Case Report  Rapport de cas

Caudal vena cava thrombosis-like syndrome in a horse

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Abstract – A 9-year-old Quarter horse was presented for chronic refractory pneumonia. On necropsy, an hepatic abscess, caudal vena cava thrombosis, pulmonary thromboembolism, and embolic pneumonia were identified. Similar lesions have been reported in cattle as caudal vena cava thrombosis syndrome, however this syndrome has not previously been reported in horses.

Résumé – Syndrome de la veine cave caudale s’apparentant à une thrombose chez un cheval. Un cheval Quarter horse âgé de 9 ans est présenté pour une pneumonie réfractaire chronique. À la nécropsie, un abcès hépatique, une thrombose de la veine cave caudale, un thromboembolisme pulmonaire et une pneumonie embolique ont été identifiés. Des lésions semblables ont été signalées chez le bétail comme étant le syndrome de la thrombose de la veine cave caudale; cependant, ce syndrome n’avait pas déjà été signalé chez les chevaux.

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A 9-year-old, 450 kg American Quarter horse gelding was referred to the Ontario Veterinary College Teaching Hospital (OVC-TH) due to signs of pneumonia of 1 month’s duration. Prior to referral, the gelding had been treated by the referring veterinarian for pyrexia and weight loss following transportation from Oklahoma to Ontario, Canada. Pleuropneumonia (shipping fever) was suspected at that time, and the horse was treated with penicillin, gentamicin, metronidazole, phenylbutazone, and flunixin meglumine. The horse’s condition failed to improve; therefore, he was referred to the OVC-TH.

Case description

On presentation, the gelding was dull, tachycardic (heart rate 64 beats/min), dyspneic and mildly tachypneic (respiratory rate 24 breaths/min), and pyrexic (rectal temperature 39.8°C). Increased bronchial sounds were auscultated diffusely over the lungs bilaterally. Mucopurulent nasal discharge and cough were evident. Mucous membranes were pale. Venous blood gas values, including pH, pO2, pCO2, BE, and HCO3–, were within normal limits. Hemogram and serum biochemical abnormalities included anemia [red blood (cell) count 5.14 × 1012/L, reference range: 6.9 to 10.7 × 1012/L; hematocrit 23 L/L, reference range: 28 to 44 L/L], leukocytosis (17.4 × 109/L, reference range: 5.1 to 11 × 109/L), neutrophilia (14.1 × 109/L, reference range: 2.8 to 7.7 × 109/L), hyperfibrinogenemia (4.2 g/L, reference range: 0.9 to 2.8 g/L), hypoalbuminemia (19 g/L, reference range: 30 to 37 g/L), and hyperglobulinemia (65 g/L, reference range: 26 to 41 g/L)]. Bronchoscopy revealed large quantities of mucopurulent discharge in the trachea. Culture of an endoscopic tracheal aspirate yielded large numbers of Streptococcus equi ssp. zooepidemicus, which was susceptible to erythromycin, rifampin, ampicillin, cefiofur, chloramphenicol, and penicillin. Ultrasonographic evaluation of the thoracic cavity showed bilateral pleural roughening with areas suspicious for pulmonary abscession on the right side. No free fluid was visible in the pleural space. Thoracic radiographs showed a diffuse peribronchial interstitial pattern with alveolar opacities in some areas. Septic pneumonia was diagnosed.

The horse was hospitalized and treatment was initiated with intravenous fluids lactated Ringer’s solution (Baxter, Mississauga, Ontario), 4 to 6 mL/kg per hour, IV; flunixin meglumine (Cronyxin; Bioniche Animal Health Canada, Belleville, Ontario), 1.1 mg/kg, q12h, IV; erythromycin meglumine (Cronyxin; Bioniche Animal Health Canada, Belleville, Ontario), 1.1 mg/kg, q12h, IV; erythromycin phosphate (Gallimycin PFC; Vetoquinol N-A, Montreal, Quebec), 37.5 mg/kg, q12h, PO; and rifampin (Rofact; Valeant Canada, Montreal, Quebec), 10 mg/kg, q24h, PO. Over the course of hospitalization, the gelding responded well to treatment: the fever, tachycardia, dyspnea and tachypnea resolved, leukocytosis decreased to 14.0 × 109/L, and neutrophilia decreased to 11.2 × 109/L. The horse was discharged to the care of the owner on day 8 to continue treatment at home with erythromycin and rifampin at the same dose and frequency.

The gelding continued to improve at home for the 1st wk after discharge, but the animal’s condition began to deteriorate thereafter. Antimicrobial therapy was changed to...
chloramphenicol (Chlor 1000; Vetoquinol N-A, Lavaltrie, Quebec), 50 mg/kg, PO, q8h, on approximately day 30, as per the recommendation of the referring veterinarian following consultation with an internist. Treatment with flunixin meglumine was reinstituted as needed. Salbutamol and fluticasone were also administered by inhalation at unspecified doses and frequency as per the referring veterinarian. No improvement was seen and the gelding became inappetent, depressed, intermittently febrile, and once again developed markedly increased respiratory effort and coughing.

The horse was presented to the OVC-TH for the 2nd time on day 41. At that time, the gelding was quiet, alert, in very poor body condition (body condition score 1/5) and appeared weak. The horse was tachycardic (heart rate 60 beats/min), mildly tachypneic (respiratory rate 20 breaths/min) and markedly dyspneic. No cardiac murmurs or arrhythmias were detected. Dried mucopurulent nasal discharge was present at both nares. Lung sounds were diffusely increased and harsh on auscultation, and crackles and wheezes were audible over the caudoventral border of the lung bilaterally on expiration. Palpation of the thoracic wall bilaterally, especially on the right. On thoracic ultrasonography, marked pleural roughening was detected over most of both lungs, and several 2 to 3 cm areas of consolidated lung were noted bilaterally in the mid-caudal lungs. Pleural effusion was not detected. Thoracic radiographs showed significant progression of the previously described diffuse peribronchial interstitial pattern on the radiographs from 5 wk earlier. Several alveolar opacities, approximately 2 to 3 cm in diameter, were also present in the caudodorsal and caudoventral lungs (Figure 1). Approximately 15 cm dorsal to the ventral border of the lungs and caudal to the cardiac silhouette, a focal area (5 to 7 cm diameter) of more defined soft tissue opacity was present, which was consistent with a lung abscess. The diffuse distribution of the lesions was more consistent with embolic pneumonia than bronchopneumonia.

Hemogram and serum biochemistry abnormalities on day 41 included anemia (red blood cell count 5.2 × 10^12/L, hematocrit 22 L/L), leukocytosis (42.7 × 10^9/L), neutrophilia (38.86 × 10^9/L), monocytosis (1.71 × 10^9/L, reference range: 0.1 to 0.8 × 10^9/L), hyperfibrinogenemia (4.6 g/L), hyperhaptoglobinemia (1.92 g/L, reference range: 0.1 to 1.7 g/L), hypoalbuminemia (18 g/L), and hyperglobulinemia (66 g/L).

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On necropsy, multifocal random abscesses and areas of dark red consolidation involving approximately 20% of the lung parenchyma bilaterally were present. The rest of the lung was meaty, wet, and failed to collapse. A 15-cm long thrombus was found within one of the main pulmonary arteries of the left lung. A thick-walled abscess was found within the liver, which extended into the adjacent hepatic vein. An adherent 5-cm thrombus was present within the hepatic vein at this site which extended into the caudal vena cava, obstructing approximately 75% of the vena cava lumen. The thrombus was yellow-brown in appearance and friable. The abscess content and the anterior aspect of the thrombus contained yellow-green, malodorous, suppurative debris. No significant lesions were found within the heart. Bacterial culture of the hepatic abscess yielded E. coli, Clostridium spp., and mixed anaerobes. Bacterial culture of the lung tissue yielded E. coli and mixed anaerobes. Histopathology results were consistent with gross observations on postmortem examination. The bacteriological findings and the random multifocal distribution (embolic pattern) of the lung abscesses suggest that the pulmonary lesions originated from septic emboli from the thrombus in the hepatic vein and vena cava.

**Discussion**

Lung abscesses have been reported in a variety of species (1–4) including humans (5,6). In humans, liver abscesses are most frequently associated with an underlying disease of the hepato-biliary tract, septic portal vein thrombosis, or inflammatory colonic disease; however, most bacterial liver abscesses in humans are idiopathic. In feedlot cattle, hepatic abscesses are usually part of the rumenitis-liver abscess complex (2). In these cases, an episode of rumenitis results in erosion of the ruminal epithelium, leading to formation of microabscesses in the rumen wall and bacterial translocation from the rumenal lumen into the portal circulation. The bacteria thereby reach the liver where, if they are not efficiently eliminated by the immune system, they can cause infection and abscessation of the hepatic parenchyma (2,7). Cattle are predisposed to the condition by sudden dietary changes and consumption of sharp feed particles. The bacteria most commonly involved in hepatic abscessation in cattle include Fusobacterium necrophorum, Arcanobacterium pyogenes, Actinomyces spp., Corynebacterium spp., Bacteroides spp., Clostridium spp, and various coliforms (2).

Thrombosis of the caudal vena cava is a well-known sequela to liver abscesses in cattle (8). These cases usually present with chronic weight loss, loss of appetite, and decreased ruminal motility (8). Embolic pneumonia secondary to vena cava thrombosis resulting from hepatic abscission in cattle has also been well-described, and the clinical syndrome is known as caudal vena cava thrombosis syndrome (2,9–11). This condition may be accompanied (usually terminally) by pulmonary hemorrhage, hemoptysis, and epistaxis, as a result of erosion of a pulmonary arterial abscess (formed secondary to a septic embolus) into a bronchus (2,8). Treatment of caudal vena cava thrombosis...
syndrome is typically ineffective, and the case fatality rate for this syndrome is usually 100% (9).

In horses, intra-abdominal abscesses are rare (12). Liver abscesses account for 12% to 50% of intra-abdominal abscesses in this species (12,13). Hepatic abscesses in horses have been associated with septic portal vein thrombosis, and with intestinal pathology such as pressure necrosis due to enteroliths, resulting in the formation of adhesions between the colon and liver (14). Sellon et al (1) described 3 cases of hepatic abscessation in horses, 2 of which were also associated with caudal vena cava thrombosis. In 1 horse it was suggested that intermittent bacteremia from a hepatic abscess, which may have developed secondary to inflammatory bowel disease, led to septic thrombosis of the caudal vena cava. In another horse, it was suggested that the hepatic abscess was a sequela of abdominal surgery for anterior enteritis, and that the abscess then caused thrombosis of the caudal vena cava, which subsequently led to septic arthritis. The 3rd horse in the case series was presumed to have developed an hepatic abscess following foreign body penetration from the colon. Thrombosis of the caudal vena cava without concurrent liver abscessation has been reported in 1 horse, in which traumatic duodenitis due to foreign body penetration lead to the formation of an abscess around the vena cava that ultimately broke into the lumen of the vein (15).

Hepatic abscesses in cattle can be diagnosed by transcutaneous ultrasonography, depending on their location within the parenchyma (2). Visualization of the caudal vena cava using ultrasonography is possible; however, visualizing a thrombus in the vena cava is not possible under most circumstances due to the typically cranial location of the thrombus (8,16). Visualization of such a thrombus has been described in rare cases (17). In some cases a diagnosis of caudal vena cava thrombosis was made based on the abnormal appearance of the wall of the vena cava using ultrasonography (8,16). Ultrasonographic diagnosis of hepatic abscesses in horses has also been described (18,19). However, in the case reported here, it is unlikely that the hepatic abscess could have been detected by transcutaneous ultrasonography due to its location near the hilus of the liver. To the authors’ knowledge, antemortem diagnosis of caudal vena cava thrombosis in a horse has not been described.

In the case reported here, serum biochemistry failed to show any significant changes in liver enzyme activity at either the 1st or 2nd presentation. Other reports of hepatic abscessation in cattle and horses show that abnormalities in liver indices on routine bloodwork in these cases are inconsistent and frequently mild. In the aforementioned case series by Sellon et al (1), a horse with a $23 \times 12 \times 2$ cm liver abscess had normal liver indices. Another horse with several large liver abscesses affecting approximately 50% of the hepatic parenchyma had only slightly increased total bilirubin, ammonia, and sorbitol dehydrogenase (SDH) activity. The 3rd horse in the report had slightly elevated ammonia, SDH activity, and gamma-glutamyltransferase (GGT) activity, but there was no comment regarding the extent of liver abscessation in that animal (1). In a study of 18 cattle with liver abscesses, only 5 had elevated GGT levels (7). In an experimental study, GGT activity was significantly higher in affected cattle after day 3 following experimental induction of liver abscesses (20); however, a clinical study showed no significant difference in GGT activity between cattle with and without liver abscesses (21). A wide variation of the GGT activity in cattle with liver abscesses was also observed in another clinical trial (22). The variation in hepatic indices in animals affected by liver abscesses may be due in part to animals presenting in different stages of disease (7). Serum liver enzyme activity increases when there is ongoing hepatocellular necrosis and/or cholestasis, which is more likely to be present in the early stages of abscess formation within the liver or with a poorly encapsulated abscess. In the chronic stages, if the abscess is well walled-off from the surrounding hepatic parenchyma, there may be very little ongoing damage to hepatic cells and therefore serum liver enzyme activities may remain within normal limits despite significant liver pathology. Hepatic abscessation, with or without involvement of the caudal vena cava, should not be ruled out based on normal serum enzyme activities.

Based on the mixed population of bacteria found in the liver abscess and the limited number of other lesions, it is believed that the most likely etiology of hepatic abscessation in the case described here was bacterial translocation from the gastrointestinal tract via the portal circulation. There was no history or evidence of prior abdominal surgery, traumatic duodenitis, or inflammatory bowel disease. Subsequent thrombosis of the caudal vena cava resulting in intermittent showering of the lungs with septic emboli would then account for the embolic pneumonia and poor long-term clinical response to antimicrobial therapy.

There are several possible explanations for the culture results of the original tracheal aspirate compared with the postmortem culture results of the lung abscesses. *Streptococcus equi* ssp. *zooepidemicus* is an upper respiratory tract commensal and opportunist which could have overgrown secondary to the ongoing lung disease caused by the embolic showering of the lungs. It is also possible that the horse had pneumonia due to *S. zooepidemicus* at the time of the first presentation, which may explain the initial positive response to treatment, and then subsequently developed the vena cava thrombosis-like syndrome.

Although embolic pneumonia secondary to hepatic abscessation and caudal vena cava thrombosis is well-described in cattle as caudal vena cava thrombosis syndrome, this syndrome has not previously been described in a horse. An embolic distribution of pulmonary abscesses in a horse without evidence of another primary focus of infection should alert clinicians to the possibility of vena cava thrombosis secondary to hepatic abscessation.

References