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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Pulmonary thromboembolism associated with caudal vena caval thrombosis in a heifer

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Clinical History:

A 2-year-old heifer from a herd of approximately 300 Angus crossbred heifers kept at range was evaluated because of a 3-day history of hemoptysis (Fig. 1). The cow stopped eating and spent long periods standing in a pond away from the herd. Anti-inflammatory and antibiotic treatment was initiated with no avail. The heifer was euthanized and autopsied on the fourth day after the onset of clinical signs.



Figure 1. Clinical signs. hemoptysis

**The Diagnosti Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation (DTF). These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the DTF website: https://davisthompsonfoundation.org/diagnostic-exercise/*

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Autopsy:

The liver was slightly enlarged with an irregular surface. Small abscesses were distributed in the hepatic parenchyma (Fig. 2); on the cut surface, dark areas alternating with light foci (nutmeg liver) were distributed throughout (Fig. 3). The caval vein, at its insertion into the liver, was distended, firm, and markedly obstructed by compact, yellow material (pus and fibrin) (Fig. 4). The vein wall was thickened due to fibrosis. The lungs had nodular and coalescing, dark red areas predominantly in the caudal lobes (Fig. 5); on the cut surface, these areas were dark red (hematomas) and were accompanied by small nodular areas containing pus (abscesses) (Fig. 6). The interlobular septa were distended by translucent, gelatinous material; in the trachea, there was a moderate amount of brown, viscous content. There was no bacterial growth on culture with agar blood and MacConkey medium.

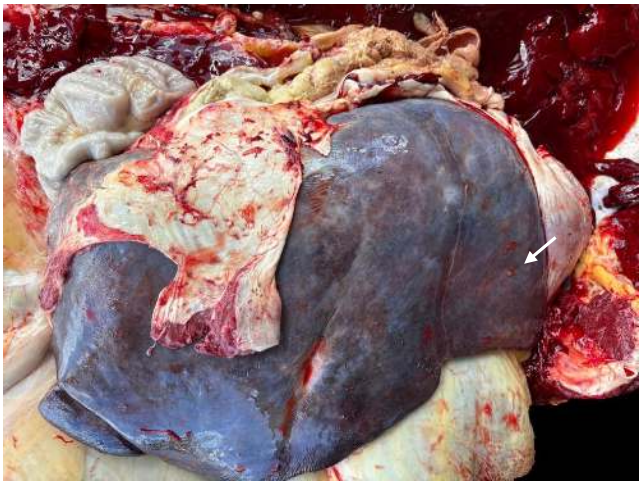


Figure 2. Chronic passive congestion, liver, heifer. The liver is slightly enlarged with an irregular surface; multiple small abscesses were distributed in the hepatic parenchyma (white arrow).



Figure 3. Chronic passive congestion (nutmeg liver), liver, cut surface, heifer.

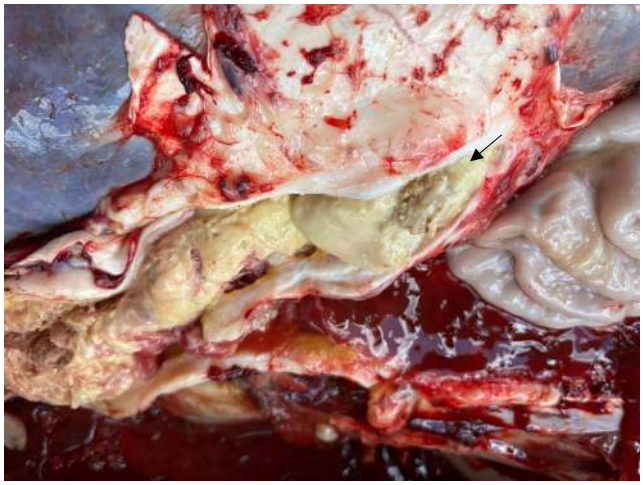


Figure 4. Thrombophlebitis, caudal vena cava, heifer. The caudal vena cava has a large and long thrombus (arrow) attached to the thickened wall of the vein.

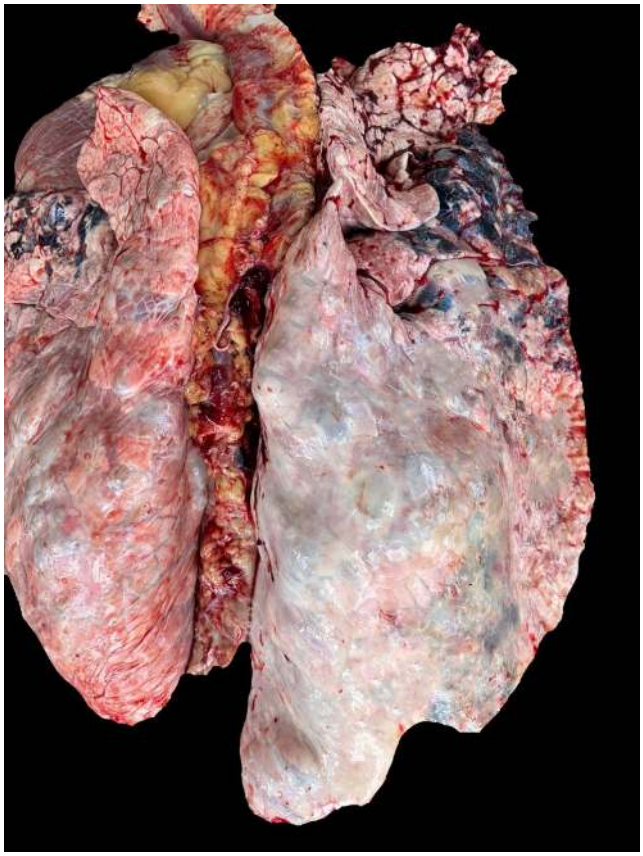


Fig. 5. Lung. Multiple pulmonary hematomas, heifer. The pleural is irregular due to Prominent black nodules protruding from the lung parenchyma.

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Follow Up Questions:

- *Morphologic diagnosis*?
- *Name of the condition*?

ANSWERS

Histopathology:

In the intima of the caudal vena cava there were areas of inflammation composed predominantly of intact and degenerate neutrophils associated with marked thickening of the tunica media. The liver had chronic passive congestion of the centrilobular veins and vacuolization and necrosis of hepatocytes in adjacent areas (Fig. 7). In the lung, there were thrombi with marked inflammatory infiltrates composed of intact and degenerate neutrophils (Fig. 8). The wall of the vena cava was thickened because of inflammation and fibrosis (Fig. 9). There were focally extensive areas of necrosis, edema of interlobular septa, and marked inflammatory infiltrates of intact and degenerate neutrophils in the airways. There was focally extensive suppurative pneumonia, mainly around the areas of thrombosis. In the kidneys, there were randomly distributed tubular hyaline casts.

Morphologic diagnosis:

Liver, chronic passive congestion

Vena cava, vasculitis and fibrosis, chronic, with thrombosis

Lung, embolic suppurative pneumonia and hemorrhage

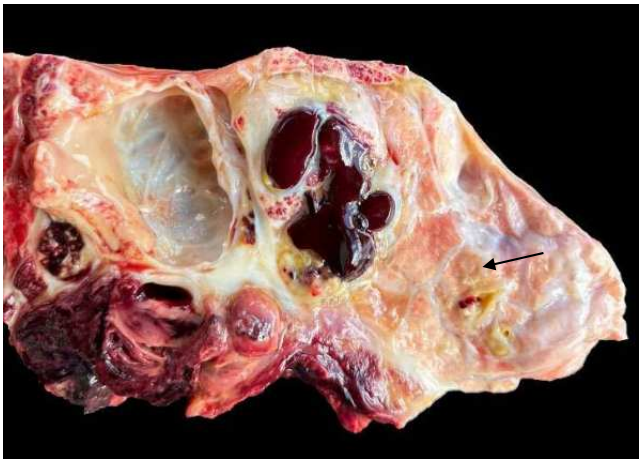


Fig. 6. Lung. Cut surface. Multiple pulmonary hematomas and small abscesses (arrow), heifer.

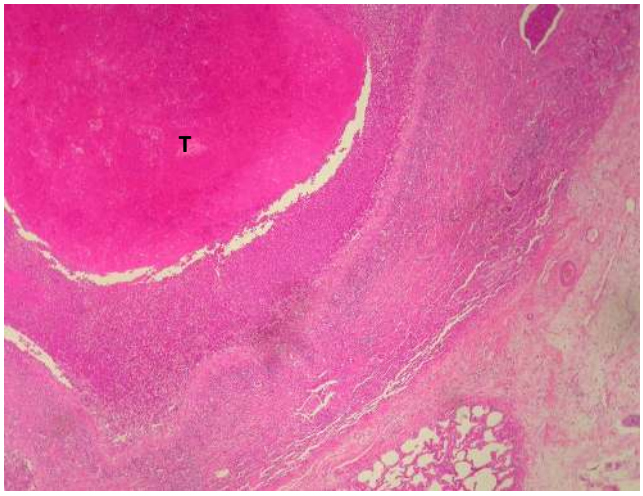


Figure. 8. Lung. A thrombus (T) occludes a branch pulmonary vessel, heifer. The wall of the vessel is thickened by sever inflammation.

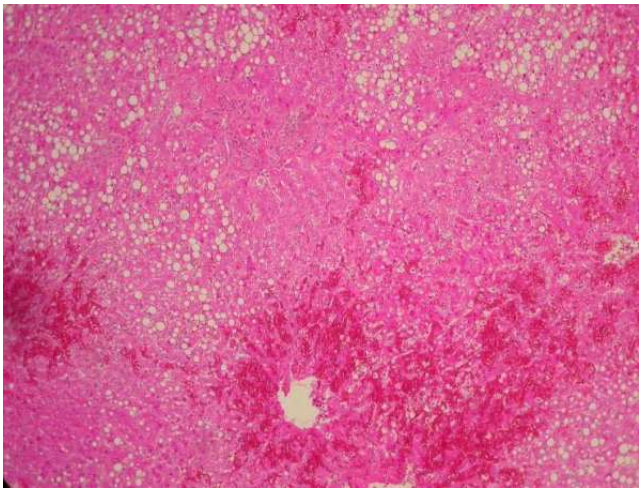


Figure 7. Liver. Histopathology, chronic passive congestion, heifer.

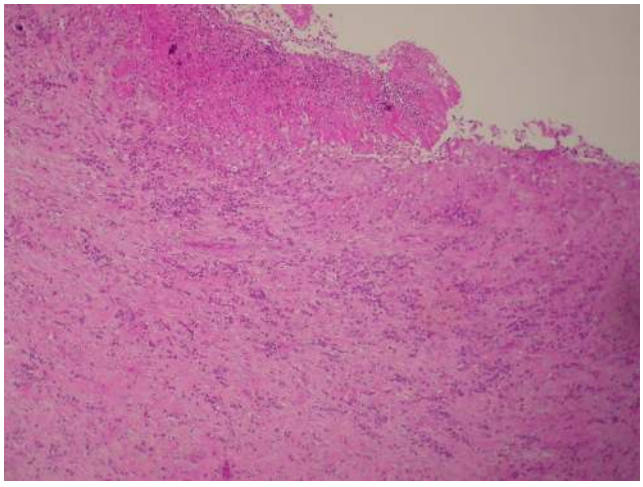


Figure 9. Vena cava wall. Marked vasculitis and fibrosis, heifer.

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Name of the condition:

Pulmonary thromboembolism associated with caudal vena cava thrombosis

Comments:

Metastatic pneumonia or thromboembolism in cattle, also known as caudal vena cava thrombosis, pulmonary thromboembolism, embolic pulmonary aneurysm, vena cava syndrome, results from the formation of abscesses in the lungs caused by septic thromboemboli that reach the pulmonary circulation. The emboli arise from septic thrombi of the caudal vena cava or, less frequently, of the cranial vena cava. The origin of vena cava thrombi is the consequence of various septic events, such as jugular phlebitis, mastitis, metritis, hoof rot, or, more frequently, liver abscesses secondary to rumenitis. Several bacteria may be involved in this condition (2,7).

Vena cava syndrome mainly affects dairy and feedlot cattle (2,3) and is often associated with diets rich in concentrated feed (6). The most frequently observed clinical signs are hemoptysis, dyspnea, tachypnea, cough, pale mucous membranes, and anorexia. The progression of the condition can vary from 2-18 days, and hyperacute cases may occur with death shortly after the onset of clinical signs (5).

Septic thrombophlebitis of the caudal vena cava, liver abscesses, pulmonary hemorrhage, and embolic pneumonia were the main lesions observed in a study of 30 cases of the disease observed in dairy cows (5).

In the classic pathogenesis of this condition, rumenitis occurs initially secondary to lactic acidosis caused by highly fermentable diets used in feedlots and cattle stations. In toxic rumenitis, necrosis of the rumen lining epithelium occurs. Bacteria such as *Fusarium necrophorum*, *Trueperella pyo*genes, staphylococci, streptococci, and *Escherichia coli* (1,2,4,5,7) penetrate through the rumen and are transported to the liver by the portal system, where they are filtered and cause abscesses. Abscesses on the left edge of the liver, close to the caudal vena cava, can form septic thrombus by infiltrating the vein wall. Eventually, septic fragments break away from the thrombus to reach the lungs through the pulmonary arterial system. Other approaches to the lung are rare and include cranial vena cava thrombosis from primary lesions such as jugular phlebitis; caudal vena cava thrombosis from other subdiaphragmatic abscesses; mitral endocarditis; and emboli from specific other septic foci such as mastitis, metritis, and foot rot (7). Large emboli may block lobar or even larger arteries, causing death after acute respiratory distress.

More typically, smaller emboli lodge in arterioles, where they cause arterial thromboembolism, endarteritis, and lung abscesses. Disseminated arterial emboli, pulmonary arterial hypertension, and weakened vascular walls lead to formation of aneurysms.

Large abscesses cause aneurysms and, at the same time, erode a nearby bronchial wall. Thus, when the aneurysm ruptures, the abscess cavity opens into the bronchus, communicating the two channels (blood vessel and airway) resulting in hemoptysis and/or large interstitial hematomas. Both processes result in anemia. If the animal swallows the blood, melena may occur.

In the current case, the heifer had profuse bleeding from the mouth, severe respiratory distress, apathy, and anorexia that lasted for three days. This clinical presentation is strongly indicative of vena cava syndrome (5,7).

The fact that the heifer was a beef breed raised on pasture suggests that rumenitis due to acidosis was the primary cause in this case is very small. Accordingly, no lesions were observed in the mucosa of the fore stomachs at autopsy.

Although also unlikely, an umbilical infection may have caused liver abscesses, which, over time, induced septic thrombi that lodged in the vena cava. Cases of omphalophlebitis have already been associated with the occurrence of vena cava syndrome in young cattle (6).

In the case of this report, there was no bacterial growth from the abscesses. The heifer was treated with anti-inflammatory and antibiotics, which may have impaired bacterial growth.

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