Case Report

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Hemorrhagic bile pleuritis and peritonitis secondary to traumatic common bile duct rupture, diaphragmatic tear, and rupture of the spleen in a dog

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Abstract

Objective – To describe the diagnosis and successful treatment of bile pleuritis and peritonitis secondary to traumatic rupture of the common bile duct and a diaphragmatic tear in a young dog.

Case Summary – A 1-year-old German Shepherd dog was referred for evaluation of vomiting and icterus 4 days after being hit by a car. Thoracic radiographs, thoracic and abdominal ultrasonographic examinations, thoraco- and abdominocentesis, and positive contrast celiogram indicated hemorrhagic pleuritis and peritonitis, left dorsal diaphragmatic tear, and rupture and infarct of the spleen. Surgical exploration of the abdomen confirmed these findings in addition to a circumferential tear of the common bile duct, leading to a diagnosis of hemorrhagic bile pleuritis and peritonitis. Aerobic and anaerobic bacterial culture of the abdominal fluid yielded no growth. Surgical correction of the traumatic injuries was achieved via common bile duct anastomosis, cholecystojejunostomy, repair of the diaphragm, and splenectomy. The dog developed postoperative signs consistent with aspiration pneumonia but was successfully treated and discharged from the hospital. Clinical signs and laboratory abnormalities resolved and the dog was alive and healthy 8 months after discharge.

New or Unique Information Provided – Bile pleuritis is rare in dogs and cats and is usually associated with penetrating, not blunt, abdominal trauma. Multiple organ injury in cases of traumatic bile duct rupture is uncommon; in this dog, rupture of the common bile duct was accompanied by rupture of the diaphragm and spleen.

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Introduction

Extrahepatic biliary tract rupture is an uncommon but well-documented occurrence in dogs, most often associated with traumatic injury.[1–4] Development of clinical signs is often delayed following the initial trauma, and animals often present to the emergency room days to weeks following the traumatic event with marked hyperbilirubinemia accompanied by nonspecific gastrointestinal signs, lethargy, or anorexia.[1–4] Concurrent bile pleuritis and peritonitis as a complication of extrahepatic biliary tract rupture in dogs is extremely rare and is usually associated with penetrating abdominal trauma, which results in compromise of both the biliary tree and the diaphragm. Reported causes of diaphragmatic compromise include gunshot wounds and iatrogenic diaphragmatic puncture associated with thoracocentesis or abdominal lavage system placement.[3,5–8] Diagnosis of biliary tract and diaphragmatic compromise can be difficult with the imaging modalities readily available in veterinary emergency rooms today, yet early detection of biliary tract rupture can be critical in optimizing corrective surgery. Comparison of bilirubin levels in pleural and abdominal effusions with patient serum values provides the most definitive mode of diagnosing bile pleuritis/peritonitis.

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Case Summary

A 1-year-old spayed female German Shepherd dog, weighing 26 kg, was referred to the emergency service

at Oradell Animal Hospital for diagnostic workup for vomiting and possible traumatic diaphragmatic hernia. The dog had been hit by a car 4 days earlier (day 0) at which time it was presented to the referring veterinarian (RDVM), initially nonambulatory, with pale, tacky mucous membranes and multiple superficial lacerations on its left tarsus and hind paw, left antebrachium, left front paw, and right elbow. Within 3 hours after the initial injury, the dog's nonambulatory status reportedly resolved. The RDVM was suspicious that a mild pneumothorax was present based on a single lateral thoracic radiograph but reported that the dog was eupneic that afternoon. The dog was hospitalized and treated by the RDVM for 3 days with single IV dexamethasone and metoclopramide injections, SC buprenorphine, procaine penicillin G, and cimetidine, and SC and IV lactated Ringer's solution. The RDVM suspected the presence of pleural effusion and a possible diaphragmatic hernia based on a repeat lateral thoracic radiograph obtained on day 2; no evidence of pneumothorax was noted. The dog was placed under general anesthesia that afternoon and the limb lacerations were surgically repaired. A CBC and serum chemistry profile were submitted and the dog was discharged from the hospital on day 2 with amoxicillin (19.2 mg/kg, PO, q 12 h) and instructions for the owners to take the dog to a specialty hospital for diagnostic workup of pleural effusion and possible diaphragmatic hernia.

Twenty-four hours later (day 3), the dog was presented to the RDVM a second time for evaluation of vomiting. The dog was visibly icteric and had pigmenturia. The RDVM performed an upper gastrointestinal barium series, which was negative for the presence of gastrointestinal viscera within the thoracic cavity. Results from the CBC (submitted day 2) indicated that dog had a normochromic, normocytic anemia (HCT, 27.6%; reference interval, 36–60%; hemoglobin, 97 g/L [9.7 g/dL]; reference interval, 121–203 g/L [12.1–20.3 g/dL], RBC, 4.19×10^6 cells/μL; reference interval, 4.8–9.3 $\times$ 10^6 cells/μL), and a leukocytosis (WBC, 27,600 cells/μL; reference interval, 4,000–15,500 cells/μL) consisting of a mature neutrophilia (23,184 cells/μL; reference interval, 2,060–10,600 cells/μL) and a monocytosis (1,656 cells/μL; reference interval, 0–840 cells/μL). Spherocytosis (1+) was noted. Serum chemistry from day 2 revealed an elevated BUN (17.1 mmol/L [48 mg/dL]; reference interval, 2.1–8.9 mmol/L [6–25 mg/dL]), normal creatinine (114.9 μmol/L [1.3 mg/dL]; reference interval 44.2–141.4 μmol/L [0.5–1.6 mg/dL]), hypoalbuminemia (24 g/L [2.4 g/dL]; reference interval 27–44 g/L [2.7–4.4 g/dL]), hyperbilirubinemia (10.3 μmol/L [0.6 mg/dL]; reference interval 1.7–5.1 μmol/L [0.1–0.3 mg/dL]), and elevations in serum alanine aminotransferase (ALT, 2,377 U/L; reference interval, 12–118 U/L), aspartate aminotransferase (AST, 1,493 U/L; reference interval, 15–66 U/L), and creatine phosphokinase (CPK, 15,186 U/L; reference interval, 59–895 U/L). The pet was discharged and the owners were advised to bring the dog to a referral hospital for further diagnostics and treatment.

At presentation to the emergency service at Oradell Animal Hospital (day 4) the dog was quiet, alert, and responsive with a respiratory rate of 36 breaths/min and rectal temperature of 39.3 °C (102.8 °F). Physical examination abnormalities included icteric sclera; pale pink/icteric, tacky mucous membranes; prolonged capillary refill time; a slight increase in respiratory effort; bilateral dull cranioventral lung sounds; tachycardia (140/min); weak femoral pulses; and decreased skin turgor. No heart murmur, cardiac arrhythmia, or jugular pulsation were present. The dog had multiple surgically repaired lacerations on the distal extremities and was ambulatory without lameness.

The dog was admitted to the hospital. An initial systolic blood pressure measured using an ultrasonic Doppler flow detector was 120 mm Hg and pulse oximetry on room air was 95–97%. Initial PCV and total plasma protein (TPP) were 41% and 56 g/L (5.6 g/dL), respectively (reference intervals 37–55% and 52–78 g/L [5.2–7.8 g/dL], respectively). The only abnormality present on an initial venous blood gas and electrolyte analysis was hypokalemia (3.5 mmol/L; reference interval, 3.6–5.5 mmol/L). Initial therapy was comprised of an isotonic crystalloid[a] bolus (1 L, 40 mL/kg, IV), ampicillin[b] (22 mg/kg, IV, q 8 h), enrofloxacin[c] (10 mg/kg, IV, q 24 h), and hydromorphone[d] (0.05–0.10 mg/kg, IV, q 4–6 h). Following the fluid bolus, IV fluid therapy was continued with addition of potassium chloride[e] (20 mmol/L) and vitamin B_{12} complex[f] (2 mL/L) supplementation.

An in-house CBC confirmed a mild normochromic, normocytic anemia (HCT, 35.2%; reference interval, 37–55%, RBC, 5.42×10^6 cells/μL; reference interval, 5.5–8.5 $\times$ 10^6 cells/μL). Leukocytosis and mature neutrophilia were still present (WBC, 18,000 cells/μL; reference interval, 6,000–17,000 cells/μL; neutrophils, 15,560 cells/μL; reference interval, 3,000–11,800 cells/μL). Spherocytosis, RBC polychromasia, and nucleated RBCs were noted. In-house serum chemistry revealed marked hyperbilirubinemia (193 μmol/L [11.3 mg/dL]; reference interval, 0–15 μmol/L [0.0–0.9 mg/dL]). Alkaline phosphatase was elevated (ALP, 517 U/L; reference interval, 23–212 U/L) and ALT was decreased compared with the initial value but still elevated. The dog was also hyperphosphatemic (2.5 mmol/L [7.7 mg/dL]; reference interval, 0.8–2.2 mmol/L [2.5–6.8 mg/dL]) and BUN was elevated (11.1 mmol/L [31 mg/dL]; reference interval, 2.5–9.6 mmol/L [7–27 mg/dL]). The dog's albumin was within normal limits. An in-house coagulation profile (prothrombin time/activated partial thromboplastin time)

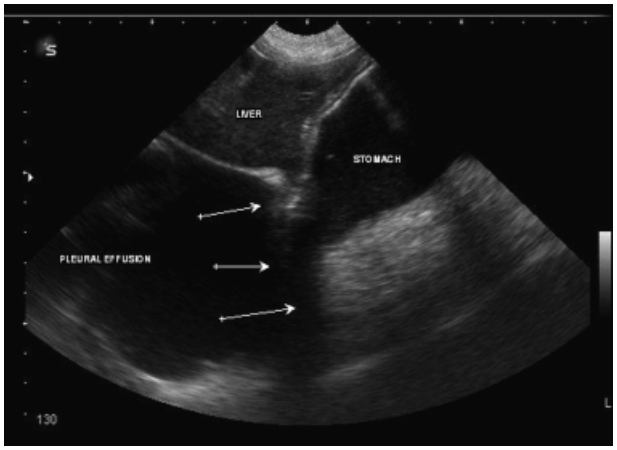

Figure 1: (Diaphragmatic tear) B-mode image of the left cranial abdomen demonstrating the fundus of the stomach filled with mixed echogenicity fluid, bulging cranially through a defect (arrows) in the dorsal aspect of the left side of the diaphragm. Pleural effusion is visible in the adjacent caudal dorsal pleural cavity.

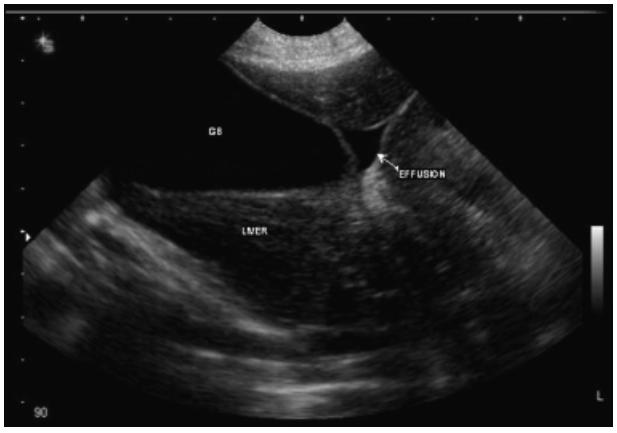

Figure 2: B-mode ultrasound image demonstrating the full, normal appearing gall bladder and peritoneal effusion between adjacent liver lobes.

was within normal limits. Abnormal findings on a urinalysis were significant hematuria, bilirubinuria, and proteinuria with amorphous urate crystals.

Emergency room ultrasonographic examination confirmed the presence of bilateral pleural and abdominal effusions. Survey ventrodorsal and right lateral thoracic radiographs confirmed that a small volume of bilateral pleural effusion was present. In addition, a mild, diffuse, interstitial pulmonary pattern and an irregular cranial border of the dorsal diaphragm were also present. An ultrasonographic examination of the abdomen was performed by a board-certified radiologist and revealed an irregularly shaped spleen with a poorly perfused tail and mottled echogenicity, consistent with traumatic splenic rupture and partial infarction. The gastric fundus and left side of the liver appeared to be displaced cranially, and there appeared to be a tear in the left dorsal lateral aspect of the diaphragm (Figure 1). Gastric stasis and moderate abdominal effusion were also noted. The gall bladder was moderately distended with a normal wall thickness (Figure 2). There was no evidence of biliary duct dilation or discontinuity. Abdominocentesis yielded a dark sanguinous effusion.

Left-sided thoracocentesis yielded 700 mL of dark, sanguinous fluid, similar in appearance to the abdominal fluid. In-house cytologic examination of the effusions indicated the presence of marked numbers of erythrocytes and degenerate neutrophils, with occasional macrophages. No intracellular or extracellular bacteria were identified. A positive contrast celiogram was performed using a 22-Ga needle to administer 30 mL (1.2 mg/kg) of sterile diatrizoate sodium[g] iodinated contrast medium intraperitoneally. A left lateral thoracic radiograph was taken 2–3 minutes postinjection. The postinjection radiograph showed a mildly increased opacity of pleural effusion, but was not definitive for compromise of the diaphragm (Figure 3).

The history, clinical and laboratory abnormalities, and imaging findings were suggestive of traumatic dorsal diaphragmatic tear and splenic rupture resulting in bicavitary hemorhagic effusion. The cause for the dog's hyperbilirubinemia and bilirubinuria was not

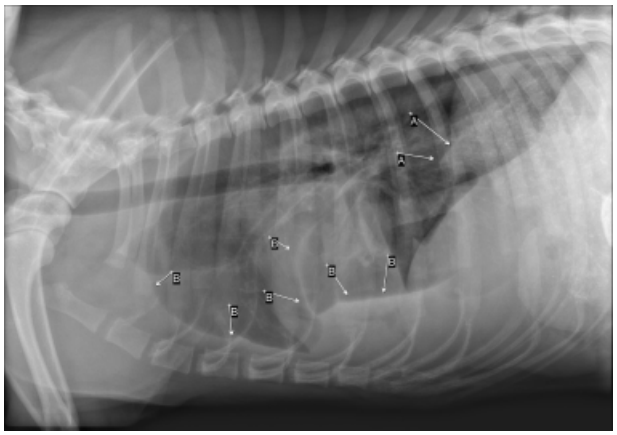

Figure 3: Left lateral thoracic radiograph obtained 2–3 minutes post–ultrasound-guided injection of 30 mL ionic sterile iodinated contrast into the cranial peritoneal cavity. The pleural fissure lines (arrows labeled B) are mildly increased in opacity, suggesting probable movement of contrast through a diaphragmatic tear and mixture with pleural fluid. Arrows labeled A point out an irregular diaphragmatic contour, suggesting possible disruption of the diaphragm.

clear, and the mild degree of hemolysis did not seem to account for the degree of hyperbilirubinemia. Abdominal exploratory surgery was recommended, based on a high index of suspicion for diaphragmatic tear and the need to better assess the liver and biliary tract for evidence of insult (rupture, torsion, strangulation, laceration, and infarction).

An exploratory laparotomy was performed on day 4 postinjury. The patient was premedicated with hydromorphone (0.05 mg/kg, IV) and midazolam[h] (0.3 mg/kg, IV), and anesthesia was induced with propofol[i] (3 mg/kg, IV, given to effect). Anesthesia was maintained with isoflurane in 100% oxygen using positive-pressure ventilation. Intraoperative monitoring consisted of serial blood pressure assessment (ultrasonic Doppler flow monitoring), pulse oximetry, and continuous electrocardiography in addition to monitoring of vital signs. IV crystalloid fluids were administered throughout the procedure (20 mL/kg/h during the first hour and 10 mL/kg/h every hour thereafter). No major anesthetic complications were encountered and the patient remained stable under anesthesia throughout the procedure.

At surgery, a completely avulsed distal common bile duct was identified in addition to a ruptured spleen with an infarcted tail segment and a large amount of bile-stained hemorrhagic abdominal fluid. A 5-cm radial tear was identified in the left dorsal diaphragm and bile-stained hemorrhagic fluid was identified within the thoracic cavity as well. No abdominal viscera were herniating into the thoracic cavity at the time of surgery. Abdominal fluid was sampled and submitted for aerobic and anaerobic bacterial culture and susceptibility. A common bile duct anastomosis and cholecystojejunostomy were performed to reestablish patency of biliary flow. A diaphragmatic herniorraphy was performed to repair the left dorsal diaphragmatic tear. A routine splenectomy and copious peritoneal lavage were performed before closure. The peritonitis was considered mild, and an abdominal drainage system was not placed. Postoperative care included continuation of IV crystalloid solution, hydromorphone (0.05–0.10 mg/kg, IV, q 4–6 h), metoclopramide[j] (1 mg/kg/d, IV, CRI), famotidine[k] (0.5 mg/kg, IV, q 24 h), metronidazole[l] (10 mg/kg, IV, q 12 h, initiated because of development of hematochezia during recovery), ampicillin (22 mg/kg, IV, q 8 h), and enrofloxacin (10 mg/kg, IV, q 24 h). IV colloid therapy[m] was initiated when hypoproteinemia was detected on a postoperative PCV/TPP (28%/38 g/L [3.8 g/dL]).

The dog developed bilateral purulent nasal discharge within 12 hours postoperatively and infrequent vomiting and regurgitation began in the initial 12–24-hour postoperative period. Dolasetron[n] (1 mg/kg, IV, q 24 h) and sucralfate[o] (500 mg, PO, q 6 h) were added to the treatment protocol and buprenorphine[p] (0.01 mg/kg, IV, q 6–8 h) was substituted for hydromorphone. The dog was otherwise bright, alert, afebrile, stable, and eating (small, intermittent feedings of a commercially formulated gastrointestinal veterinary diet[q]) and drinking voluntarily. Intermittent vomiting and regurgitation continued on day 6 (2 days after surgery) and the dog became partially inappetant. A recheck CBC on day 6 revealed a worsened leukocytosis with a regenerative left shift (WBC, 32,000 cells/μL; reference interval, 6,400–15,500 cells/μL; neutrophils, 24,960 cells/μL; reference interval, 3,000–11,800 cells/μL; band neutrophils, 960 cells/μL; reference interval, 0–300 cells/μL), and monocytosis (3,200 cells/μL; reference interval, 0–840 cells/μL), and confirmed a regenerative anemia (reticulocytes 404,840 cells/μL; reference interval, 0–60,000 cells/μL; 11.6%; reference interval, 0–1%, corrected 6.3%). Serum chemistry on day 6 indicated a marked improvement in total bilirubin (29 μmol/L [1.7 mg/dL]; reference interval, 0–5.1 μmol/L [0.0–0.3 mg/dL]), ALP (310 U/L; reference interval, 5–131 U/L), ALT (359 U/L; reference interval, 12–118 U/L), AST (115 U/L; reference interval, 15–66 U/L), and CPK (722 U/L; reference interval, 59–895 U/L). The patient's hyperphosphatemia and elevated serum urea nitrogen had resolved, but hypoalbuminemia had returned (16 g/L [1.6 g/dL]; reference interval 27–44 g/L [2.7–4.4 g/dL]). During the 24-hour period of partial inappetance the dog's vomiting and regurgitation decreased in frequency, and the dog was syringe fed one-quarter of its resting caloric energy requirement (via a low-fat adult veterinary diet[r]) divided into 5 small feedings, which were well tolerated. By the morning of day 7 the dog's appetite had returned completely, and syringe feeding was discontinued; the dog's caloric intake was gradually increased to meet estimated caloric requirements by day 8.

Ultrasonographic examination of the thorax and abdomen on day 7 (3 days after surgery) found only a small volume of thoracic and abdominal effusion and a hyperechoic appearance of mesenteric tissues, consistent with resolving pleuritis and peritonitis. Thoracic radiographs were repeated to evaluate the lungs for evidence of aspiration pneumonia, as well as to rule out megaesophagus or other cause of regurgitation. A focal alveolar pulmonary pattern was present in the the ventral right middle lung lobe. Differentials included aspiration pneumonia or pneumonitis, less likely pulmonary contusions, pulmonary edema secondary to fluid therapy volume overload, and acute lung injury/acute respiratory distress syndrome. Further diagnostic testing was not permitted by the owner and the dog was treated presumptively for aspiration pneu-

monia with the addition of saline nebulization and coupage to the broad-spectrum antimicrobial therapy. The patient's respiratory parameters and pulse oximetry readings on room air remained within normal limits. Nasal discharge and all vomiting and regurgitation had resolved by the morning of day 8.

No growth resulted from the aerobic and anaerobic bacterial cultures of the abdominal effusion at surgery. Financial limitations of the owner and continued clinical improvement prompted discharge of the patient from the hospital the morning of day 9 (5 days after surgery). The patient was discharged on oral amoxicillin[s] (19.2 mg/kg, PO, q 8 h), enrofloxacin (7.8 mg/kg, PO, q 24 h), and metoclopramide (0.3 mg/kg, PO, q 8 h × 2 days, then q 12 h × 3 days). A CBC and serum chemistry performed 2 weeks after surgery revealed complete resolution of all laboratory abnormalities except a mild hypoalbuminemia (24 g/L [2.4 g/dL]) and hyperbilirubinemia (6.8 μmol/L [0.4 mg/dL]). During a follow-up phone call 8 months after surgery, the owner reported that the dog was doing well at home and had had no further complications.

Discussion

Bile pleuritis in dogs has been infrequently reported in the veterinary literature.[3,5–8] In 2 of the 5 reported cases, penetrating gunshot wounds were the cause of diaphragmatic compromise, and in 2 others puncture of the diaphragm was iatrogenic in nature (secondary to thoracocentesis, secondary to placement of an abdominal lavage system).[5–8] In the fifth case, analysis of pleural fluid from a dog with concurrent bile peritonitis and pleural effusion after being hit by a car revealed an elevated bilirubin content, but the authors do not mention of any diaphragmatic compromise identified at surgery.[3] To the author's knowledge, the case presented here is the first reported case of bile pleuritis secondary to blunt trauma in a dog in which the site of communication between the thoracic and abdominal cavities was definitively identified.

Extrahepatic biliary tract rupture can result from abdominal trauma or obstruction (secondary to cholelithiasis, cholecystitis, neoplasia, or parasites), and is generally divided into 2 anatomical categories: gall bladder rupture and rupture of the bile duct, which includes the cystic duct, hepatic ducts, and common bile duct.[9,10] Gall bladder rupture is more commonly associated with necrotizing cholecystitis or cholelithiasis (with or without obstruction) while bile duct rupture is more commonly associated with trauma.[1,3,4] Rupture of the bile duct is generally attributed to 3 possible factors: a short cystic duct allowing for rapid gall bladder emptying; sufficient force applied to the gall bladder to empty it rapidly; and a simultaneous shearing force applied to the duct.[1] The common bile duct is the most common site of ductal rupture.[1,9] Concurrent injury to other abdominal structures is usually not reported with bile duct rupture secondary to trauma.[10] Development of clinical signs is often delayed days to weeks following trauma, and signs are usually nonspecific including vomiting, diarrhea, lethargy, anorexia, and weight loss.[2–4,10] Icterus; abdominal distension, pain, or both; and fever are the most common clinical signs noted while clinicopathologic findings often include anemia, leukocytosis, neutrophilia, elevated liver enzymes (ALT, ALP), hyperbilirubinemia, hypoalbuminemia, hyponatremia, hypokalemia, and hypochloremia.[2–4] With the exception of electrolyte abnormalities, the presentation and findings in this case were consistent with those previously reported.

Ultrasonography is a useful diagnostic tool for evaluation of the gall bladder and biliary tract in humans and small animals, particularly for assessing cholelithiasis, gall bladder sludge and wall thickening, and extrahepatic biliary tract distension.[9–15] It is much less definitive, however, in its ability to determine the cause of these abnormalities, assess for bile leakage, and to identify defects in the integrity of the bile duct.[15–17] In humans, computed tomography (CT), nuclear imaging, and magnetic resonance imaging are the modalities of choice for diagnosis of bile duct rupture.[16] Access to emergency CT and magnetic resonance imaging technology in veterinary medicine is limited at this time and requires that the patient be placed under general anesthesia or heavy sedation, which is not often practical or safe in many veterinary trauma patients. Definitive diagnosis of common bile duct rupture in this case was made at surgery. No ultrasonographic findings were present to suggest compromise of the integrity of the biliary tract in this case before surgery and it is notable the gall bladder appeared distended at the time of the ultrasound exam (Figure 2).

Elevation of bilirubin levels within abdominal (and pleural) effusions compared with serum levels is diagnostic of bile leakage into the abdomen in dogs.[3] Sufficient evidence existed for abdominal exploratory surgery in the case presented here; however, comparison of peritoneal and pleural effusion bilirubin concentrations with serum levels likely would have provided additional support for the diagnosis of biliary tract leakage. Paired samples should be considered in cases where biliary tract damage may be present.

Early diagnosis of bile duct rupture is imperative, as tissue necrosis and adhesion formation can complicate surgical repair.[9,10] Surgical options for treatment of a ruptured common bile duct involve primary bile duct

repair or biliary diversion.[9,10] Primary repair is technically challenging, associated with higher complication rates, and should only be attempted if ductal tissue appears healthy and the repair is being performed by an experienced surgeon.[10] Stenting of the duct following repair is sometimes performed. Options for biliary flow diversion include cholecystoduodenostomy, cholecystojejunostomy, choledochoduodenostomy, and cholecystojejunoduodenostomy (jejunal limb interposition). Adequate stoma size is important, regardless of which procedure is performed. Cholecystoduodenostomy is generally preferred if it can be accomplished as it maintains a more normal physiologic relationship between the biliary tract and intestinal tract.[10] Stricture formation following anastamosis of an avulsed bile duct is a significant postoperative risk, and concern for this complication following inspection of the anastomosis site is the reason a cholecystojejunostomy was performed in addition to the anastomosis in this case.

The prognosis in dogs with bile peritonitis varies, with reported mortality rates ranging from 28% to 64%.[3,4,14,18] Factors associated with decreased survival in dogs include septic effusions, marked leukocytosis, increased number of circulating band neutrophils, increased serum creatinine concentration, prolonged activated partial thromboplastin time, and postoperative hypotension.[3,4] One study suggested a higher survival rate with bile duct rupture as compared with gall bladder rupture.[4] Common postoperative complications include disseminated intravascular coagulation, anemia, hypoproteinemia, vomiting, edema, and sepsis. Aspiration pneumonia has been reported in multiple cases.[3,4]

The dog in this case was both anemic and hypoproteinemic after surgery, and developed regurgitation and aspiration pneumonia. The anemia (and to some extent, hypoproteinemia) was present before surgery; however, as the dog had significant intra-abdominal hemorrhage from splenic rupture; it was likely masked by the dog's relative dehydration at presentation to our hospital. The anemia was never severe and the dog never developed clinical signs (tachypnea, dyspnea, and tachycardia) secondary to it in this case, but transfusion of blood products in the form of packed RBCs or whole blood is indicated in cases of severe, life-threatening anemia secondary to traumatic intra-abdominal hemorrhage (splenic or other). Given the development of hypoalbuminemia after surgery, reassessment of the patient's coagulation parameters at that time would have been reasonable to determine if other blood products (plasma) may have been necessary; disseminated intravascular coagulopathy associated with systemic inflammatory response syndrome or sepsis is a concern when hepatobiliary injury or dysfunction is present.[4] Owing to the owner's financial constraints in light of the dog's stable hemodynamic (and overall) status this was not pursued. The etiology of the dog's recurrent postoperative hypoalbuminemia likely also involved a combination of gastrointestinal losses via vomiting and hematochezia and losses via residual effusion into the pleural and peritoneal space. As albumin is a negative acute-phase reactant, hepatic synthesis of this protein may have been impaired secondary to systemic inflammation from the peritonitis or pneumonia.

Whether the dog's aspiration pneumonia was truly a pre- or postoperative complication is not known as the dog was vomiting before admission, no known perioperative regurgitation occurred under anesthesia or during recovery, and by the time the dog's postoperative regurgitation began, nasal discharge had already been noted. It is arguable that the radiographic pulmonary pattern that developed was secondary to other etiologies, but given the history and clinical signs of the patient, and focal nature and specific location of the alveolar pattern within the right middle lung lobe, aspiration pneumonia seems most likely.

Diaphragmatic rupture secondary to blunt trauma is relatively common in small animals. Defects tend to be larger than those seen with penetrating trauma. In humans, left-sided tears are more common than right-sided tears, which is attributed to differences in strength of attachment (either inherent or via congenital defects), a protective effect exerted by the liver on the right side, and possibly underdiagnosis.[19] Left- and right-sided tears appear to be more evenly distributed in dogs and cats; young (1–3 years) male dogs are at higher risk.[20] Imaging diagnosis in humans usually involves thoracic radiography and CT.[19] Ultrasound has been shown to be useful in diagnosis of diaphragmatic rupture in humans, but CT is preferred and readily available in most human emergency facilities.[19,21] Radiography is the most useful test for diagnosis of diaphragmatic rupture in small animals.[20] Though less often utilized, ultrasound appears to be effective, with 1 study reporting accuracy as high as 93%.[13,20,22,23] Contrast radiography can be diagnostic but may be hindered by dilution of contrast medium by pleural or abdominal effusion.

Surgical repair is corrective and associated with relatively high survival rates, though some variability exists in the veterinary literature about the effect of the timing of surgical intervention on survival.[20,24] It has traditionally been accepted that surgical intervention for traumatic diaphragmatic hernia within the first 24 hours after injury is associated with greater complications (shock and multiorgan insult), and thus a higher mortality rate (33% reported).[20] The implied benefit in delaying surgery beyond the initial 24-hour period

seems likely to be related to better cardiovascular and respiratory stability and complete or partial resolution of metabolic derangements before surgery.[20] Recent data exist supporting much higher survival rates (94%) for patients operated within the initial 24-hour time period.[24] This discrepancy may be related to improvements in pre- and postoperative monitoring and veterinary care. Regardless, global assessment and stabilization of all organ systems and traumatic injuries before surgical intervention is ideal and likely to reduce the incidence of perioperative complications or secondary organ insult. This may not always be possible as some patients may face life-threatening hypoventilation secondary to compression of the lungs by abdominal viscera.[19] While this dog presented 4 days after trauma, she was not placed under anesthesia until all systemic parameters had been stabilized and any foreseeable complicating factors (ie, pleural effusion) had been addressed.

A different approach to aspects of the timing and management of diagnostic and therapeutic intervention from the time of initial trauma could have resulted in more rapid identification of the extent of underlying traumatic injuries in this patient and a more organized time frame for stabilization and surgical correction.

A more aggressive diagnostic workup based on the initial suspicion of thoraco-abdominal trauma likely would have led to earlier diagnosis of the traumatic injuries and surgical intervention, resulting in decreased morbidity, hospitalization time and cost. Even if the patient's hyperbilirubinemia had remained mild (as it was initially) and diagnosis of the diaphragmatic tear had been confirmed earlier, biliary peritonitis could have been identified via abdominocentesis and fluid analysis or on surgical exploration of the abdomen for the diaphragmatic tear; early repair of the avulsed bile duct (with potentially healthier tissue) might have occurred as a result.

The case presented here is an example of blunt abdominal trauma in a young dog leading to concurrent multiorgan injury including rupture of the common bile duct, diaphragmatic tear, and rupture of the spleen, ultimately resulting in bile pleuritis and peritonitis. It highlights the importance of complete assessment of all organ systems in the initial posttraumatic time period to identify not only obvious, immediate traumatic injuries but also to search for less overt sites of trauma that may be latent in presentation. Early identification and proper stabilization provide the best opportunity for intervention and improved morbidity and mortality. Surgical treatment of the traumatic injuries in this case was still corrective and led to resolution of clinical and laboratory abnormalities.

Footnotes

[a] Lactated Ringer's injection, Abbott Laboratories, Chicago, IL.
[b] Ampicillin inj., American Pharmaceutical Partners Inc, Schaumberg IL.
[c] Enrofloxacin, Bayer HealthCare LLC, Shawnee Mission, KS.
[d] Hydromorphone inj., Abbott Laboratories.
[e] Potassium chloride inj., Hospira Inc, Lake Forest, IL.
[f] Vitamin B_{12} complex inj., Vedco, St Joseph, MO.
[g] Hypaque sodium inj., Amersham Health Inc, Princeton, NJ.
[h] Versed, Roche Pharmaceuticals, Nutley, NJ.
[i] Propoflo, Abbott Laboratories.
[j] Metoclopramide (Reglan), Hospira Inc, Chicago, IL.
[k] Famotidine inj., Ben Venue Labs, Bedford, OH.
[l] Metronidazole inj., Baxter Healthcare Corp.
[m] Hetastarch, Abbott Laboratories.
[n] Dolastron inj. (Anzemet), Aventis Pharmaceuticals Inc, Kansas City, MO.
[o] Sucralfate, Carafate, Hoescht Marion Roussel, Kansas City, MO.
[p] Buprenorphine inj., Reckitt Benckiser Pharmaceuticals Inc, Richmond, VA.
[q] Hill's prescription diet i/d. Hills Pet Nutrition Inc, Topeka, KS.
[r] Royal Canin veterinary diet canine Low Fat LF, Royal Canin USA, St Charles, MO.

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