Acute Pancreatitis in a Horse – a Case Report

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ABSTRACT
This report presents a case of acute pancreatitis in a 30 year old local breed horse. The horse was diagnosed clinically with severe acute abdominal pain, distended small intestine, a left dorsal large colon displacement and large colon impaction. On post mortem examination pathological changes in the pancreas were observed without intestinal impaction. Histopathologically, the pancreatic lesions were diagnosed as acute pancreatitis with peripancreatic fat necrosis. In addition to these findings, multifocal necrotizing hepatitis was identified as well as a mild interstitial nephritis and tubular nephrosis. This case demonstrates the difficulty in making a clinical diagnosis of pancreatitis in a horse and the importance of a thorough macroscopic and histological evaluation of the pancreas in horses with a history of abdominal pain.

Keywords: Horse; Colic; Abdomen; Impaction; Pancreas; Pancreatitis; Hepatitis.

INTRODUCTION
Acute pancreatitis is rarely diagnosed in horses and the true prevalence of the disease is probably underestimated appearing less commonly than in other species (1, 2, 3). It appears to be much less commonly reported in horses than in other species (1). Both acute pancreatitis and chronic pancreatitis have been documented. Acute pancreatitis is usually associated with severe acute colic, often characterized as an acute small intestinal obstruction with significant reflux and peritonitis. Chronic pancreatitis tends to cause inappetance, weight loss, lethargy and mild recurrent signs of colic (1, 2). Previous studies have identified horses with gastric distention and/or rupture with acute pancreatitis (1, 3, 4, 5).

Pancreatitis has been reported to occur in adult horses although cases of pancreatitis have been described in foals at post mortem (4, 5, 6, 7). Ante-mortem diagnosis is difficult on the basis of clinical and laboratory findings. No specific diagnostic tests are available and although reference values for serum and peritoneal fluid amylase and lipase activities have been published, their diagnostic accuracy has not been established (4, 8). Furthermore, trypsin is produced in small amounts by the equine pancreas (4).

Clinical signs are non-specific; the most important are signs of colic, gastrointestinal reflux and shock (2-5). The abdominal pain originates from gastric distention, peritonitis and hemoperitoneum (9). Thus the lack of specific clinical pathological parameters and clinical signs makes the clinical diagnosis of equine pancreatitis a challenge in the live horse.

In dogs, cats and humans pancreatitis is associated with nutritional imbalance, abdominal trauma, hypercalemia, hyperlipidemia, drug induced, bacterial and viral infection, vascular impairment, cholecystitis, small intestinal obstruction and duodenal reflux (9, 10). Etiology in horses includes partial or complete destruction of the pancreatic duct (cholangiohepatitis and cholethiasis), migration of Strongylus equinus and Parascaris equorum, duodenitis, duodenal ulcers and possible vasculitis in foals and associated with other disorders, commonly those involving the gastrointestinal tract or liver (4, 10, 11). Migration of Strongylus equinus and Parascaris equorum to the pancreas can produce pancreatic tis-
sue destruction and extensive fibrosis (12). Histopathology of acute pancreatitis includes large numbers of neutrophils that infiltrate the pancreatic parenchyma and percolate between the intralobular septae and acini, the peripancreatic fat shows evidence of necrosis. Histopathology of chronic pancreatitis is indicated by marked fibrosis between acinar lobuli and surrounding the pancreatic ducts. Interstitial mononuclear cell infiltrate may be present (4).

This case study describes an adult horse with pancreatitis diagnosed at post mortem examination while clinically as a case of severe acute colic that did not respond to analgesic medication and required abdominal emergency surgery. The case illustrates the difficulty in the diagnosis of equine pancreatitis in the clinical situation and proposes the consideration of pancreatitis as a differential diagnosis under conditions of colic of unknown origin.

**CASE HISTORY**

**Clinical history**

A 30-year-old local breed horse with an acute abdomen was admitted to the Koret School of Veterinary Medicine - Veterinary Teaching Hospital (KSVM-VTH). On physical examination before referral, the horses demonstrated severe abdominal pain even after NSAID (flumixin meglumine) administration and had elevated heart rate (60 beats/minute). Nasogastric intubation was performed by the referring veterinarian and resulted in spontaneous reflux of approximately 25 L of fluid.

Rectal examination revealed swollen small intestines, large colon displacement and large colon impaction. Prior to referral the horse received butorphanol, medetomidine and xylazine. On arrival the horse was dehydrated (dry mucus membranes and decreased skin turgor). Heart rate was 72/minute. Passage of a nasogastric tube resulted in 4 liter reflux. Rectal examination at the hospital diagnosed a left dorsal displacement of the colon and large colon impaction.

The packed cell volume was 53% (Reference interval (RI) 32-52%), total solids were 8.6 g/dL (RI 5.3-7.9 g/dL), creatinine 4.54 mg/dL (RI 0.9-2.0mg/dL) and lactate 7.6 mmol/L (RI up to 2mmol/L). The horse was prepared for exploratory laparotomy, while showing continuous and unrelenting pain. It was therefore treated with xylazine (Vetmarket, Shoham, Israel) and butorphanol (Alvegesic, Dachra Veterinary products, Shropshire, UK). While preparing the horse, signs of shock were evident: mean arterial pressure of 20 mm Hg, weak pulse, disrupted ECG and cyanotic mucous membranes. Prior to induction after consulting with the owners, the horse was euthanized at the request of the owners due to the poor prognosis.

**Post mortem examination**

The horse was sent for a full post mortem examination. Post mortem examination revealed slightly collapsed lungs and fibrous tags on the abdominal surface of the diaphragm. The stomach contained sparse fluid content. The pancreas was edematous and with multiple white yellowish foci (Figure 1). The small intestine showed multifocal serosal hemorrhage for almost its entire length. The large intestine contained dry content without pathological lesions in the mucosa. The liver and kidney showed no macroscopic pathological changes. There was no evidence of an impaction.

**Histopathology**

Histopathological examination of the intestines showed engorgement of blood vessels and multifocal serosal hemorrhage of the small intestine with diffuse submucosal edema. In the pancreatic tissue, multifocal to diffuse infiltrations by inflammatory cell mainly neutrophils and histiocytes were present, as well areas of peripancreatic fat necrosis (Figure 2 and 3). In some areas intralesinal bacterial colonies could be seen (Figure 4). A few of the kidney’s tubules were distended and contained an amorphus eosinophilic material. Numerous interstitial...
foci of inflammatory mononuclear cells were evident in the parenchyma. Diffuse congestion was present mostly in cortex. The liver showed multifocal areas of necrosis and infiltrations with mainly neutrophils. Diffuse congestion was also present.

**DISCUSSION**

Pancreatitis in horses can be a primary condition or may be associated with other disorders, commonly those involving the gastrointestinal tract or liver (4). Pancreatitis may be under diagnosed owing to the presence of nonspecific abdominal pain.

In this case report, it is unclear and difficult to determine whether the horse suffered from primary pancreatitis or secondary pancreatitis associated with hepatitis. The case history (pain duration, other symptoms, appetite, etc.) is lacking and in addition, blood tests including complete blood count and chemistry are also absent. Laboratory abnormalities associated with acute pancreatitis include increased activities of serum (and sometimes peritoneal) amylase and lipase (6).

The histopathological finding of multifocal necrotizing hepatitis has many etiologies: Causes of hepatitis in horses include serum hepatitis, cholangiohepatitis and chronic active hepatitis with occasional cases of hematogenous bacterial hepatitis, abscesses, viral hepatitis, parasitism and chronic infiltrative inflammatory disease (7).

It has previously been hypothesized that horses with enteritis or strangulated small intestinal lesions may develop acute pancreatitis and hepatitis as a result of ascending influx of intestinal fluid through the pancreatic and bile duct, with subsequent activation of pancreatic enzymes (4, 6, 10, 11). Once activated, these enzymes are responsible for autodigestion of pancreatic tissue, resulting in necrosis of the acini and pancreatic islets with interstitial fat necrosis and necrotizing vasculitis. The release of pancreatic enzymes stimulates the production of inflammatory cytokines, which, in turn triggers an inflammatory cascade which leads to a systemic inflammatory response syndrome (SIRS), multiple organ dysfunction syndrome (MODS), shock and death (4, 8).

Large colon disorders such as volvulus and displace-
ments could cause reduced blood flow to the pancreas and induce pancreatitis (4). In this case report, the rectal examination diagnosed large colon displacement and obstruction, but this was not verified in post mortem examination (5). Similarly to previous cases, this horse had distended small intestines and large amount of gastric reflux before referral, signs that are sometimes encountered in acute cases of pancreatitis (1, 4, 5).

In conclusion, pancreatitis should be considered in horses with unexplained moderate to severe abdominal pain with or without gastric reflux. Although the ante-mortem diagnosis of acute pancreatitis in the horse is difficult, it should be included as a differential diagnosis. Results from this case study emphasize the importance of a pathological and histopathological evaluation of the pancreas in horses with a history of abdominal pain.

REFERENCES