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Cardiocongestive insufficiency due to vena cava thrombosis with secondary necro-exudative bronchopneumonia

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Abstract. The aim of this study was to report a case of vena cava thrombosis with secondary bronchopneumonia in a two-year-old Holsten breed cow from the municipality of Jacutinga, Rio Grande do Sul State, Brazil. Vena cava thrombosis is a disease affecting adult cattle fed on concentrate-based diets. It is characterized by the presence of septic emboli in the bloodstream, which is formed from caudal vena cava thrombi that obliterate the vascular lumen, causing tissue hypoxia. The most common vena cava thrombosis etiology is ruminal acidosis, generating ruminitis and subsequent liver abscessation and pulmonary artery thromboembolism, abomasum ulcers, and polioencephalomalacia. The animal was in the final third of its gestation and it was fed with corn-silage-based feed and a 20% protein content balanced feed when it presented mastitis, walking pain, and coughing. Treatment for mastitis was carried out and 15 days after that, intramuscular enrofloxacin-based antibiotic therapy was performed. The animal died and necropsy and histopathological data indicated to be compatible with cardiocentesis insufficiency, possibly due to vena cava thrombosis with secondary necro-exudative bronchopneumonia.

Keywords: Acidosis, pneumonia, ruminant

Insuficiência cardiocongestiva por trombose na veia cava com broncopneumonia necroexudativa secundária

Resumo. O objetivo deste estudo foi relatar um caso de trombose da veia cava com broncopneumonia secundária em uma vaca holandesa de dois anos de idade oriunda do município de Jacutinga, Rio Grande do Sul. A trombose da veia cava é uma enfermidade que acomete bovinos adultos que são alimentados com dietas a base de concentrados, caracteriza-se pela presença de êmbolos sépticos na corrente sanguínea, os quais são formados a partir de trombos da veia cava caudal que obliteram o lúmen vascular, causando hipóxia tecidual. A etiologia mais comum de trombose da veia cava é a acidose ruminal, gerando ruminite e subsequente abscedação do fígado e tromboembolismo da artéria pulmonar, úlceras de abomaso e polioencefalomalácia. O animal estava no terço final da gestação quando apresentou mastite, dor ao caminhar e tosse, recebia alimentação a base de silagem de milho e ração balanceada com 20 % de proteína. Foi realizado o tratamento para mastite e após 15 dias ao primeiro tratamento foi realizada antibioticoterapia a base de enrofloxacina, intramuscular. O animal veio ao óbito e dados de necropsia e histopatológicos indicaram ser compatíveis com insuficiência cardiocongestiva possivelmente por trombose na veia cava com broncopneumonia necroexudativa secundária.

Palavras-chave: Acidose, pneumonia, ruminante

Introduction

Vena cava thrombosis usually affects adult bovines that are fed concentrate-based diets, being associated with abscesses as a result of septic embolism of the pulmonary artery vascular system, originating from septic thrombi in the caudal vena cava (CVC) that obliterate the vascular lumen, causing tissue hypoxia (Braun et al., 2002). The most common vena cava thrombosis etiology is ruminal acidosis, generating ruminitis, and subsequent liver abscesses, which may result in thrombus in the CVC if the vessel wall is infiltrated by the abscess (Smith, 2006) and pulmonary artery thromboembolism, abomasum ulcers, and polioencephalomalacia (Plaizier et al., 2008).

Metabolic acidosis causes alterations in the ruminal mucosa, characterized by hyperkeratosis and inflammation due to local lactic acid accumulation, allowing bacteria translocation from the gastrointestinal tract into the bloodstream, and the formation of hepatic abscesses (Pillai et al., 2021; Quevedo et al., 2015). For Constable et al. (2016) and Smith (2006), the bacteria involved in ruminitis are *Fusobacterium necrophorum*, *Arcanobacterium pyogenes*, *Staphylococci*, *Streptococcus*, *Escherichia coli*, which reach the liver forming abscesses that rupture, causing secondary pneumonia (Motta et al., 2016).

The symptoms of the disease include tachycardia, expiratory dyspnoea with groans, hyperpnea, coughing, puffing, and pale mucous membranes, generalized wheezing and hemoptysis, yet to Radostits et al. (2010), fever, chest pain on palpation of the externum and intercostal spaces, hepatomegaly, nostrils froth, and melena caused by expectorated blood swallowing depression, ruminal stasis, and poor milk production (Simpson et al., 2012). In chronic cases, color pulmonale may occur with right ventricular failure signs, such as jugular distention or chest edema. The objective of this study is reporting the case of a Holsten breed cow who died due to suspected vena cava thrombosis and secondary bronchopneumonia.

Case description

A two-year-old Holsten breed cow (Figure 1) in the final third of its gestation, fed with corn-silage-based feed and a 20% protein content balanced feed presented mastitis, anorexia, walking pain, and coughing. One month before the expected delivery date the cow was medicated for mammary gland inflammation, using intramammary tubes and ceftiofur-based injectable antibiotics; 15 days after the first treatment, the cow was medicated again with intravenous enrofloxacin-based injectable antibiotic as it did not show any clinical symptoms improvement.

Delivery occurred on schedule, after that the cow presented retained placenta and an estradiol cypionate treatment, at the manufacturer's recommended dose, was indicated. From that moment, the cow started on a selective eating pattern in smaller quantities, presenting dry coughing that intensified after feed ingestion. The animal presented dehydration, anorexia, the respiratory movements increased, and was quite breathless, with subsequent death.

After death, heart, spleen, lung, liver, breast tissue, intestine, skeletal muscle, kidney, tongue, bone mass, brain, and lymph nodes fragments were collected for necropsy. These materials were stored in flasks containing 10% buffered formalin and sent to the Laboratory of Animal Pathology - LAPA, University of Santa Catarina State (UDESC-Lages Campus). The bacteriological culture was not carried out.

Figure 1. Two-year-old Holsten breed cow with anorexia, dehydration, and prostration.

During necropsy, the lung had alveolar abscesses and emphysema (Figure 2A), enlarged liver (hepatomegaly) with a hemorrhagic abscess (Figure 2B), some rumen areas did not present papillae.

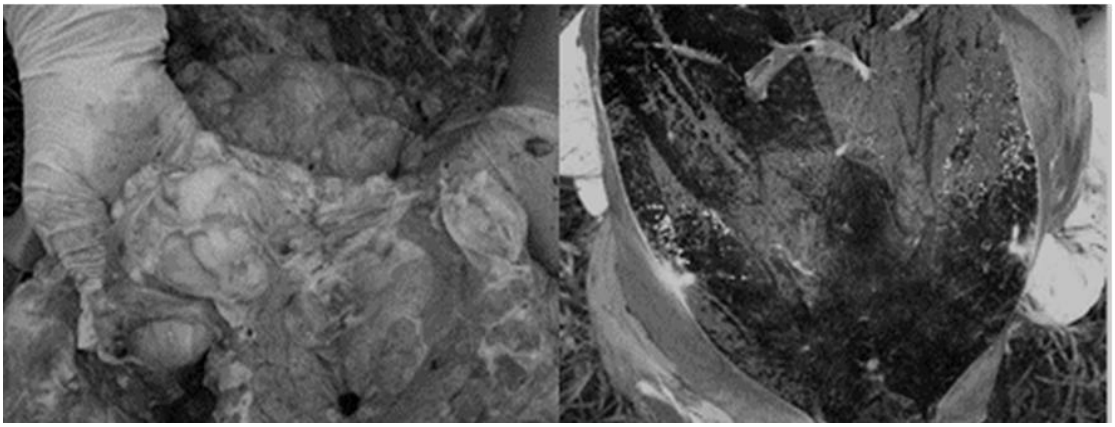

Figure 2. Lung with alveolar abscess **(A)**; and hemorrhagic and enlarged liver (hepatomegaly) **(B)**.

In the histopathological examination, the lung presented multiple granulomas characterized by central necrotic region and cellular debris, surrounded by mononuclear cells, giant cells, and fibrous capsule. In some necroses areas, congestive liver and accentuated intermediate zone lobular center that were diffused with hepatocytes vacuolar degeneration areas associated with a mild perilobular multi-focal inflammatory infiltrate, kidneys with multifocal areas of mononuclear cell infiltration in the cortex, spleen with mild multi-focal hemosiderosis, tissue mass with massive necrosis presenting cellular debris and polymorphonuclear infiltrate, and multiple radiated eosinophilic formations (rosettes), were observed. Necropsy and histopathological data were compatible with cardiocongestive insufficiency possibly due to vena cava thrombosis with secondary necro-exudative bronchopneumonia.

Discussion

Pulmonary thromboembolism as a result of posterior vena cava thrombosis has been discussed since 1975 in Europe and since 1976 in the USA. From 1935 to 1960, at the Royal Veterinary College in Sweden, the necropsy on 1,279 adult cattle presented a 4.35% pulmonary thromboembolism incidence. In the USA, the necropsy of 1,998 yearling feedlot cattle showed a 1.3% incidence, where 40% occurred during the first 45 days of the feedlot period and 28% occurred during the next 45 days (Gudmundson et al., 1978).

The present report is in accordance with Motta et al. (2016), who found hepatomegaly abscesses, of the "nutmeg" appearance verified in 100, 80, and 13% of the cases, respectively, from the liver necropsy of the cattle. For Braun (2008), caudal vena cava thrombosis may also cause hepatomegaly and marked ascites, but most of these animals may also have acute respiratory signs. Thrombus emboli may spread to other organs resulting in endocarditis, embolic pneumonia, hepatic and renal abscesses, or pulmonary vessels erosion causing intrapulmonary or intrabronchial hemorrhage that may end in sudden death (Miller & Gal, 2017).

Motta et al. (2016), in your study analyzed, were observed in 85% of the intercurrent diseases, such as metritis, mastitis, locomotor problem, and hemoparasitoses. The rumen wall did not present papilla in some areas, the heart had its anatomical aspects preserved, and there was mammary gland inflammation. Corroborating with that work, Schild et al. (2017), found in the necropsy of a Holsten breed cow that the lung presented suppurative pneumonia with the presence of embolic thrombi and intra-alveolar colonies of Gram-negative bacilli and coccobacilli, and severe interstitial fibroplasia with a mixed inflammatory infiltrate. Edema, emphysema, and hemorrhages focal areas were observed in the alveoli. The same necrosuppurative exudate was found in the bronchi. In the adventitia of the pulmonary arteries, the elastic fibers were degenerated and dissociated (Braun, 2008). On a necropsy of a 6-year-old Jersey breed cow by sudden death, Gerspach et al. (2011) found that the right atrium was thicker than normal and the pulmonary parenchyma was yellow. The culture of these lung lesions showed *Streptococcus* spp. The hepatic bile ducts had massive numbers of small liver flukes. Based on the histological findings, the cow was diagnosed with cranial vena cava thrombosis, severe multifocal

necrotizing pneumonia with multiple pulmonary thrombi, ischemic renal infarction, *Dicrocoelium dentriticum* infestation, and peritonitis in the reticulum region. In our work, several of these lesions were also observed during necropsy, characterizing caudal vena cava thrombosis as being.

Gudmundson et al. (1978) described a 2.26% hepatic abscess incidence in 7,545 bovine cattle necropsied in a diagnostic laboratory, and thrombosis of the posterior vena cava was present in 19.3% of hepatic abscessation cases. It is known that the critical periods for acidosis development in dairy cattle are the periparturient and mid-lactation periods when the animals receive a concentrate-rich diet in order to meet the lactation demands (Enemark, 2008; Stauder et al., 2020).

Hepatic abscesses and rumenitis are lesions frequently associated with ruminal acidosis (Nagaraja & Lechtenberg, 2007). A Gram-negative anaerobe bacterium *Fusobacterium necrophorum*, is the primary etiologic agent of liver abscesses of beef cattle. The bacterium, a member of the microbial community of the rumen, travels to the liver via portal circulation to cause abscesses. The severity of liver abscesses varies from mild with one or two small abscesses to severe with medium to large multiple abscesses (Pillai et al., 2021). Abscesses on the hepatic surface may extend causing phlebitis in the caudal vena cava, whose inflammation leads to bacterial thrombi and emboli formation and, depending on the number of thrombi and microorganisms involved, a series of changes might occur that collectively form the so-called caudal vena cava syndrome. The outcome might be fatal and caudal vena cava rupture, endocarditis, pulmonary embolism, pneumonia, hemoptysis, and epistaxis may occur (Miller & Gal, 2017; Nagaraja & Lechtenberg, 2007; Radostits et al., 2010), some of these symptoms were found in this case description.

Since caudal vena cava thrombosis in bovine cattle is a rarely described severe and fatal clinical syndrome, caused by nutritional failures attributed to a high-grain diet, yet bulky deficient. Unusual cases of this syndrome in cows, secondary to infection by *Trueperella (Arcanobacterium) pyogenes* (Motta et al., 2016) and *Fusobacterium necrophorum* (Simpson et al., 2012) were reported, which presented a morbid and hyperacute evolution. Caudal vena cava thrombosis.

Ruminal acidosis detection or other causes that may lead to vena cava thrombosis must be highlighted, including a sound clinical evaluation for symptoms such as diarrhea, anorexia, depression, dehydration, in conjunction with an assessment of the conditions in which they may occur, as well as food intake and milk production. The presence of hepatic abscesses, rumen wall areas without papillae, and exposure to highly fermentable foods, such as milled corn grains, allowed the assumption that the animal had ruminal acidosis (Constable et al., 2016; Plaizier et al., 2008).

For Woolums (2015) and Divers & Peek (2007), the treatment of thrombosis of the caudal vena cava is generally not successful. A better approach is prevention by reducing of the incidence of rumen lactic acidosis in high-producing dairy cows, including periodic control of feeding practices, such as quantity, type, composition and structure of grains and fibers, frequency of feeding and degree of adaptation. In addition, a gradual adaptation of the rumen to diets should be made, for cows at the beginning of lactation, in transition, in pre-calves and heifers. Buffers like sodium bicarbonate and magnesium oxide and antibiotics like monensin or lasalocide also gave good results (Constable et al., 2016; Divers & Peek, 2007; Enemark, 2008; Garry & McConnel, 2015).

Conclusion

There has not been any report of bovine vena cava thrombosis in the Rio Grande do Sul State-Brazil, urging the need for further studies regarding this disease, as it causes substantial economic output losses for the dairy belt, and it is suggested that this disease be included in a differential diagnosis from other respiratory and circulatory diseases.

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