Case Report

Journal of Veterinary Emergency and Critical Care 20(6) 2010, pp 595–600
doi: 10.1111/j.1476-4431.2010.00582.x

Trauma-associated acute mesenteric ischemia in a dog

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Abstract

Objective – To describe the successful management of acute mesenteric ischemia (AMI) in a dog.
Case Summary – An 11-year-old, 21.4 kg, male castrated Rough-Coated Collie was referred for evaluation of an acute abdomen following sustaining injuries in a vehicular accident. On presentation to the hospital, clinicopathologic assessments were within normal limits and distended intestinal loops were evident on abdominal palpation. Plain and contrast radiography demonstrated diffusely distended gas-filled intestinal loops with a delayed barium transit time. Emergency surgical exploration was performed. The cecum and the proximal two-thirds of the colon were dark purple to black in color and there were no palpable pulses in the mesenteric arteries associated with the cecum and colon. The cecum and proximal two-thirds of the colon were resected and an ileocolic anastomosis was performed. The dog recovered well from surgery and was discharged 2 days later. Histopathologic examination was consistent with AMI.
New or Unique Information Provided – AMI is uncommonly reported in human medicine and is a rare complication from even minor blunt abdominal trauma. This is the first reported case of trauma-associated AMI in a dog.

(J Vet Emerg Crit Care 2010; 20(6): 595–600) doi: 10.1111/j.1476-4431.2010.00582.x

Keywords: Gastrointestinal injury, Gastrointestinal tract surgery

Introduction

Acute mesenteric ischemia (AMI) is reported in people and results from emboli, arterial or venous thrombi, or intestinal vasoconstriction due to hypovolemia.[1] Despite aggressive management, the mortality rates for human patients remain high.[2–6] Rapid diagnosis and intervention of AMI is critical for a successful outcome as prognosis for return of intestinal viability decreases precipitously with increasing time delay between the development of symptoms and diagnosis and intervention.[7]

AMI has been reported in dogs in experimentally induced settings,[8–10] but not as a naturally occurring disease. The purpose of this case report is to describe the successful management of a dog with trauma-associated AMI.

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The authors declare no conflicts of interest.

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Submitted September 18, 2009; Accepted August 23, 2010.

Case summary

An 11-year-old, 21.4 kg, castrated male Rough-Coated Collie was in the rear compartment of a van that was rear-ended by another vehicle. The dog sustained unknown trauma from the accident and was lethargic immediately after the incident but did not exhibit any other clinical signs. The following day the dog was anorexic and exhibited pelvic limb tremors. The dog was evaluated at an emergency hospital and back pain was suspected based on physical examination. The dog was administered an unknown dose of buprenorphine[a] and discharged from the hospital. The dog remained inappetent throughout the remainder of the day and presented to his primary care veterinarian the next day, approximately 36 hours after the accident. The veterinarian noted splinting and discomfort on abdominal palpation. A complete blood count and serum biochemistry panel was performed and demonstrated an increased creatine kinase at 415 U/L (reference interval, 10–200 U/L), hypercholesterolemia (9.9 mmol/L [377 mg/dL]; reference interval, 2.9–8.6 mmol/L [112–328 mg/dL]), and a leukocytosis at 21.1 $\times$ 10^9 cells/L (reference interval 5.7–16.3 $\times$ 10^9 cells/L) [21.1 $\times$ 10^3 cells/μL; reference interval, 5.7–16. $\times$ 10^3 cells/μL) characterized by

a mature neutrophilia of 18.4×10^9 cells/L (reference interval, 3.0–11.5 $\times 10^9$ cells/L) (18.4×10^3 cell/μL; reference interval 3.0–11.5 $\times 10^3$ cells/μL) and a monocytosis of 1.69×10^9 cells/L (reference interval, 0.15–1.35 $\times 10^9$ cells/L) (1.69×10^3 cells/μL; reference interval 0.15–1.35 $\times 10^3$ cells/μL). The neutrophils were noted to be slightly toxic. Diffuse, markedly distended gas-filled loops of intestine were noted on survey abdominal radiography, and microcardia with a small caudal vena cava was noted on survey thoracic radiographs (Figures 1 and 2). A positive contrast barium study was also performed and demonstrated a delayed transit time of barium through the gastrointestinal tract (Figure 3). The veterinarian suspected a mesenteric tear or a diaphragmatic hernia and administered dexamethasone[b] (0.93 mg/kg) and enrofloxacin[c] (3.18 mg/kg) IV and referred the dog for further diagnostics and treatment.

On presentation, the dog was tachycardic at 160/minute and had a body condition score of 2/9. Significant distension of intestinal loops was noted on abdominal palpation, but the remainder of the physical examination was within normal limits. The owners reported the dog's body condition had been unchanged for several years before presentation. The dog had received an incisional gastropexy for a gastric-dilatation and volvulus (GDV) 3 years before presentation.

The primary differential for the dog's condition was a mesenteric torsion due to the diffusely distended gas-filled loops of intestine. Because of the concern of ischemic bowel precipitating bacterial translocation and subsequent sepsis, coagulation testing[d] was performed and indicated a prothrombin time of 12 seconds (reference interval, 12–17 s), an activated partial thromboplastin time of 98 seconds (reference interval, 71–102 s). The dog had a PCV of 36% (reference interval, 35–55%) and a total plasma protein of 56 g/L (reference interval 60–75 g/L) (5.6 g/dL [reference interval, 6.0–7.5g/dL]). Systolic blood pressure on presentation was 180 mm Hg on Doppler measurement. The dog was suspected of being hypovolemic due to the microcardia and small caudal vena cava identified on thoracic radiographs and tachycardia present on presentation despite normal systolic blood pressure. Therefore, crystalloid fluid therapy[e] was initiated with a 1 L (46.7 mL/kg) bolus over 30 minutes followed by a infusion rate of 4 mL/kg/h and the dog's tachycardia resolved. Based on the

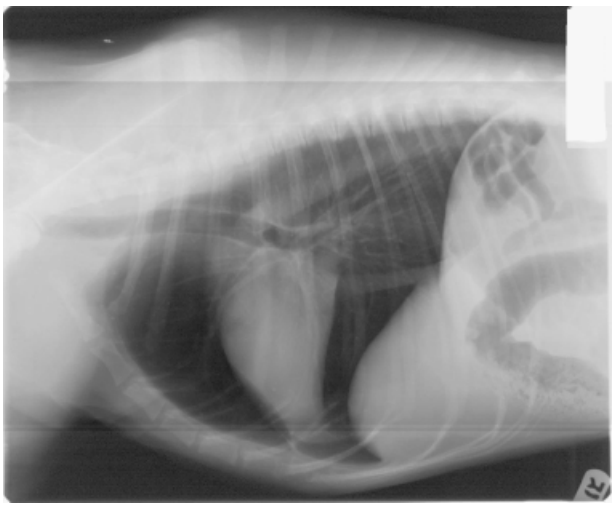

Figure 2: Survey lateral thoracic radiograph of dog taken 36 hours post trauma at referring veterinarian showing microcardia and a small caudal vena cava suggestive of hypovolemia.

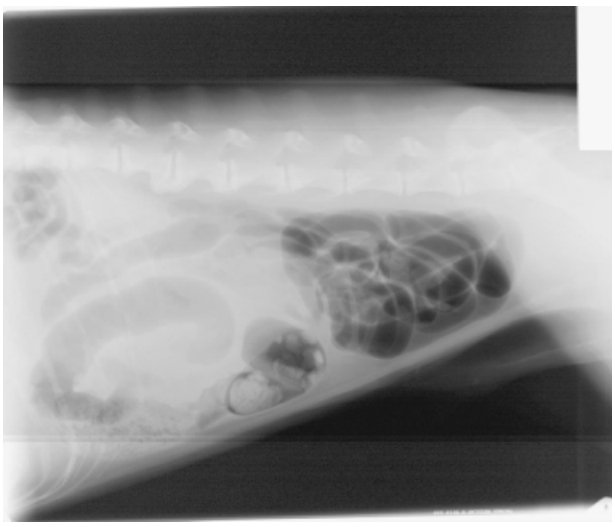

Figure 1: Survey lateral abdominal radiographs of dog taken 36 hours post suspected abdominal trauma at referring veterinarian.

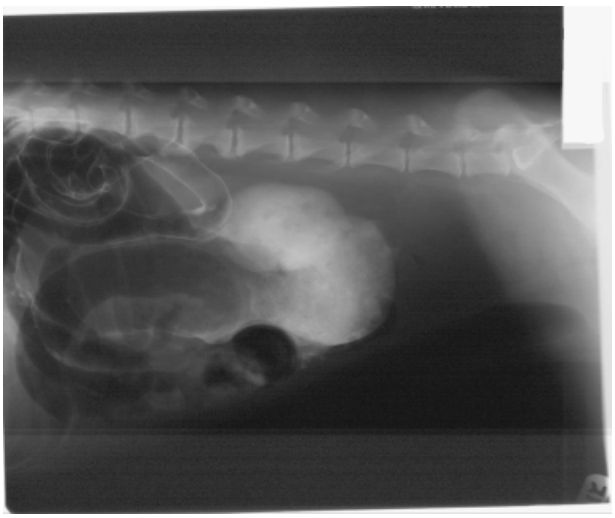

Figure 3: Lateral abdominal radiograph taken 8 hours post oral administration of positive constrast (barium sulfate) showing gas dilated loops of intestine and delayed barium transit time.

referral abdominal radiographs, surgical exploration was recommended.

The dog was pre-medicated with hydromorphone[f] (0.1 mg/kg IM) and glycopyrrolate[g] (0.01 mg/kg IM). General anesthesia was induced with diazepam[h] (0.45 mg/kg IV) and hydromorphone (0.2 mg/kg IV) and maintained with isoflurane in oxygen. The dog received perioperative ampicillin[i] (22 mg/kg IV) and was placed on crystalloid fluids[e] at a rate of 11 mL/kg/h.

A ventral midline exploratory laparotomy was performed and minimal abdominal effusion was noted. There was diffuse ileus throughout the intestinal tract and the cecum and colon were in appropriate orientation. However, the cecum and the proximal two-thirds of the colon were dark purple to black in color and there were no palpable pulses in the associated mesenteric arteries (Figure 4). The previous incisional gastropexy site was stretched but intact and the rest of the abdomen was unremarkable. To better visualize the cecum and colon, the gastropexy was broken down.

The cecum and proximal two-thirds of the colon were resected and an ileocolic anastomosis was performed using 3-0 polydioxanone suture[j] in a simple interrupted pattern. The resected cecum and colon were submitted for histopathological evaluation. The anastomosis site was leak tested and the gastropexy site was restored using an incisional technique. The abdomen was thoroughly lavaged with sterile saline[k] before routine closure.

The dog recovered without incident from surgery and was monitored in the critical care unit postoperatively. The dog was maintained on crystalloid fluid therapy[e] at a rate of 3.7 mL/kg/h and administered parenteral antibiotics including metronidazole[l] (10 mg/kg, IV, q 12 h), ampicillin[i] (23.8 mg/kg, IV, q 8 h), and enrofloxacin[c] (10 mg/kg, IV, q 24 h). Analgesia was provided with a 50 mcg transdermal fentanyl patch[m] and hydromorphone[f] (0.1 mg/kg, IM, q 6 h).

The following day the dog was offered small amounts of food and water and started on metronidazole[n] (11 mg/kg, PO, q 12 h), amoxicillin[o] (18 mg/kg, PO, q 8 h), ciprofloxacin[p] (6 mg/kg, PO, q 12 h), tramadol[q] (2 mg/kg, PO, q 12 h), and famotidine[r] (0.5 mg/kg, PO, q 12 h). The dog continued to improve and was discharged from the hospital 2 days later.

Histopathological evaluation of the resected cecum and the colon revealed moderate to marked hemorrhage throughout all layers of the cecal and colonic wall extending from the underlying muscularis to the serosa. Several poorly organized fibrin thrombi were observed within the mesenteric arteries. The morphology of the intestine was assessed to be consistent with a recent episode of infarction.

At recheck examination 2 weeks postdischarge the dog was doing well. The owners reported a transient 24-hour period of anorexia that resolved without further intervention or diagnostics. The dog was producing normal formed stool at that time.

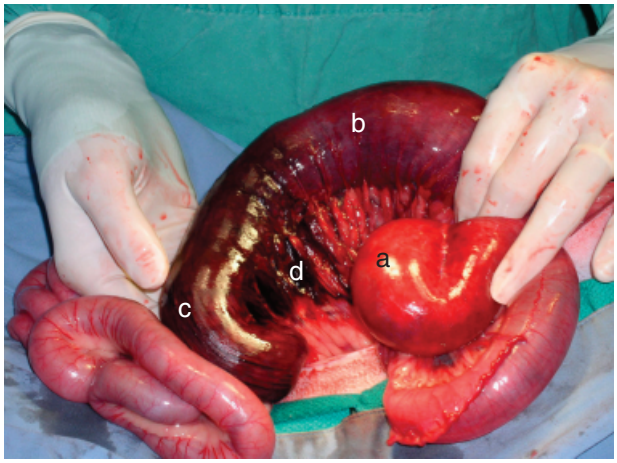

Figure 4: Intraoperative photograph showing thrombosed cecum (a) and ascending (b) and transverse (c) colon with some hemorrhage noted in mesenteric vessels (d). The dog's head is to the right.

Discussion

This is the first reported case of trauma-associated AMI in a dog. AMI has been reported to occur in people secondary to abdominal trauma, with the ileum and ascending colon being the most commonly affected locations. Even mild abdominal contusion may induce intestinal infarction without any other intra- or retroperitoneal lesion.[11,12]

The pathophysiology of AMI is poorly understood, but intense intestinal vasoconstriction during periods of shock is postulated to be the inciting event. With prolonged vasoconstriction, damage to the intestinal microcirculation leads to irreversible intestinal necrosis and activation of endothelial cells, leukocytes, and platelets. Activated neutrophils, endothelial cells, and platelets produce inflammatory cytokines precipitating the systemic inflammatory response syndrome and sometimes disseminated intravascular coagulation.[13]

Reperfusion injury is also critical to the pathophysiology of AMI with activated neutrophils in the reperfused intestine producing free radicals such as superoxide (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (OH^-) that damage cell membranes through lipid peroxidation.[14] Reperfusion injury also induces increased intestinal capillary permeability leading to interstitial edema and luminal fluid accumulation.[15] The loss of intestinal barrier integrity

facilitates bacterial translocation and the development of subsequent bacteremia and endotoxemia.[16–18] In human medicine it is suspected that the poor prognosis associated with AMI is related to the multiple organ failure induced secondary to bacterial translocation and sepsis.[19] The dog in this case report had evidence of hypovolemia based on thoracic radiographs and the fact that the tachycardia on presentation was responsive to fluid resuscitation. The hypovolemia may have stimulated the intestinal vasoconstriction necessary to initiate the development of AMI. In addition, the use of systemic corticosteroids had precipitated ischemic damage in the colon and may have further contributed to the development of the AMI.[20]

The primary symptom of AMI in people is sudden and severe abdominal pain disproportionate to physical findings, but this is not a consistent finding.[13] Bloodwork is typically nonspecific and there is no serum marker sensitive or specific enough to establish or exclude the diagnosis of AMI.[1] The dog in this case report demonstrated clinical signs of abdominal pain, anorexia and lethargy, and bloodwork abnormalities were nonspecific.

Diagnosis of AMI in human medicine is typically difficult as both radiographs and abdominal computed tomography have shown to have a poor ability to identify patients with confirmed intestinal infarction.[21] Radiographic signs of AMI include portal venous gas and pneumatosis intestinalis but are typically only seen late in the disease course and are associated with a high mortality rate.[1,22] None of those radiographic signs were identified in the dog reported here and surgical exploration was recommended based on clinical signs and the diffuse gas-filled loops of intestine. Selective mesenteric angiography is considered the gold standard for the diagnosis of AMI in people,[7,23] but is not available at our hospital, which precluded its use in the dog reported here.

Treatment of the dog in this case report was surgical resection of the ischemic bowel although surgical intervention is typically only recommended in humans with AMI when signs of peritonitis are present.[1] Intra-arterial perfusion with a thrombolytic agent, intra-arterial infusion of vasodilators and simple systemic anticoagulation have been reported in humans for management of an AMI.[24] Because of the severity of the AMI identified intraoperatively in the dog, the decision was made to perform surgical resection rather than attempt medical therapy.

The use of broad-spectrum antibiotics in the management of this case was due to the concern of bacterial translocation from the apparent devitalized intestine identified at surgery. In human medicine, most physicians recommend the use of antibiotics for management of colonic ischemia despite the lack of clinical evidence of beneficial effects of such therapy.[1] However, use of antibiotics improved survival after intestinal ischemia in rats[25] and has theoretic protection against bacterial translocation, which develops with the loss of mucosal integrity.[26,27] The benefit of antibiotics in the outcome of this case is unclear as they were continued for 2 weeks postdischarge and then discontinued.

Shock-induced colonic ischemia and necrosis is another rare condition that has been reported in human medicine.[28] All reported cases had documented hypotension with a systolic blood pressure <90 mm Hg, with the right colon the most commonly affected location.[28] Pathologic examination of resected specimens showed ischemic necrosis but no evidence of vascular thrombosis of mesenteric vessels.[28] The necrosis is proposed to develop secondary to intense adrenergic constriction of mesenteric vessels and shunting of blood away from the colonic mucosa during periods of hypotension.[28] Shock-induced colonic ischemia and necrosis appears unlikely in the dog reported here as there was no documented evidence of systemic hypotension following the trauma, although systemic blood pressures may not accurately reflect the perfusion state of individual organs such as the intestinal tract. Further, thrombosis of the mesenteric vessels was identified on histopathology, which is not a feature of shock-induced colonic ischemia and necrosis.

Though there was no evidence of a cecal-colic volvulus at surgery, we cannot rule out the possibility that this occurred early in the course of this dog's illness but resolved before surgical intervention. The dog in this report had a GDV approximately 3 years before presentation, and a weak and undefined relationship between GDV and both small intestinal and colonic volvulus has been reported.[29,30] However, we suspect a colonic volvulus of sufficient severity to precipitate thrombosis and necrosis of the cecum and colon would not spontaneously resolve without surgical intervention.

No testing was performed on the dog postoperatively to determine if there was an underlying cause of the dog's poor body condition. Intestinal biopsies at the time of surgical exploration would have been ideal, but were not performed given the presence of devitalized intestine and the concern of increasing morbidity and mortality with additional diagnostic biopsies. As such, we cannot infer any relationship between concurrent conditions such as gastrointestinal disease that may predispose dogs to the development of AMI.

Thromboelastography (TEG) is utilized in human medicine and select veterinary hospitals to evaluate clotting function more compherensively than traditional prothrombin time and activated partial thromboplastin time, and to identify animals with hypercoagulability.[31]

TEG has been reported to identify hypercoagulability in dogs with disseminated intravascular coagulation,[32] neoplasia,[33] and parvovirus.[34] TEG in the postoperative period would have been helpful to determine if the dog in this case had a preexisting hypercoagulable state which precipitated the AMI. However, the results would have been difficult to interpret due to the trauma[35] and corticosteroid[36,s] administration, which are known to precipitate a hypercoagulable state. No diseases that predispose to thrombus formation were identified on bloodwork, radiographs, surgical exploration, or biopsy. However, TEG is not available at our hospital, which precluded an evaluation in this case. Other assessments of hypercoagulability such as measurement of thrombin-antithrombin, fibrinogen, and D-dimers have been reported in other veterinary studies but were also not performed in this case.[37,38]

In conclusion, we suspect the AMI in the dog reported here developed from hypovolemia due to the vehicular accident 3 days before presentation. We theorize that the hypovolemia triggered compensatory intestinal vasoconstriction and subsequent reperfusion injury precipitated the AMI. AMI is a rare condition that should be considered in a dog that develops an acute abdomen after trauma. As in this case, however, with rapid diagnosis and intervention, complete recovery is possible.

Acknowledgements

The authors would like to thank Dr. Cassandra Carroll and Dr. Audra Fenimore for their assistance in the management of this case, and Dr. Emily Tompkins for her assistance in the preparation of this manuscript.

Footnotes

[a] Buprenorphine Hydrochloride Injection, Hospira Inc, Lake Forest, IL.
[b] Dexamethasone-SP, Bimeda-MTC Animal Health Inc, Cambridge, ON.
[c] Baytril, Bayer Healthcare LLC, Shawnee Mission, KS.
[d] SCA Veterinary Coagulation Analyzer, Synbiotics Corp, San Diego, CA.
[e] Normosol-R, Hospira Inc, Lake Forest, IL.
[f] Hydromorphone HCl Injection, Baxter Healthcare Corporation, Deerfield, IL.
[g] Glycopyrrolate Injection, American Regent Inc, Shirley, NY.
[h] Diazepam Injection, Hospira Inc.
[i] Ampicillin for Injection, G.C. Hanford Mfg Co, Syracuse, NY.
[j] 3-O PDS II, Ethicon Inc, Somerville, NJ.
[k] 0.9% Sodium Chloride Irrigation, Hospira Inc.
[l] Metronidazole Injection, Hospira Inc.
[m] Fentanyl Transdermal System, SANDOZ Inc, Broomfield, CO.
[n] Metronidazole tablets, PLIVA Krakow, Pomona, NY.
[o] AmoxiTabs, Pfizer Animal Health, New York, NY.
[p] Ciprofloxacin tablets, Ranbaxy Pharmaceuticals Inc, Jacksonville, FL.
[q] Tramadol Hydrochloride Tablets, Amneal Pharmaceuticals, Glasgow, KY.
[r] Famotidine Tablets, IVEX Pharmaceuticals Inc, Miami, FL.
[s] Rose L, Bedard C, Dunn M. Effect of prednisone administration on thromboelastography parameters in healthy beagles (Abstr). J Vet Intern Med 2008;22(3):738.

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