Pulmonary Thromboembolism in Cattle Due to Thrombosis of the Posterior Vena Cava Associated with Hepatic Abscessation

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One of the most common causes of pulmonary thromboembolism in cattle is thrombosis of the posterior vena cava associated with hepatic abscessation.

The purpose of this paper is to briefly review the literature on the subject and to report on the prevalence and clinicopathological findings on the disease in cattle in Saskatchewan.

LITERATURE REVIEW

Pulmonary thromboembolism in cattle may be a sequel to several different diseases or lesions including phlebitis of the jugular veins, mastitis, metritis, traumatic reticuloperitonitis, hepatic abscessation, infection of the interdigital space (28) and thrombosis of the posterior vena cava associated with hepatic abscessation (9).

Pulmonary thromboembolism as a result of thrombosis of the posterior vena cava has been described in Europe (4, 5, 6, 21-23, 27, 28) and in the U.S.A. (17). At the Royal Veterinary College in Sweden from 1935 to 1960 the incidence was 4.35% of 1,279 adult cattle examined at necropsy (21). In a continued study from 1961 to 1966 the incidence was 1.7% of 687 necropsies of cattle (22). In the U.S.A. the necropsy of 1,998 yearling feedlot cattle revealed an incidence of 1.3% (17) and 40% occurred during the first 45 days of the feedlot period and 28% during the next 45 days.

The clinical findings in cattle affected with pulmonary thromboembolism include coughing, inspiratory dyspnea, hemoptysis and abnormal lung sounds on auscultation. Thoracic pain may be detectable on deep palpation of the abdomen at the level of the sternum (4, 5, 6, 17, 21-23, 27, 28). Anemia is common in the later stages and hepatomegaly may be present (5, 6, 23, 27). Melena due to the coughing up and swallowing of blood may occur in some cases (5). The pulmonary hemorrhage may be fatal and exhibited clinically by epistaxis and hemoptysis.

Endocarditis is not generally a feature but may occur in about 10% of cases (28). Hypergammaglobulinemia may also occur in long standing cases (28). Treatment is ineffective and the case fatality rate is usually 100% (27).

All of the vascular lesions are complications of hepatic abscessation and thrombosis of the posterior vena cava. The thrombus forms as a result of thrombophlebitis of the vessel due to infiltration by a perivascular hepatic abscess. Thrombosis of the posterior vena cava results in embolism to the pulmonary arteries, pulmonary arterial thromboembolism, pulmonary arteritis, chronic suppurrative pneumonia and the formation of multiple pulmonary abscesses. Pulmonary arterial aneurysms may occur and rupture of these cause extensive intrapulmonary and intrabranchial hemorrhage (4, 5, 6). These lesions explain the clinical findings of dyspnea, abnormal lung sounds and hemoptysis. Hemoptysis occurs because the pulmonary arteries closely follow the air passage distribution to the level of the terminal bronchioles (2). The causes of death include coronary arterial occlusion, congestive heart failure, cerebral hypoxia (21, 27), respiratory insufficiency and fatal pulmonary hemorrhage (5).

At necropsy the most remarkable lesions are in the lungs, posterior vena cava and liver. There is pulmonary edema, emphysema and intrapulmonary and intrabranchial hemorrhage. The intrapulmonary hemorrhage due to the ruptured aneurysms may result in large globular masses of clotted blood adjacent to a dilated portion of a pulmonary artery. Large deposits of clotted blood may be present in the major bronchi and trachea. A large thrombus is usually present in that portion of the posterior vena cava which lies between the liver and the right atrium. There is usually an adjacent hepatic abscess and varying degrees of venous congestion of the liver and hepatomegaly are present depending on the size of the thrombus in the posterior vena cava. Clots of blood may be present in the rumen.

The hepatic abscesses are due to infection of the portal circulation with pyogenic bacteria from suppurrative lesions elsewhere in the body or more commonly as a complication of rumenitis caused by lactic acidosis associated with carbohydrate engorgement (1, 10, 15, 25). Hepatic abscesses may contain a mixture of Gram positive and Gram negative bacteria (7) and while Fusiformis necrophorus is the most common organism isolated (8, 16, 24, 26), Corynebacterium pyogenes is also a common isolate (21). The two species of organisms occur commonly together in liver abscesses and exhibit a pyogenic synergy (18, 19). Whereas antibiotics in the feed have been used to reduce the incidence of hepatic abscessation with varying results (3, 12, 13, 20) the rate of change and

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proportion of concentrate in the diet are the important factors in the development of rumenitis (14). The random anatomical distribution of abscesses in the liver of cattle is due to the lack of right and left laminar flow in the portal circulation of the bovine liver (11).

M A T E R I A L S A N D M E T H O D S

The necropsy records of all cattle affected with hepatic abscessation and thrombosis of the posterior vena cava from January, 1968 to March, 1974 at the Western College of Veterinary Medicine. The epidemiological data, clinical history and necropsy findings were collected in an attempt to characterize the disease as it occurs in Saskatchewan.

In addition, 40 livers condemned for abscessation and adhesion were obtained from a local abattoir. Each liver was diagramatically divided into quadrants and the parietal and visceral lesions, including scars, were counted. Each liver was then cross sectioned at intervals of 2 cm and the subsurface lesions counted. Notations of gross vascular involvement were also made. Selected areas of abnormal liver and gall bladders were taken and fixed in 10% formalin for microscopic examination.

The incidence of condemnation of livers due to hepatic abscessation was determined by examination of the daily inspection records of the abattoir during the first and last ten week periods of 1973.

R E S U L T S

Statistical Epidemiology

During the period of study, six years and three months, the incidence of hepatic abscessation was 2.26% (171 of 7,545 bovine necropsies). Thrombosis of the posterior vena cava occurred in 19.3% of cases of hepatic abscessation. There were four cases of thrombosis of the vessel without evidence of hepatic abscessation.

The seasonal incidence, the time of death after clinical signs, the circumstances of death and whether or not the animal came from a feedlot are summarized and appear in Figure 1. There appeared to be an increased incidence in March and April, and from September to December which are periods of the year which coincide with the fattening and finishing periods of feedlot cattle. Sudden death accounted for 43.2% of cases of thrombosis of the posterior vena cava, death occurred within 24 hours after the onset of clinical signs in 21.6% and the remainder of 35.2% had an illness which lasted from two to 18 days. Those cases from feedlots accounted for 40.5%.

The disease occurred in cattle from three weeks to 12 years of age. Approximately 46% of all cases occurred in cattle from one to two years of age and only three cases occurred in animals less than one year of age. There was no evidence of sex or breed predilection.

During the 20 week period surveyed at the abattoir, 23.7% of the livers were condemned for abscessation and/or adhesions from a total of 20,226 cattle and 684 calves slaughtered. The lesions present in the 40 abattoir specimens were concentrated in quadrant 1, the quadrant consisting of the largest mass of liver (Figure 2). Fourteen (35%) of the 40 livers had abscesses which were adjacent to blood vessels with the
FIGURE 3. An abscess (A) involving the posterior vena cava (B). A photographic enlargement of H & E section from a condemned liver.

FIGURE 4. The bile duct from a condemned liver with cholangitis showing lymphocytic infiltration. H & E x 15.

FIGURE 5. The gall bladder from a condemned liver with cholecystitis showing lymphocytic infiltration. H & E x 15.

FIGURE 6. Lung showing alveolar, lobular and intrabronchial hemorrhage. Also note interlobular edema present. A photographic enlargement of an H & E section.

FIGURE 7. Lung showing lobular separation due to edema, noncollapse due to emphysema and lobular hemorrhage in varying stages of resolution.

and cholecystitis were present in one liver (Figures 4 and 5).

Clinical History
The clinical findings reported included respiratory insufficiency with coughing and hemoptysis, an expiratory grunt, subcutaneous emphysema and frothing at the mouth. Digestive disturbance included ruminal stasis, scant feces and inappetence. Nervous signs were seen in one animal with a history of lameness, weakness and recumbency. Most affected cattle were in good bodily condition; only two were in poor condition. The tentative clinical diagnosis submitted included pneumonia, hepatic abscessation, traumatic reticuloenteritis and its various sequelae, brain abscess, hydrops amnion and poisonings.

Pathology
On gross examination of the lungs there was usually extensive emphysema and edema (Figures 6 and 7). Hemorrhage was lobular in distribution (Figure 7). Rupture of the pulmonary arteries resulted in intrapulmonary hemorrhage and the formation of large hematomas, and intrabronchial hemorrhage (Figure 8) was characterized by clots
FIGURE 8. A cross section of lung with hemorrhage into the bronchial tree due to caval thrombosis and pulmonary embolism.


of blood in the major bronchi and trachea. Thrombosis of the pulmonary arteries was distributed at random throughout the lung (Figure 9) and there was a close association between thrombosed vessels and the lumen of the bronchi. Microscopically there was edema, hemorrhage, pulmonary arterial embolism, and focal pneumonia (Figure 6).

The most consistent lesion in the digestive tract was hepatic abscessation which was associated with thrombosis of the posterior vena cava in 33 out of 37 cases. Diaphragmatic adhesions were present in 60% of the cases. The abscesses varied in size and some ruptured directly into the posterior vena cava (Figure 10). The size of the thrombus of the posterior vena cava was variable (Figures 10-12) and the thrombus was most frequently located in the area where the posterior vena cava passes through the diaphragm. In one case, a hepatic abscess 8 cm lateral to the posterior vena cava had an inflammatory reaction extending through the diaphragm to involve the vessel 7.5 cm anterior to the diaphragm. In another case, an abscess which was present in a lymph node, between the liver and the posterior vena cava, initiated the thrombus formation (Figures 11 and 12). Chronic passive congestion of the liver was present in some instances and was usually due to a large thrombus of the posterior vena cava which almost totally occluded the vessel. Blood was present in the rumen of 30% of all cases of pulmonary thromboembolism and in 75% of these, blood was present in the oral cavity. There was evidence of ruminal scars in one case and mild rumenitis in another. Septic embolism occurred in the kidney, brain and mesenteric artery of two cases.
DISCUSSION

The results of the retrospective study of the necropsy records are in general agreement with the published literature on pulmonary thromboembolism due to thrombosis of the posterior vena cava in association with hepatic abscession in cattle (4, 21-23, 27, 28). Hepatic abscesses may cause thrombosis of the posterior vena cava, usually near the location where the vessel passes through the diaphragm. Thromboembolism of the pulmonary arteries is a common complication which results in suppurative embolic pneumonia, pulmonary arterial aneurysms which may rupture and cause fatal hemorrhage. The presence of an hepatic abscess is not a prerequisite; any infectious process, such as an abscessed lymph node, adjacent to the posterior vena cava may initiate the thrombus formation. The syndrome may occur as early as three weeks of age in which case it is probably associated with omphalophlebitis rather than the rumenitis-liver abscess complex which occurs in yearling and adult cattle.

In the livers obtained from the abattoir the largest number of abscesses were present in the largest quadrant of the liver from which the posterior vena cava originates. The abscesses were close to and involving blood vessels but not their endothelium in 35% of the abattoir livers. The higher incidence of abscesses in the abattoir livers compared to the livers from the necropsy examinations requires further epidemiological examination. The difference may be due to the failure to examine all dead animals, incomplete necropsies and the practice of slaughter for salvage which may spuriously increase the incidence of hepatic abscession in cattle examined at the abattoir compared to a diagnostic laboratory (21, 27).

The disease complex occurs with sufficient frequency that it should be considered at necropsy when the lungs are affected with extensive emphysema, edema and hemorrhage, and clinically when cattle are affected with a sudden onset of respiratory insufficiency and hemoptysis. Because the case fatality rate is usually 100%, an accurate clinical diagnosis is desirable and slaughter for salvage is the disposition of choice.

SUMMARY

During a period of six years and four months the incidence of hepatic abscession in 7,545 cattle examined by necropsy in a diagnostic laboratory was 2.26% and thrombosis of the posterior vena cava was present in 19.3% of all livers with abscesses. The clinical histories indicated that 65% of the animals affected with thrombosis of the posterior vena cava died within 24 hours after clinical signs were noted while 35% lived for two to 18 days. The clinical signs were referable to pulmonary thromboembolism due to thrombosis of the posterior vena cava usually associated with hepatic abscession.

A survey of livers from cattle slaughtered in an abattoir during a 20 week period revealed that 23.7% of all livers were condemned for abscession and adhesions. In 40 of the condemned livers there was a 35% incidence of gross perivascular and/or vascular involvement by abscesses.

RÉSUMÉ

Au cours d’une période de 76 mois, l’incidence d’abcès hépatiques chez 7,545 bovins dont on effectua la necropsie dans un laboratoire de diagnostic, s’élevait à 2.26%. On démontra la présence d’une thrombose de la veine cave postérieure dans 19.3% des cas d’abcès hépatiques. L’anamnèse révela que 65% des sujets atteints d’une thrombose de la veine cave postérieure moururent en l’espace de 24 heures après l’apparition des signes cliniques, tandis que 35% survécurent de deux à 18 jours. Les signes cliniques permettaient de soupçonner une thrombo-embolie pulmonaire, attributable à une thrombose de la veine cave postérieure résultant d’abcès hépatiques.

Une analyse de la condamnation du foie des bovins abattus dans un abattoir, sur une période de 20 semaines, révéla que 23.7% de tous ces foies subirent la condamnation à cause d’abcès et d’adhérences; 35% des 40 foies condamnés présentaient des abcès péri ou intra-vasculaires.

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REFERENCES


ABSTRACT

Scottish workers have previously reported briefly on the use of ultraviolet light to demonstrate areas of brain necrosis. The technique has proved useful in the diagnosis of polioencephalomalacia. Studies in New Zealand by the author have confirmed the value of this technique for the rapid identification of this disease and for extending studies on the distribution of the brain lesions.

Ultraviolet light at 365 nm leads to autofluorescence of the cortical lesions, however in mild cases and animals affected less than two to three days the test may be spuriously negative.

Preliminary studies of affected brains using Folch-Lee extraction, thin layer chromatography and fluorescent microscopy have identified the fluorescence originating in cereoid-lipofuscin in lipophages. This explains the tests failure in early and mild cases when insufficient autofluorescent pigment has yet formed.

The application of this test for laboratory diagnosis of polioencephalomalacia reduces the diagnostic interval from two to three days to several minutes in most cases. The test can be expected to be useful to identify malacic foci caused by other pathological processes.

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