Cholelithiasis and cholecystitis in a dairy cow

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- Cholelithiasis is uncommon in cattle but should be a differential diagnosis in cattle with signs of abdominal pain and serum biochemical evidence of cholestasis.
- Cholelithiasis in cattle may be treated by manual cholelithotripsy, which would relieve the obstruction.

A 9-year-old Holstein cow was admitted to the large animal clinic because of colic and decreased milk production of 2 days' duration. The cow was 4 months into its lactation period and had been treated with flunixin meglumine and calcium gluconate solution without improvement. On examination, the cow was lethargic and intermittently displayed signs of abdominal pain. The cow was moderately dehydrated (Hct, 33%), febrile (40.1 C [104.2 F]), tachypneic (46 breaths/min), and had a normal heart rate (82 beats/min). The cow was voiding small amounts of yellowish diarrhea. A ping was detected in the right caudal abdominal quadrant. Abnormalities found on transrectal palpation were distended loops of small intestine caudally within the abdomen and cranially on the right against the body wall and a large viscus or fluctuant mass about the size of a volleyball. On the basis of the history, clinical signs, and transrectal palpation results, a tentative diagnosis of proximal small-bowel obstruction was made. It was not possible to identify the large fluctuant viscus because of its cranial location, but possible structures included a distended loop of the proximal portion of the small intestine, a right-displaced abomasum associated with or secondary to the presumed small-bowel obstruction, or a loop of the proximal portion of the colon.

Serum biochemical abnormalities included hypokalemia (2.4 mEq/L; reference range, 3.9 to 6.0 mEq/L), hypochloremia (79 mEq/L; reference range, 92 to 117 mEq/L), hypocalcemia (8.2 mg/dl; reference range, 8.3 to 10.4 mg/dl), hyperbicarbonatemia (47 mEq/L; reference range, 21 to 31 mEq/L), hypophosphatemia (3.9 mg/dl; reference range, 4.9 to 9.1 mg/dl), and hyperglycemia (130 mg/dl; reference range, 31 to 77 mg/dl). Total bilirubin concentration was 1.6 mg/dl (reference range, 0.1 to 0.5 mg/dl), with direct bilirubin (0.3 mg/dl; reference range, 0.0 to 0.2 mg/dl) and indirect bilirubin (1.3 mg/dl; reference range, 0.1 to 0.5 mg/dl) being high. Activities of aspartate transaminase (498 IU/L; reference range, 47 to 138 IU/L), sorbitol dehydrogenase (153.5 IU/L; reference range, 10 to 50 IU/L), alkaline phosphatase (171 IU/L; reference range, 23 to 78 IU/L), γ-glutamyltransferase (240 IU/L; reference range, 11 to 39 IU/L), and creatine kinase (444 IU/L; reference range, 77 to 225 IU/L) were high. A venous blood gas analysis revealed metabolic alkalosis (pH 7.55). A CBC was not performed.

On the basis of persistent signs of abdominal pain that were nonresponsive to analgesics and abnormal transrectal examination findings, a right-flank exploratory laparotomy was performed with the cow under general anesthesia. Ceftiofur sodium (2.2 mg/kg [1 mg/lb] of body weight, IV) was administered. The cow was given xylazine hydrochloride (15 mg, IV) followed by an infusion of glyceryl guaiacolate, IV, until muscle relaxation was achieved. Ketamine hydrochloride (1 g, IV) was then administered. Anesthesia was maintained with isoflurane.

Obstruction in the jejunum and ileum was not evident. The duodenum was mildly distended with fluid and, within the lumen, had numerous, palpable, small (approx 1 cm) masses, which felt like gravel. The abomasum was in its typical position against the ventral body wall. The gallbladder was large and thickened. The serosal surface of the gallbladder was diffusely hemorrhagic. Multiple 2- to 3-cm-diameter firm masses were palpable within the gallbladder. Cholecystocentesis was performed, and samples were submitted for cytologic examination and bacterial culture. The bile sample was also submitted for detection of *Dicrocoelium dendriticum*, a parasite endemic in the geographic area where the cow lived. Cholecystotomy was considered but not performed because of the difficulty in exteriorizing the gallbladder and risk of bile peritonitis. Choleliths were reduced by moderate digital pressure. Cholelithotripsy was continued until stones were small enough to pass through the cystic duct, common bile duct, and into the duodenum. The contents of the duodenum were then massaged distally into the jejunum. The abdominal incision was closed in a routine manner.

Postoperatively, the cow was treated by IV administration of physiologic saline (0.9% NaCl) solution supplemented with potassium chloride, procaine penicillin G (22,000 U/kg [10,000 U/lb], IM, q 12 h) and ceftiofur (2.2 mg/kg [1 mg/lb], IM, q 12 h). The cow resumed a normal appetite and voided feces. Two days after surgery, serum biochemical analyses were performed, and electrolytes, total bilirubin concentration (0.2 mg/dl), and activity of sorbitol dehydrogenase (46.9 IU/L) had returned to within reference ranges. Activities of aspartate transaminase (219 IU/L), alkaline phosphatase (135 IU/L), and γ-glutamyltransferase (177 IU/L) were decreased from preoperative values. Venous blood gas values also had returned to within the reference range.

Results of cytologic examination of the bile sample aspirated at surgery revealed a mixed bacterial population. Aerobic culture yielded a moderate growth of *Escherichia coli* that was susceptible to penicillin and ceftiofur. Bacterial culture results were determined, using broth dilution minimum inhibitory concentrations following Naccell's guidelines. Anaerobic culture yielded a moderate growth of *Clostridium perfringens*. *Dicrocoelium dendriticum* eggs were not found in the bile sample, but 2 *Strongyle* sp eggs were identified. Eggs were not detected in feces.

The cow's rectal temperature and respiratory rate were within reference ranges within 12 hours of surgery and remained within reference ranges for the duration of hospitalization. The cow was discharged on the third postoperative day, with recommendations to the owner to continue antibiotics for 1 month. The cow was considered clinically normal for 2 months after discharge, but

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then reportedly deteriorated, having signs of progressive abdominal distention believed by the referring veterinarian to be related to a vagal indigestion. The cow died 3 months after surgery. Reported signs of vagal indigestion in this cow could have been caused by adhesions, leading to obstruction of the small intestine or interference with proper function of the vagus nerve. Other causes include liver abscessation and reticulopericarditis. Unfortunately, a postmortem examination was not performed.

Cholelithiasis is a common cause of abdominal pain in human beings and, reportedly, in horses.[1-3] Cholelithiasis has been reported uncommonly in domestic ruminants,[4] and to the authors' knowledge, this is the first description of surgical treatment of cholelithiasis in a cow. A report[5] of choledocholithiasis in a dairy cow indicated evidence of abdominal pain, icterus, and diarrhea; IV treatment with dextrose, calcium, and magnesium was not effective, and the cow was euthanatized because of progressive icterus and diarrhea. A cholelith was partially obstructing the common bile duct and was believed to be responsible for clinical signs.

In the cow of this report, preoperative serum biochemical results suggested hepatic damage and cholestasis, although the cow was not icteric at the time of admission to the hospital. High γ-glutamyltransferase activity is indicative of cholestasis, cholangitis, or cholangiohepatitis in large animal species.[6] High sorbitol dehydrogenase activity in the cow of this report indicated active liver necrosis. However, rapid return to normal enzyme activity indicated that liver necrosis was not ongoing postoperatively. High aspartate aminotransferase and alkaline phosphatase activities are not liver specific results in cows; however, with other liver-specific enzyme activities high, they supported a diagnosis of liver damage and cholestasis. The gradual decline of these enzyme activities was an indicator that the damage was not a continuing process.[6] The high total bilirubin concentration was also supportive of a diagnosis of biliary stasis; however, the higher indirect bilirubin concentration was more supportive of a diagnosis of liver dysfunction. The rapid return to normal suggested that the process did not remain active.[6]

The biliary system preserves sterility by the flow of bile into the duodenum; however, bacteria can ascend into the biliary system secondary to obstruction.[7] Enteric bacteria, followed by anaerobic bacteria, are the most common species isolated from the biliary tree in human beings with brown pigment stones (brown pigment stones are characterized by a high content of bilirubin and low amount of cholesterol).[8] The pathogenesis of cholelithiasis in animal species is controversial, and hypothesized mechanisms include changes in bile composition, cholecystitis, ascariasis, foreign bodies, and ascending infection.[9] Bacteria are believed to be responsible for the formation of most choleliths in animals.[10] In a retrospective study[2] on horses with cholelithiasis, gram-negative organisms were the most common isolates, and bacteria within the biliary system were believed to be a nidus for stone formation. Cholelithiasis is considered rare in cattle and has been reported as a clinical problem only once.[11] The exact cause of cholelithiasis in cattle is uncertain. Unfortunately, a sample stone was not obtained for analysis from this cow. Therefore, it was not possible to determine whether choleliths were primary or secondary to the infection in the cow of this report. The growth of *E coli* and *C perfringens* in the bile sample from this cow was believed to represent reflux of bacteria from the duodenum. Antimicrobials were administered long term in an effort to resolve bacterial infection within the gallbladder.

A common cause of cholecystitis and cholelithiasis in cattle is believed to be liver flukes.[12] *Dicrocoelium dendriticum* is endemic within central New York, the area of the referring veterinarian's practice. Young flukes encyst within the duodenum of the final host and migrate upward into the biliary system.[13] Heavy infections with this parasite have led to obstruction of the biliary system; however, it is believed that most cases are self-limiting.[14] The cow in this report only had evidence of *Strongyle* sp eggs within the bile sample. These were believed to be the result of reflux of ingesta. Other causes of gallbladder obstruction in cows are tumors, fascioliasis, suppurative cholecystitis, and foreign bodies such as nails, sticks, and sand.[9,11,14]

Treatment of cholelithiasis in horses is focused on relief of the obstruction and antimicrobial administration.[2] Choledocholithotripsy has been performed in horses,[15] and cholelithotripsy was performed in the cow of this report to relieve biliary obstruction. This method was preferred to cholecystotomy to avoid the risk of bile peritonitis. Although the cow in this report underwent an exploratory laparotomy under general anesthesia, the procedure can be performed in a standing animal under local anesthesia through a right-flank laparotomy.

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