Case report

Pathological Findings of Cholelithiasis in Two Horses

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Abstract

Despite of being a relatively rare disease in horses, cholelithiasis is the most common cause of biliary obstruction in this species. Simultaneous intrahepatic and extrahepatic gallstones are the most frequent presentation. Usually, there are no clinical signs associated with this condition, although biliary obstruction could occur as a consequence. Two cases of cholelithiasis in horses, including gross and histopathological findings are described. In one of the cholelithiasis cases there was no association with clinical signs or cause of death, but in the other one, chronic loss of weight may have been caused by cholelithiasis.

Key Words: horse, biliary ducts, gallstones.

Introduction

Different from other domestic species, the hepatobiliary system in horses lacks a gall bladder. As a result, bile excretion in this species occurs via the common hepatic duct, formed by the convergence of the right and left hepatic ducts (8). This anatomical peculiarity results in functional consequences. Unlike domestic ruminants and carnivores, bile excretion is continuous in horses, as they cannot store bile (1). Cholelithiasis, a biliary system disease, despite of being commonly observed in human beings (10,11), is relatively rare in horses (12). Cholelithiasis has an estimated prevalence of 0.08% (7), and is the primary cause of biliary obstruction in horses (3). Two cases of horse cholelithiasis, including gross and histological findings, are described.

Case report

Case 1

A 2.5-year-old crossbred mare with history of sudden death in the stall was received for postmortem examination. In the stomach, there was a 25.0 cm laceration, with 5.0 cm complete perforation in the greater curvature. Furthermore, biliary ducts in the liver had a moderate thickening wall and intense luminal distension due to accumulation of an intense amount of granular bile and several moderately dry friable yellow-orange choleliths. The size of the choleliths ranged from 0.1 to 2.0 cm in diameter (Figure 1). The morphological diagnosis of intense large biliary ducts ectasia and cholelithiasis was confirmed. Histopathological evaluation was not performed in this case due to intense autolysis of the carcass.

Case 2

A 5-year-old Campolina horse was admitted to the veterinary hospital for treatment of chronic laminitis in all four limbs. The horse remained hospitalized for nearly three months, and had positive response to treatment, but showed progressive weight loss and difficulty in standing. Complete blood count parameters were within normal limits, except for hyperproteinemia (9.1 g/dl), hypoalbuminemia (1.9 g/dl) and hyperglobulinemia (7.2 g/dl). The horse died and, at necropsy, intrahepatic biliary ducts distension with several gallstones (Figure 2) and a

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larger gallstone in the common hepatic duct were found. All gallstones were greenish-brown, smooth and friable.

Thickening of the ducts wall were observed in both horses. Samples of kidneys, lungs, spleen, liver, and brain were obtained for histopathological analysis. Histologically, both kidneys had multifocal tubular mineralization, lungs had intense peribronchiolar smooth muscle hyperplasia, and spleen and brain were morphologically normal. The liver showed intense portal and periportal fibroplasia with interlobular fibrous binding, moderate biliary ducts hyperplasia, light lymphocytic infiltration around portal biliary ducts and moderate congestion. Isolated biliary ducts were dilated and their lumens were filled with cellular debris. Morphological diagnosis for the liver was severe bridging portal fibrosis with moderate biliary duct hyperplasia and ectasia.

Discussion

Cholelithiasis is classified in hepatoliths, if gallstones are intrahepatic, choleliths, if occurs in biliary ducts, and choledocholiths, if found in the common hepatic duct (7). The most frequent form of cholelithiasis in horses is the simultaneous formation of intrahepatic gallstones and choledocholiths (4). In the present report, only intrahepatic gallstones were detected in the first case, but in the second there were both intrahepatic and extrahepatic gallstones.

Although cholelithiasis usually affects horses older than 9 year, younger animals can also be affected (2). Predisposing factors, such as gender, obesity and pregnancy are important in human beings, but not in horses (7, 10). Both animals in this present report were 2.5 and 5-year-old, out of the age group most frequently affected (5,2).

The etiopathogenesis of cholelithiasis in horses is uncertain (3); however, it is believed to be closely related to the uroliths formation mechanism, when solid cellular debris or thick material act as a core for crystallization (2). Ascariasis, biliary stasis, ascendent bacterial infection (6), foreign bodies (3) and bile composition changes are considered predisposing factors (3, 6). *Salmonella* spp., *Escherichia coli*, *Aeromonas* spp., *Citrobacter* spp., and *Streptococcus* spp. group D infection were reported in horses with cholelithiasis (3).

Sometimes, cholelithiasis may be related to drug sensitive recurrent, abdominal pain (3, 5, 12), pyrexia and jaundice (3, 5). Leukocytosis, hyperbilirubinemia with higher direct bilirubin, alkaline phosphatase, aspartate, aminotransferase gamma-glutamyl tranferase, creatine phosphokinase, lactate dehydrogenase and fibrinogen parameters may be observed in cholelithiasis cases, as a result of biliary obstruction (6). The horse in case 1 did not show any cholelithiasis-related clinical signs, such as abdominal pain, icterus or pyrexia, and its death was not related to this disease, which was just an incidental necropsy finding (3, 5, 11, 12). In case 2 the horse showed progressive weight loss, which may a consequence of cholelithiasis and hepatic fibrosis. Usually, cholelithiasis in horses does not concur with any clinical signs. Probably, the leading reason for this is the fact that the most common form of this disease, intrahepatic cholelithiasis, does not cause any bile obstruction (11).

Intrahepatic biliary ducts were dilated in both cases, which is consistent with the findings of Gerros et al. (3), Van der Luer & Kroneman (12), Ryu et al. (6), Peek & Divers (4) and Santos et al (7). In case 2, severe portal fibrosis was observed upon histopathological examination, which suggests a chronic disease (7, 12), but no hepatic lobe atrophy was observed, unlike a previous report by Santos et al. (7). In neither of the cases, chemical analysis of the gallstones was performed; however, gallstones in horses are usually composed of bilirubin, cholesterol esters and biliary pigments, cholic and carboxylic acid esters, calcium phosphate and sodium taurodeoxycholate (3).

Hyperproteinemia, hypoalbuminemia and hyperglobulinemia were observed in case 2, in contrast to what is commonly described in horses with cholelithiasis.
(6). Chronic laminitis may justify hyperproteinemia and hypoalbuminemia (9), and the absence of other sera biochemical alterations may suggest a partial biliary duct obstruction (6). Biliary duct obstruction, that causes biliary fibrosis and even cirrhosis, are consequences of cholelithiasis (12). Hepatitis, hepatic encephalopathy (11, 5), coagulopathies, bacteremia (3), chronic weight loss and photosensitization (5) are less commonly observed.

Based on the pathological findings a diagnosis of cholelithiasis in two horses was made. The incidental finding of cholelithiasis in horses during postmortem examination is the most frequent scenario for this condition, as there is usually no clinical signs associated. Nevertheless, chronic weight loss, as observed in case 2 of the present report, despite of being a non-specific clinical presentation, might be caused by cholelithiasis in this horse.

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References