A 9-year-old, 954.8-lb (434-kg) Arabian gelding presented to The University of Georgia Veterinary Teaching Hospital (VTH) with an 8- to 9-month history of chronic, intermittent weight loss and a 6-day history of progressively increasing liver enzymes. Five years previously, the gelding had an episode of colic attributed to left dorsal displacement (LDD) of the large colon that resolved with medical management. In the 12 months before presentation, the horse had lived with five other geldings on a predominantly coastal and fescue grass pasture. Within 6 months of presentation, dental flotation had been performed, and the gelding had been vaccinated against equine influenza virus, Eastern and Western encephalitis viruses, West Nile virus, equine herpesvirus-1, Streptococcus equi subsp equi, and tetanus. Three months before presentation, fecal flotation revealed a moderate number of strongyle eggs. The gelding was subsequently dewormed with a 5-day course of fenbendazole (10 mg/kg PO q24h) and a single dose of ivermectin (0.2 mg/kg PO), which resulted in appreciable weight gain. One month before presentation, the gelding was dewormed with pyrantel pamoate (6.6 mg/kg PO).

Eight days before presentation, the gelding again appeared to be losing weight, so it was brought in from pasture to a stall and fed alfalfa and orchard grass hay supplemented with 1 kg of a 10% protein sweet feed twice daily. The gelding ate well for the following 2 days but then experienced an episode of fever (103°F) and anorexia. The results of a complete blood count (CBC) were unremarkable. However, a biochemistry profile revealed mild hyperglycemia (148 mg/dL; normal: 60 to 125 mg/dL) and a mild increase in γ-glutamyl transferase (GGT) activity (82 U/L; normal: 1 to 35 U/L). The referring veterinarian administered phenylbutazone (2.2 mg/kg IV once) and procaine penicillin G (22,000 U/kg IM q12h for 5 days). The fever and anorexia resolved within 24 hours; the origin of the fever was not determined. Blood samples collected 4 days later revealed a normal CBC, but the biochemistry profile indicated a further increase in GGT activity (128 U/L) and a mild increase in alkaline phosphatase activity (279 U/L; normal: 50 to 250 U/L). Serum bile acid concentration was normal. Because of the progressive increase in liver enzyme activity, the gelding was referred to the VTH for further evaluation. No signs of colic and no changes in fecal output or consistency were observed during the 8 days before presentation.

On presentation to the VTH, the gelding was moderately thin (body condition score: 3/9) but bright, alert, and responsive. Physical examination findings were within normal limits except for mild hirsutism; icterus was not observed. A rebreathing examination revealed no abnormalities, and no parasites were observed on fecal flotation. CBC results were within normal limits. A biochemistry profile revealed a mild decrease in blood urea nitrogen concentration (12 mg/dL; normal: 13 to 29 mg/dL).
mild hyperproteinemia (7.8 g/dL; normal: 5.4 to 7.5 g/dL) with a high-normal albumin concentration (3.3 g/dL; normal: 2.2 to 3.4 g/dL), mild hyperglycemia (147 mg/dL; normal: 64 to 132 mg/dL), and increased GGT activity (147 U/L; normal: 3 to 23 U/L). Sorbitol dehydrogenase activity was also increased (14.5 U/L; normal: 1 to 8 U/L) but was considered clinically insignificant. Total bilirubin (0.99 mg/dL; normal: 0.1 to 3.7 mg/dL) and serum bile acid (5 µmol/L; normal: 0 to 15 µmol/L) concentrations were normal.

Rectal palpation revealed the presence of gas-filled large colon entrapped within the nephrosplenic space. Transabdominal ultrasonography of the left kidney was partially obscured by the presence of a gas-filled viscus (FIGURE 1). Transabdominal ultrasonography of the liver revealed rounded edges, but the margins of the liver did not extend beyond the costochondral junction (FIGURE 2). The hepatic parenchyma was subjectively normal in appearance, and no other abnormalities were noted on abdominal ultrasonography. Peritoneal fluid obtained via abdominocentesis was mildly contaminated with blood and had a normal nucleated cell count (1400 cells/µL; normal: <5000 cells/µL) and total protein concentration (<2 g/dL; normal: <2.5 g/dL). Transcutaneous liver biopsy was performed with ultrasound assistance to obtain samples for histopathology and culture.

Based on clinical, ultrasonographic, and laboratory findings, the primary diagnostic differential was chronic LDD of the large colon with a secondary increase in liver enzyme activity. The gelding was held off feed for 24 hours and allowed free-choice water. Repeat rectal examination on day 2 of hospitalization revealed persistence of the LDD but resolution of gas distention within the large colon. Transabdominal ultrasonography at the left flank and last intercostal space confirmed the presence of a gastrointestinal viscus that impeded imaging of the left kidney. Phenylephrine (46 µg/kg diluted in 1 L saline) was administered IV over 20 minutes, and the gelding was then trotted for 20 minutes in an attempt to free the colon from the nephrosplenic space. The following morning (day 3), rectal examination revealed resolution of the LDD. Feed was gradually reintroduced over the following 48 hours. Repeated rectal examinations remained within normal limits. Serum biochemistry analysis on day 5 of hospitalization revealed that GGT activity had decreased to 132 U/L and sorbitol dehydrogenase activity was within normal limits. Histopathology of the liver biopsy samples revealed diffuse, moderate hepatocellular vacuolation, consistent with anorexia; culture revealed the presence of an α-Streptococcus spp considered to be a contaminant.

The gelding remained comfortable during the 6 days of hospitalization. A biochemical profile repeated 6 weeks after discharge revealed further decrease in GGT activity (39 U/L).

Seven months after discharge, the horse had a normal appetite and fecal output and was in excellent body condition (6/9). GGT activity was 42 U/L, which was consistent with mild cholestasis.

**Discussion**

This case represents an unusual presentation of LDD of the large colon in that an increase in liver enzyme activity was the primary presenting complaint rather than signs of abdominal discomfort. The gelding had a history of chronic, intermittent weight loss but no change in fecal output or consistency. The cause of the chronic weight loss is unknown but may have been related to parasitism or feed competition among herd mates. We have speculated that the LDD was secondary to a mild bout of colitis, which resulted in fever, anorexia, gas distention of the colon, and subsequent displacement of the colon. On presentation to the VTH, diagnosis of LDD was made by palpation per rectum and supported by ultrasonographic examination. Based on the clinical findings, case progression, and rapid decrease in GGT activity after correction of the LDD, it seems most probable that the increase in GGT activity was associated with the colon displacement.

GGT is a membrane-bound enzyme localized on the luminal surface of bile canalicular membranes and ductular cells of a number of organs, including the kidneys and mammary glands. Although the enzyme is widespread, increases in plasma GGT activity are almost exclusively associated with liver disease. In many species, increases in GGT activity are a specific marker of cholestasis. However, in large animal species, GGT appears to be a more general indicator of hepatic disease. Increases in GGT activity are frequently accompanied by increases in the activity of enzymes indicating hepatocellular injury, making the distinction between cholestatic and hepatocellular disease in horses difficult. Consequently, increased GGT activity has been observed in horses with a wide variety of hepatic disorders, including cholelithiasis, hepatic abscess formation, cholangiohepatitis, hepatic lipidosis, amyloidosis, neoplasia, and hepatotoxicities. Mild increases in GGT activity have also been observed in horses with colon displacement, proximal enteritis, and, less commonly, other forms of gastrointestinal disease, such as strangulating small intestinal lesions. Both cholestatic and hepatocellular enzyme activities may be increased in horses with proximal enteritis. This may occur as a result of an inflammatory insult to the liver (either via the portal vein or biliary tract) or hypoxia subsequent to hypovolemia and decreased hepatic perfusion. Large colon displacement or volvulus may cause biliary stasis and reflux of

**Figure 1.** Ultrasonogram of the liver viewed from the right 10th intercostal space. Rounded hepatic edges are prominent. L = liver; LC = large colon.
GGT into the systemic circulation, presumably by mechanically obstructing the bile duct subsequent to tension placed on the hepatoduodenal ligament.14,16 However, right dorsal displacement of the large colon (RDD) has been more commonly implicated in this phenomenon than LDD. In a study by Gardner et al.,14 almost half (18 of 37) of horses with an RDD had an increase in GGT activity, while GGT activity was increased in only one of 48 horses that presented with an LDD. Of particular note is that, in contrast to the horse in the case reported here, all of the horses in the study14 presented with colic as the primary complaint. The horse in this report had no signs of hepatic dysfunction or disease aside from mild increases in GGT activity. Test results of hepatic function (i.e., serum bile acid and total bilirubin concentrations) were normal, and histopathology revealed only moderate hepatocellular vacuolation consistent with anorexia. Although focal or multifocal liver disease cannot be completely ruled out, the lack of significant histologic abnormalities and increases in hepatocellular enzyme activities support extrahepatic biliary stasis as the cause of the increase in GGT activity.17–19 Furthermore, liver enzyme activity decreased during the 6 weeks after resolution of the LDD and was only mildly increased 7 months after presentation. The persistence of mildly increased GGT activity may indicate underlying low-grade cholestatic disease, although in the absence of other clinical signs and pathologic changes related to liver dysfunction, its significance is difficult to determine.

LDD of the large colon is a relatively uncommon cause of abdominal pain in horses, constituting approximately 3% to 5% of surgical referral cases20–22; the recurrence rate of LDD is estimated to be 7.5% to 8.1%.23,24 The incidence of chronic LDD is unknown, although a previous report suggests that it may occur.25 This report25 describes a horse with a 36-hour history of colic but multiple fibrous adhesions between the nephrosplenic ligament and large colon, consistent with entrapment of several weeks’ duration. Horses with LDD of the large colon typically present with mild but progressive signs of colic as a result of continued gas distention of the displaced viscus. The horse in this case report did not demonstrate overt signs of abdominal pain despite the presence of moderate large colon distention. The reason that there were not more obvious signs of pain is unknown; perhaps the LDD caused a partial rather than a complete luminal obstruction.

**Conclusion**

LDD should be included in the differential diagnosis of horses with mild elevations in GGT activity, even if signs consistent with abdominal pain are absent. In these cases, rectal examination and transabdominal ultrasonography may be indicated to rule out LDD as the cause of increased GGT activity.