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CASE REPORT
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Caudal Vena Cava Thrombosis in Cows

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ABSTRACT

***Background*:** Caudal vena cava thrombosis (CVCT) is a serious disease that affects cattle. Due to being commonly a fatal pathology, it causes economic losses for producers and national livestock. Thus, the present study describes the epidemiological, clinical, ultrasonographic imaging and pathological findings in 4 cattle with CVCT attended at the Veterinary Hospital (HV) of the Institute of Veterinary Medicine (IMV) of the Federal University of Pará (UFPA).
***Cases*:** The animals were crossbreds of the Gir x Holstein and Jersey x Holstein breeds, aged between 4 and 8 years old, raised in semi-extensive and intensive systems. The main clinical signs were pale mucous membranes, reluctance to move, markedly positive venous pulse, engorged jugular with positive stasis test, and serous to mucopurulent nasal exudation. The auscultation of the lung fields revealed tachypnea, silent areas, wheezing, and pleural friction, in addition to coughing, expiratory dyspnea, mouth breathing, and expiratory grunts. One animal had severe hemoptysis. The ultrasound examination performed on a bovine revealed a circular and dilated caudal vena cava in cross-section. Laboratory tests in 3 cattle revealed anemia, leukocytosis with neutrophil left shift, and increased liver enzymes. At necropsy, all cattle had thrombi in the hepatic segment of the caudal vena cava. In the lung, multiple abscesses and areas of parenchymal consolidation, crateriform areas, as well as thrombi in the arteries were observed. Pleural effusion and ascites were seen in all cattle. Clotted blood was seen in the trachea, bronchi, and on rumen contents of an animal. Histopathological alterations seen in the liver were centrilobular hepatocytes with frequent intensely eosinophilic cytoplasm, and pyknotic, karyorrhexic, or absent nuclei and cell borders barely distinguishable. In the lung were nodular and random formations, with a thick wall of mature connective tissue and a central area full of cellular debris, necrotic cells, and intact and degenerated neutrophils (abscesses).
***Discussion*:** The set of diagnostic tools that include epidemiology, clinical signs and clinical examinations, ultrasound, necropsy, and histopathology were efficient in the diagnosis of CVCT. The possible causes that led the animals to develop CVCT were diffuse septic pododermatitis in the medial nail of the right pelvic limb associated with traumatic reticuloabomasitis and liver abscesses. In 1 cow, it was not possible to establish the probable cause of CVCT, but for the other cattle in the present study, the probable causes are in agreement with studies that have shown that this disease can occur as a sequel to several septic conditions such as jugular phlebitis, mastitis, hoof rot, enteritis, pneumonia, traumatic reticulopericarditis, acidosis and rumen laminitis, as well as omphalophlebitis in calves. The tachypnea, serous to purulent nasal exudation, pulmonary wheezing, pleural friction, coughing, and expiratory dyspnea, usually with open mouth breathing and expiratory grunts evidenced in the animals of this study, occurred as a result of embolic abscess pneumonia. The presence of multiple lung abscesses, areas of parenchymal consolidation, crateriform foci, and thrombi in the pulmonary arteries and chronic suppurative pneumonia, found at necropsy of the animals in the present study, are related to the development of a thrombus in the caudal vena cava that detaches and embolizes and lodges in the pulmonary arteries. The histopathological findings in 1 cow are compatible with lesions found at necropsy and draw attention to embolic pneumonia and liver lesions, which, are related to thrombi in pulmonary arteries and abscesses formed from CVCT, as well as venous stasis exerted in the return circulation.

Keywords: thrombi, venous stasis, cardiorespiratory system, Amazon biome.

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INTRODUCTION

Caudal vena cava thrombosis (CVCT) is a serious disease that affects cattle, and due to being commonly a fatal pathology, it causes economic losses for producers and national livestock. The disease is the outcome of several septic conditions and thus can be associated with jugular phlebitis, mastitis, foot rot, enteritis, pneumonia, traumatic reticulopericarditis, laminitis, and ruminal acidosis [21].

Clinical signs include abnormal lung sounds such as wheezing, pleural friction, tachypnea, and coughing. In advanced cases, there is expiratory dyspnea, usually with open-mouth breathing and expiratory grunts. Serous to purulent nasal exudation, increased respiratory rate, and dyspnea may also be present [2].

Regarding the diagnosis, the clinical suspicion of CVCT can be confirmed by ultrasound imaging test in the 11th or 12th intercostal space on the right side, close to the liver. Normally, the caudal vena cava appears triangular in cross-section, and with thrombosis, the vein is oval to circular and dilated [3,7]. However, the most effective method for diagnosing the disease is a necropsy. Important findings of the disease are liver abscesses, located immediately adjacent to the caudal vena cava, and occasionally found in other locations such as the kidney or chest [6].

Although CVCT has already been reported in different Brazilian states [1,9,13,20] no reports were found in cattle raised in the Amazon biome. Thus, the present study describes the epidemiological, clinical, ultrasonographic imaging and pathological findings in 4 cows with CVCT attended at the Veterinary Hospital (HV) of the Institute of Veterinary Medicine (IMV) of the Federal University of Pará (UFPA).

CASES

The study comprised observations carried out in 4 cows (Cows 1 to 4). Epidemiological data were obtained during clinical visits to properties. Animals with clinical signs compatible with CVCT were submitted to general and specific clinical examinations of the cardiorespiratory system [12]. Cows 1, 2, and 4 were from the municipality of Castanhal and kept in a semi-extensive farming system. Cow 3 was from the municipality of Paragominas and was kept in an intensive system. All of them were bred in the State of Pará, Brazil, and were females aged between 4 and 8 years. Cows 1, 2 and 3 were Gir x Holstein crossbreds and the Cow 4 was Jersey x Holstein crossbred.

Cow 1 had been treated for a diffuse septic pododermatitis 30 days before, Cow 2 was raised in a pasture of *Panicum maximum* cultivar Mombasa, Cow 3 was raised in the "Compost Barn" system with a diet rich in fast-digesting carbohydrates and Cow 4 had been prepared for a dairy tournament and received a diet rich in fast-digesting carbohydrates.

The animals had a body score between 2.5 and 3 (scale from 1 to 5), and the clinical findings were apathy, inappetence, dehydration, pale mucous membranes, and reluctance to move. Beyond these signs, Cows 2 and 4 also presented hyperthermia.

Cows 1 and 4 had a visibly positive venous pulse and an engorged jugular vein with a positive stasis test (Figure 1). In Cow 2, the vessels of the abdominal wall were prominent and visible on inspection. Cows 1, 2, and 4 presented serous to mucopurulent nasal discharge. On auscultation of the lung field of Cows 1, 2, and 4 detected tachypnea, areas of silence, lung wheezing, pleural friction, cough, expiratory dyspnea, open mouth breathing (respiratory distress), and expiratory grunts. Cow 3 presented a large amount of blood elimination through the mouth and nostrils, which characterizes a condition of severe hemoptysis (Figure 2).

In all animals, ruminal movements were reduced or absent and Cow 3 had a very distended, pear-shaped abdomen and diarrhea. The time of clinical evolution in the 4 animals ranged from 7 to 19 days, from the observation of the first clinical sign.

The ultrasound examination was performed on Cow 4 in a standing position using the Z5 Vet equipment1 with a convex transducer at a frequency of 5.0 MHz. The ultrasound scan of Cow 4 performed in the 12th intercostal space, showed a circular and dilated caudal vena cava in cross-section (Figure 3). The hepatic veins, especially the right vein, and the gallbladder were dilated. In the thoracic cavity, there was a pleural irregularity and pleural effusion in the ventral region.

Blood samples were collected by jugular venipuncture, in sterile vacuum tubes, without and with EDTA anticoagulant (Ethylenediamine Tetra-acetic Acid) for biochemical analyses and blood count, respectively. The hemogram was performed routinely and the serum enzymatic analyzes were performed in automated biochemistry equipment (BS- 120 Mindray)[1] using commercial reagents[2]. Data from the blood counts of Cows

1, 2, and 4 were included in the study. Additionally, from animal 2, the levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), and gamma--glutamyltransferase (GGT) enzymes were included. For data interpretation, blood count and biochemistry reference values were based on bovine species [16,25]. The blood counts of Cows 1, 2, and 4 revealed anemia and leukocytosis with neutrophil left shift. There was an increase in GGT (154 IU/L) enzyme activity and no increases in the serum activity of AST (64 IU/L) and ALT (18 IU/L) enzymes were seen.

As all animals died necropsy was performed on all 4 animals. Histopathological analysis was possible only in Cow 4, from which fragments of different organs fixed in 10% formalin were collected. Tissue samples were sent to the Pathological Anatomy Sector of the Federal Rural University of Rio de Janeiro (UFRRJ) and were routinely processed, embedded in paraffin, cut at 5 μm, and stained using the hematoxylin and eosin (HE)[3] technique.

The necropsy was performed and the common findings consisted of thrombi in the caudal vena cava, ranging from 7 to 30 cm in length (Figures 4 and 5), enlarged liver with rounded edges, ascites, and hydrothorax, enlarged heart with a globular appearance, especially in the right ventricle.

Specific alterations in Cow 1 were diffuse septic pododermatitis in the medial nail of the right pelvic limb and adhesion between the liver and the peritoneum and from the pleura on the right side to the lung, liver with a cut surface with "nutmeg appearance", in addition to a foreign body (nail) in the reticulum that caused traumatic reticuloabomasitis.

In Cow 2, there were accentuated edema in the omentum, rumen walls, intestines, in addition to Type 1 ulcers along the entire length of the abomasum. In Cow 3, there was blood clots in the trachea, bronchi, and in rumen contents. Another observed change in this cow was the liver with an irregular surface containing multiple abscesses. Lastly in Cow 4, infarcts areas in both kidneys (Figure 6), severe, multifocal, thromboembolic, and suppurative bronchopneumonia (Figure 7), extensive thrombi adhered to the pulmonary arteries (Figure 8), liver abscess in the caudate lobe and cutting surface with "nutmeg appearance" were seen.

The histopathological examination of Cow 4 carcass revealed occasional centrilobular hepatocytes with hypereosinophilic cytoplasm, pyknotic, karyorrhexic, and sometimes absent nuclei, as well as cell borders barely distinguishable (necrosis) in the liver. The sinusoid capillaries of the adjacent parenchyma were markedly congested with an extracellular accumulation of hemosiderin. In the lung, nodular and random formations were observed, with a thick wall of mature connective tissue and a central area full of cellular debris, necrotic cells, and intact and degenerated neutrophils (abscesses). Alveolar septa interspersed with bundles of abundant mature connective tissue (fibrosis) were noted. In the kidneys, from the medullary to the cortical layer, there was a well--defined area containing cells with hypereosinophilic cytoplasm, pyknotic to karyorrhexic nuclei, and slight loss of cell limits (coagulative necrosis - infarction). Irregularity in the vascular endothelium was observed, sometimes with large and reactive endothelial cells, and areas without the endothelium and exposure of the tunica media (erosion), associated with a moderate underlying lymphoplasmacytic inflammatory infiltrate.

DISCUSSION

CVCT was diagnosed in adult crossbred cows, with dairy aptitude, raised in semi-extensive and intensive systems. Similar data were reported in male and female cattle of different ages, breeds, and production systems [11]. However, our data diverged from the findings of some studies that observed a higher incidence of the disease in beef cattle, with no predilection for the breed, age, or sex [10,15].

The possible causes that led the animals to develop CVCT were diffuse septic pododermatitis in the medial nail of the right pelvic limb associated with traumatic reticuloabomasitis (Cow 1) and liver abscesses (Cow 3 and 4). In Cow 2, it was not possible to establish the probable cause of CVCT, but for the other cattle in the present study, the probable causes are in agreement with studies that have shown that this disease can occur as a sequel to several septic conditions such as jugular phlebitis, mastitis, hoof rot, enteritis, pneumonia, traumatic reticulopericarditis, acidosis and rumen laminitis, as well as omphalophlebitis in calves [6,21].

Clinical signs in Cows 1 and 2 involved the cardiorespiratory and digestive systems and led to the suspicion of CVCT, which was confirmed at necropsy.

In cows 3 and 4, it was possible to conclude the diagnosis of the disease by the clinical examination, because they presented clinical signs compatible with the

Figure 1. Positive jugular vein stasis test in Cow 4 with caudal vena cava thrombosis (CVCT).

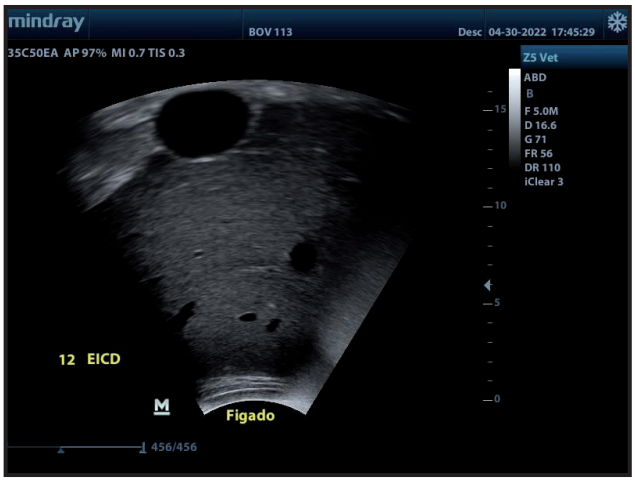

Figure 3. Ultrasonographic image of the caudal vena cava showing a circular appearance in Cow 4 with thrombosis of the caudal vena cava (CVCT).

Figure 2. Profuse mouth blood loss and epistaxis in Cow 3 with caudal vena cava thrombosis (CVCT).

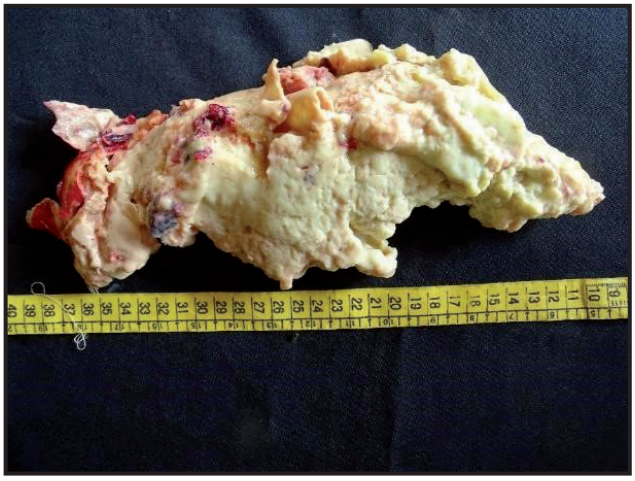

Figure 4. Extensive thrombus (27 cm long) with an irregular, yellowish appearance, with red areas, removed from the caudal vena cava in Cow 2 with caudal vena cava thrombosis (CVCT).

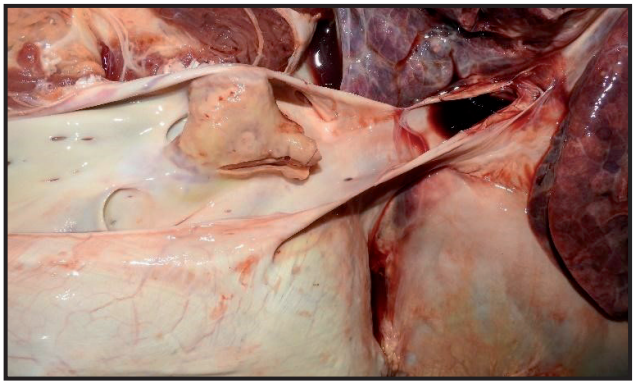

Figure 5. Extensive thrombus occluding the caudal vena cava in Cow 4 with caudal vena cava thrombosis (CVCT).

disease. Cow 3 presented severe hemoptysis and in Cow 4 the diagnosis was confirmed by the ultrasound findings in the vena cava with a circular and dilated appearance, as well as the evidence of ascites and hydrothorax.

The difficulty in concluding the diagnosis of CVCT based only on clinical examination has also been reported by some authors, as these are nonspecific clinical signs [2,6,23]. However, these authors reported that ultrasonography is the most reliable method for diagnosing CVCT in live animals, when performed in the 11^{th} or 12^{th} intercostal space on the right side, close to the liver, where the caudal vena cava normally appears with a triangular appearance, and which in CVCT becomes oval to circular and dilated, as seen in Cow 4 of the present study. This modification can be interpreted as a result of retrograde venous stasis exerted by the thrombus in the bed of the caudal vena cava. On the

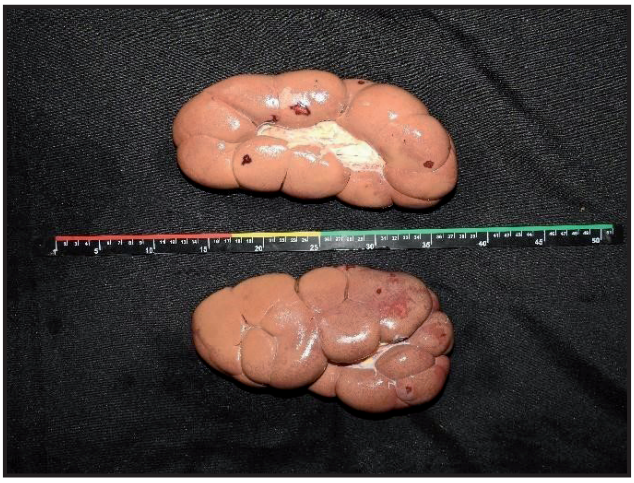

Figure 6. Numerous acute hemorrhagic infarcts of different sizes randomly distributed in the kidneys of Cow 4 with caudal vena cava thrombosis (CVCT).

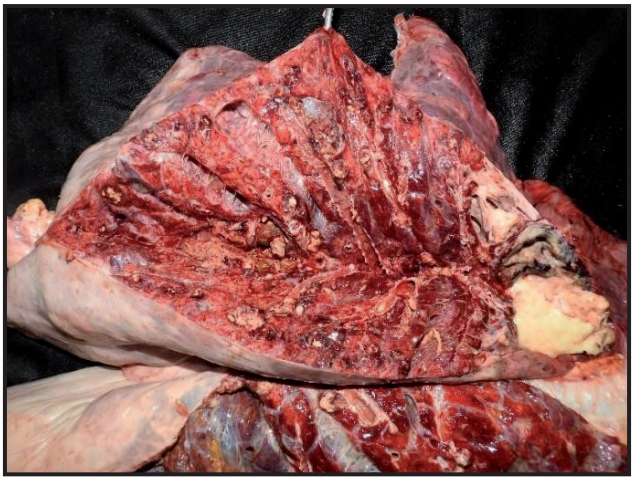

Figure 7. Severe, multifocal, thromboembolic and suppurative bronchopneumonia in Cow 4 with caudal vena cava thrombosis (CVCT).

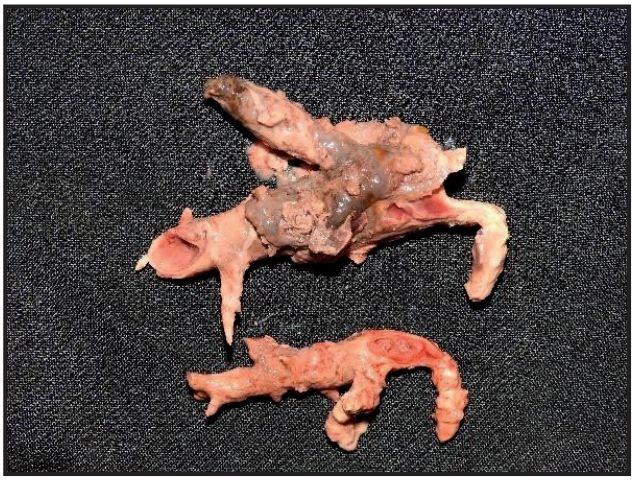

Figure 8. Extensive thrombi of irregular appearance, reddish, with gray areas, and crateriform foci, adhered to the pulmonary arteries in Cow 4 with thrombosis of the caudal vena cava (CVCT).

other hand, jugular engorgement (cranial vena cava) can be explained by the serious injury that occurred in the lungs of these animals, which prevents the normal and continuous flow of blood from the right ventricle.

Dilation of the hepatic veins that merge with the caudal vena cava, particularly the right hepatic vein, as seen in Cow 4 of this study, is also characteristic of congestion of the caudal vena cava [2].

In Cow 3, the elimination of blood through the mouth and nostrils is an evident clinical sign of thrombosis of the caudal vena cava, although nasal bleeding can occur in cases of pulmonary artery hypertension, with the development of ruptured aneurysms [22]. In addition, the thromboembolism can spread to other organs and result in endocarditis, embolic pneumonia, liver and kidney abscesses, or vessel erosion, which can culminate in intrapulmonary or intrabronchial hemorrhage and sudden death [17].

The tachypnea, serous to purulent nasal exudation, pulmonary wheezing, pleural friction, coughing, and expiratory dyspnea, usually with open mouth breathing and expiratory grunts evidenced in the animals of this study, occurred as a result of embolic abscess pneumonia [2]. It happens by the dissemination of emboli, which can lodge in the pulmonary artery, and cause embolism, multiple lung abscesses, and chronic suppurative bronchopneumonia.

The anemia observed in Cows 1, 2, and 4 can be explained by chronic inflammation and erosion/inflammation of blood vessels that can lead to blood loss [6]. Leukocytosis with neutrophil left shift and anemia presented by Cows 2 and 4 have also been reported in other studies [6,10,11,18,23]. Leukocytosis by neutrophil left shift is suggestive of an inflammatory and/or infectious process [6,19] and should always be associated with the animal clinic, as performed in this study. In the animals of the present study, this leukocyte alteration confirmed the poor prognosis of the animals.

The increase in serum GGT enzyme activity observed in Cow 2 can be explained by hepatic congestion due to obstruction of the caudal vena cava. The increase in serum GGT activity in cholestasis may be due to the higher production of the enzyme and the solubilization of GGT adhered to the cell membrane and occurs more slowly when compared to liver extravasation enzymes [24], such as AST and ALT, which showed no changes in their values.

In this study, necropsy findings were the most effective way to confirm the diagnosis of CVCT, which was also exposed by other authors [6].

All animals in the present study had thrombi in the caudal vena cava, which confirmed the diagnosis of the disease. The thrombus in the caudal vena cava is considered one of the main anatomopathological records in cattle with CVCT [8,14,23]. Other authors have already reported the presence of thrombi in all necropsies of animals with suspected CVCT [6]. However, studies reported a lower rate of thrombus in the animals studied by his group [15].

In Cows 3 and 4, thrombus formation in the caudal vena cava resulted probably from liver abscesses. A diet rich in carbohydrates leads to rumen acidosis, favoring the formation of abscesses, which increase in volume and, when they reach the wall of the caudal or hepatic vena cava, rupture, causing septic embolization and death [6]. This pathogenesis is compatible with the clinical status of Cows 3 and 4 of our study.

Liver abscesses are the most common cause of CVCT in dairy herds, as they are exposed to risk factors for the disease, such as intensive feeding management, which predisposes cattle to various digestive disorders, including ruminal acidosis [6,10]. Despite the low number of cows in the present study, two of them had liver abscesses, which confirms the authors' assertion.

In cattle with suspected vena cava thrombosis, care is required during necropsy. *Post mortem* examination should be performed before the liver removing from the carcass because, during this procedure, the thrombus or part of it can be easily lost [6].

Enlarged liver with rounded edges and firmer to palpation was observed in all cows. However, the "nutmeg appearance" was evidenced only in Cows 1 and 4. This finding was also reported by other authors and can be interpreted as a result of venous stasis due to obstruction of the caudal vena cava [5]. This liver aspect, together with the dilatation of the right ventricle of the heart, characterize a picture of congestive heart failure in affected cows.

The increase in fluid in the abdominal cavity (ascites) (Cows 1 to 4) and omental edema (Cow 2), in the present study, are similar to the observations of some authors who described these findings but did not mention the presence of fluid in the abdominal cavity thoracic cavity (Cows 1, 2 and 4) or edema in the walls of the rumen and intestine (Cow 2), as found in the animals of our study [4,6,11].

The large occlusive thrombus that was observed in the caudal vena cava (27 cm in length) in Cow 2 justifies the excess volume of fluid found in the abdominal and thoracic cavities, and the rumen, reticulum, and omentum walls edema.

Ascites associated with CVCT is rare and only develop when the thrombus is located cranially to the liver, occluding at least half of the vena cava lumen or by compression of the caudal vena cava by liver abscesses that cause hypertension [5,19].

The presence of multiple lung abscesses, areas of parenchymal consolidation, crateriform foci, and thrombi in the pulmonary arteries and chronic suppurative pneumonia, found at necropsy of the animals in the present study, are related to the development of a thrombus in the caudal vena cava that detaches and embolizes and lodges in the pulmonary arteries [2]. These findings also justify the changes in lung field auscultation at the clinical examination. Pulmonary hemorrhage with nostrils and mouth elimination of blood, with an accumulation of clotted blood in the trachea, bronchi, and ruminal contents of Cow 3, were also reported by other authors [6,19]. The hemorrhage occurs as a result of vessel erosion and consequent intrapulmonary or intrabronchial hemorrhage that can end in sudden death, which justifies the clinical picture of Cow 3 in the present study [17].

The renal infarctions observed in Cow 4 occur due to septic emboli that obstruct the vessels of the kidneys [6].

The histopathological findings of the Cow 4 are compatible with lesions found at necropsy and draw attention to embolic pneumonia and liver lesions, which, are related to thrombi in pulmonary arteries and abscesses formed from CVCT, as well as venous stasis exerted in the return circulation [2].

The set of diagnostic tools that include epidemiology, clinical signs and clinical examinations, ultrasound, necropsy, and histopathology were efficient in the diagnosis of CVCT.

This disease must be included in the differential diagnosis of diseases that course with cardiorespiratory signs, especially in dairy cattle. Especially the ultrasound examination was efficient to diagnose CVCT in live animal. The importance of necropsy as a fundamental tool for the diagnosis of CVCT is emphasized.

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Declaration of interest. The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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