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Ruptured Hepatic Abscesses in Cattle

William S. Monlux, D.V.M., PhD*

One of the important complications of hepatic abscesses in cattle is their extension into the posterior vena cava and the hepatic veins. When this occurs the abscesses discharge their contents into the blood stream and produce a thrombus at the site of endothelial injury.

The incidence of the disease is in direct proportion to the number of livers affected with hepatic abscesses. This lesion involves three to five per cent of adult cattle which are presented for postmortem examination. As pointed out by Rubarth, who has reported the greatest number of these cases, it is surprising how little attention this lesion has attracted in veterinary literature. In the 1,279 postmortem examinations of mature cattle reported by Rubarth, he found that 53 animals had hepatic abscesses, two had thoracic abscesses, and one had a retroperitoneal abscess. Fifty-two of these abscesses had ruptured into the posterior vena cava and one into a hepatic vein. These figures indicate that the incidence of hepatic abscesses with rupture into the posterior vena cava and the hepatic veins in the Rubarth series of cases is 4.5 per cent.

Two forms of the disease are observed. The first is characterized by sudden death with no previous history of illness and the second, by vague digestive disturbances followed by sudden respiratory complications.

Fifty-one of the 56 animals described by Rubarth, as having hepatic and subphrenic abscesses with rupture into the posterior vena cava or a hepatic vein, were found dead in the stable or on pasture. Two bulls and a cow in this group died following service. Only five animals showed any clinical signs of illness. Three cattle appeared to suffocate, one cow showed respiratory distress and diarrhea, and the fifth suddenly began to champ her jaws, fell forward, and died. In addition to the 56 animals submitted for postmortem examination, Rubarth also examined an additional 60 specimens submitted as lesions from post-mortem examinations made by other veterinarians.

A very rapid course is characteristic of this disease. The cattle observed by the author have not terminated in sudden death as frequently as those described by Rubarth. Since a complete clinical history was not available for all of these animals, only an estimate can be made as to the course of the disease. About 60 per cent of those animals observed have died suddenly, while about 40 per cent have had a more lingering type of illness.

The reason this lesion is usually overlooked is that complete postmortem examinations of cattle are seldom made. Even if all of the viscera is removed from the

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cadaver, it is seldom that the posterior vena cava and the hepatic veins are opened. It is absolutely essential that these structures be carefully examined otherwise the cause of nearly five per cent of bovine deaths cannot be explained. This is particularly true if death occurs suddenly, and especially if it is associated with respiratory distress and symptoms of shock.

Postmortem examination of the cadaver is quite typical. The first alteration which leads one to suspect that this type of lesion is present is that the blood is well coagulated of the red or currant jelly type, and the clot is unusually firm having somewhat of a rubber-like consistency. These clots are best observed in the heart and major blood vessels. The increased rate of coagulation of the blood is due to the discharge of purulent material from the hepatic abscesses into the circulatory system. One of the characteristics of a suppurative disease is the rapid and complete postmortem coagulation of blood.

There is an acute general passive hyperemia which is especially severe when pulmonary infarction, pneumonia, and edema and congestion of the lungs are present. The cattle usually have the lesions of a septicemia. There are multiple petechial and ecchymotic hemorrhages in many organs. If the animal lives for several hours after the blood stream is invaded with bacteria there will be hyperplasia of the spleen and lymph nodes. Those animals which die suddenly show acute general passive hyperemia, slight general edema, and edema and congestion of the lungs, the lesions associated with shock.

Two types of lesions are observed in the hepatic veins and posterior vena cava. The first, is rupture of a perivascular abscess into the venous circulation. When examining the vein an opening is observed in the wall from which a sinus tract extends into the cavity of the abscess located within the hepatic parenchyma (Fig. 1). Most of the purulent material in the abscess has usually been discharged into the circulatory system.

Fig. 1. A hepatic abscess which has perforated into the vena cava. Pus is still present within the abscess. This is the abscess which produced the thrombus shown in Fig. 2. The narrow sinus tract leading from the abscess to the lumen of the vein can be seen.

The second, is thrombosis of the hepatic veins or the posterior vena cava adjacent to a hepatic abscess (Fig. 2). Abscesses within the parenchyma of the liver can frequently be observed through the walls of the veins and some of them actually bulge into the lumens. These protruding abscesses, which are nearly perforating the wall of the vessel, damage the endothelial lining of the vein and thrombosis of the vein occurs. Since the blood passing through these large veins is flowing quite rapidly, a white thrombus (platelet thrombus) is formed rather than the red thrombus ordinarily observed in veins.

Fig. 2. A thrombus located in the posterior vena cava.
Complete occlusion of the hepatic veins may take place. No complete occlusion of the posterior vena cava has been observed, but the lumen, at times, has been so reduced in diameter that only a small amount of blood could pass through it. The thrombi within the posterior vena cava and hepatic veins vary in size from slight roughnesses on the endothelial surface to large masses which may measure as much as 12 centimeters in length and five centimeters in width. When large thrombi are found in the posterior vena cava there is local passive hyperemia of the viscera and the posterior half of the body. Those animals having the local passive hyperemia have shown weakness of the posterior extremities and vague digestive disturbances which have been diagnosed as traumatic gastritis, atony of the forestomachs, and displacement of the abomasum.

As pointed out by Rubarth, the hepatic veins will, at times, carry the blood from the caudal portion of the body through the hepatic parenchyma around the obstruction in the posterior vena cava and return it to the vena cava anterior to the obstruction. When this occurs the hepatic veins, which are shunting the blood around the obstruction, become large cavernous structures.

The bacteria present in the abscess invade the thrombus. There they attract leukocytes and the proteolytic enzymes of the leukocytes (primarily neutrophils) cause liquefaction and disintegration of the thrombus. As the thrombus disintegrates, septic emboli are carried into the lungs.

Emboli of bacterial size pass through the lungs and on into the general blood vascular circulation of the body. There they multiply and a septicemia is the result. This is one of the more common causes of septicemia in the bovine.

Septic emboli, slightly larger than bacteria but too large to pass through the pulmonary capillaries, are scattered throughout all lobes of the lungs. This occurs because they are so small in size that the centripetal force of the blood stream is not exerted upon them. Therefore, they are evenly distributed throughout the pulmonary tissue. Since these emboli contain bacteria, multiple pulmonary abscesses scattered throughout the entire lung is the result.

When larger emboli are discharged into the venous circulation, the centripetal force of the blood stream carries them into the large veins of the diaphragmatic lobes of the lungs. Since they are septic, infarction of the posterior portion of the diaphragmatic lobes is the result. The infarction of the lungs is the reason for the sudden respiratory distress observed in these animals.

Bacteriological examination of the hepatic lesions, pulmonary lesions, and blood of these animals reveals that Corynebacterium pyogenes is the organism most frequently observed. In Iowa and the eastern portion of the United States the findings are in agreement with those of Rubarth in which about 60 per cent of the lesions will contain Corynebacterium pyogenes, 10 per cent Streptococcus sp., 10 per cent Sphero­phorus necrophorus, and 20 per cent a mixture of Escherichia coli, Streptococcus sp., Corynebacterium pyogenes, Spher­phorus necrophorus, and saprophytic organisms.

Cattle originating from the Rocky Mountain States have a very high incidence of hepatic lesions due to Sphero­phorus necrophorus and a relatively low incidence of Corynebacterium pyogenes. As a result, these cattle will have a correspondingly different etiologic cause of the hepato-vascular lesions.

Since Sphero­phorus necrophorus is not a pyogenic microorganism, the sudden death due to rupture of a hepatic abscess into the venous circulation does not occur. The true Sphero­phorus necrophorus lesion is that of coagulative necrosis. The necrotic material is dry and firm and not of a consistency which will flow as will pus. Sphero­phorus necrophorus produces thrombosis of the hepatic veins and posterior vena cava, and the course of the disease is that of a lingering illness.

REFERENCE