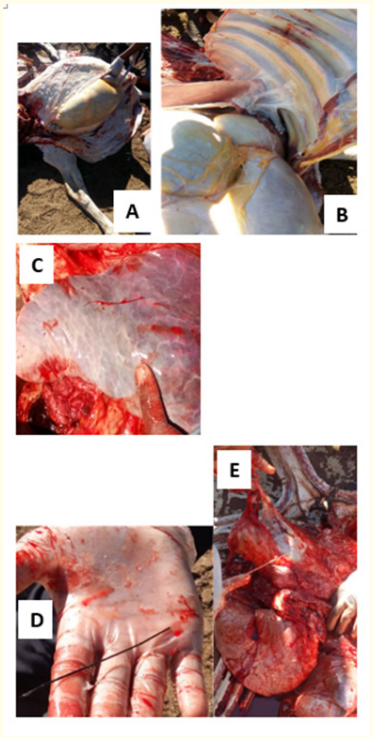

Figure 3: Showing an Incision into the abdominal cavity (A); adhesions of the lungs to the thoracic cavity (B); increased interlobular space (C); wire that was found perforating through the reticulum into the diaphragm and pericardium (D) and pericardium showing fibrinous adhesions attaching the pericardium to the cardiac muscles with tan-coloured fluid in the pericardial sac during necropsy.

Necropsy findings illustrated above confirmed the diagnosis of TRP.

The client was then advised to remove all hardware materials from the environment and consider placement of magnets in the rumen of all cattle while exercising caution when handling all hardware materials. Dipping and deworming of the herd was advised considering that the deworming status was unknown and there was presence of ectoparasites (Boophilus spp).

Case Discussion

Pericarditis is an inflammatory reaction of the pericardium that occurs with accumulated serous or fibrinous exudate between the visceral and parietal pericardium which is usually associated with prolonged progressive disturbances in the heart function and almost always result in death [5].

Although physical examination and observation of clinical signs in a live animal are not enough to make a definitive diagnosis, the clinical signs of TRP "hardware disease" have been well established and are of high diagnostic value. The clinical findings in this case were consistent with what is stated elsewhere as the lead clinical signs of pericarditis which include tachycardia, muffled heart sounds, asynchronous abnormal heart sounds, distention of the jugular veins and submandibular, brisket and ventral abdominal edema [5].

Diagnosis of this condition can be established with a fair amount of accuracy by diagnostic ultrasonography and radiography wherever facilities are available [5]. However, in the present case study, the definitive diagnosis was made based on the history and clinical examination findings which were supported by necropsy findings to confirm the diagnosis of TRP. Therefore, this case report finding suggests that a diagnosis of TRP can be made based on the stated lead clinical signs of pericarditis. While this was a classic case of TRP without peritonitis, perforation of the wall of the reticulum allows leakage of ingesta and bacteria, which contaminates the peritoneal cavity resulting in peritonitis, which is generally localized and frequently results in adhesions [16]. Less commonly, a more severe diffuse peritonitis may also develop. The FB can penetrate the diaphragm and enter the thoracic cavity causing pleuritis and sometimes pulmonary abscessation and the pericardial sac causing pericarditis, sometimes followed by myocarditis [12]. There was no evidence of pleuritis, pulmonary abscessation and myocarditis in this case. Occasionally, the liver or spleen may be pierced and become infected, resulting in abscessation, or septicaemia can develop [12]. In the present case, septicaemia and cardiac failure were suspected to have been the cause death.

An animal can live with the wire in the reticulum without it perforating for some time. However perforation is usually due to late pregnancy, parturition, bloat and any condition that causes increased ruminal movements as well as cranial displacement of abdominal organs [3]. In this case pregnancy was the suspected cause, as the growing fetus may have caused forward displacement of abdominal organs including rumen and reticulum. This may have caused the wire to perforate into the reticulum wall due to reduced occupation space of the FB. At the time of calving, forceful abdominal movements have been implicated as the initial causes of perforation or movement of FB into the heart [3]. The history in the present case revealed excessive straining during parturition and recumbency for a few days following parturition about 5 months earlier although the calf died. The pregnancy could have pushed the wire to perforate the reticulum and the excessive straining and recumbency would have further pushed it through the diaphragm and pericardium leading to TRP. According to [3], cattle of about 10 years old are more often affected with the condition than young ones of about 2 years and below and [22]. states that 97 percent of cases occur in cattle of about 2 years or more. Therefore the diagnosis of TRP in a 6-year-old middle-aged cow reflects that she was within age range of susceptibility. TRP and peritonitis sometimes may cause death in more than one animal in the herd when wires are found around the areas where they feed [8]. In the present report, from the herd of 27 cattle only the one cow that was presented exhibited the clinical signs and died from TRP. However, it is possible that other animals in the herd could have ingested FB thus predisposing them to TRP. As a best preventive measure, the farmer was advised to consider placing a magnet in the rumen of all the animals in the herd.

Conclusion

Prognosis is normally poor, and pericardiocentensis or pericardiotomy are inadequate methods of treatment. Thus, prompt and humane euthanasia is indicated for cattle with TRP [5]. The poor prognosis of the condition is reflected in the present case in which the cow died a day after it was recommended for euthanasia.

A case of TRP in a 6-year-old Sanga cow that was diagnosed based on history, clinical examination findings and necropsy is reported.

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