

# Massive transfusion and surgical management of iatrogenic aortic laceration associated with cystocentesis in a dog

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**Case Description**—A 4-year-old 29-kg (63.8-lb) spayed female Husky crossbred was referred for emergency treatment because of catastrophic hemorrhagic shock following attempts at cystocentesis for investigation of suspected urinary tract infection.

**Clinical Findings**—On arrival at the hospital, clinicopathologic assessments revealed rapidly decreasing PCV and worsening hypoproteinemia, compared with findings immediately prior to referral. The dog had severe hyperlactemia. Ultrasonography revealed the presence of free fluid in the abdomen; the fluid appeared to be blood (determined via abdominocentesis).

**Treatment and Outcome**—Urgent surgical exploration was undertaken. Two small lacerations in the ventral aspect of the abdominal aorta just dorsal to the bladder were identified and repaired. Multiple transfusions of packed RBCs (5 units) and fresh frozen plasma (3 units) were administered, and autotransfusion of blood (1.2 L) from the abdomen was performed. The dog recovered well from surgery and anesthesia, but developed signs of severe pain and swelling of both hind limbs, which were attributed to reperfusion injury following aortic occlusion during surgery. Treatment included administration of S-adenosyl-methionine (23 mg/kg [10.5 mg/lb], PO, q 24 h) and analgesia; 5 days after surgery, the hind limb problems had resolved and treatments were discontinued.

**Clinical Relevance**—In the dog of this report, aortic laceration secondary to cystocentesis was successfully treated with a combination of surgery and massive transfusion; the development of reperfusion injury was an interesting and reversible complication of surgery. The possibility of damage to intra-abdominal structures should be investigated if a dog becomes acutely ill after cystocentesis. (*J Am Vet Med Assoc* 2009;235:288–291)

A 4-year-old 29-kg (63.8-lb) spayed female Husky cross was evaluated by the emergency service of the Foster Hospital for Small Animals at Tufts Cummings School of Veterinary Medicine because of acute-onset collapse following cystocentesis by the referring veterinarian. Earlier that day, the dog had been examined by the veterinarian because of hematuria; on the basis of a suspected urinary tract infection, cystocentesis was attempted. The dog was manually restrained in dorsal recumbency for a blind attempt at cystocentesis; no sedatives were administered. A 1.5-inch, 22-gauge needle with an attached syringe was used, but the procedure was unsuccessful. Following the unsuccessful attempt, the dog was admitted for observation and further attempts at urine sample collection. Cystocentesis was attempted again later that day; the second attempt yielded a small amount of frank blood, and the third and final attempt yielded urine. The dog was reported as noncooperative and struggled during the procedures despite restraint. A presumptive diagnosis of a urinary tract infection was made, and the dog was discharged to the owner 1 hour later. The dog was apparently clinically normal at the time of discharge from the clinic.

During the journey home (approx 20 minutes after discharge from the clinic), the dog had an episode of

## ABBREVIATIONS

aPTT	Activated partial thromboplastin time
FFP	Fresh frozen plasma
PT	Prothrombin time
SAMe	S-adenosyl-methionine
TP	Total protein

collapse and was returned immediately to the primary care veterinarian for evaluation. The dog was weak and tachycardic. Results of in-house clinicopathologic testing performed at this time indicated that the PCV was 46%, blood glucose was 79 mg/dL (reference range, 82 to 117 mg/dL), and TP concentration was 5.0 g/dL (reference range, 6 to 7.2 g/dL), respectively. Blood urea concentration was 5 to 15 mg of urea/dL of blood, as determined by use of a reagent testing strip.<sup>a</sup> An IV catheter was placed in the left cephalic vein, and 1.5 L (52 mL/kg [23.5 mL/lb]) of lactated Ringer's solution was administered as a bolus. The dog was referred for ongoing management.

On arrival at the hospital (within 20 minutes of leaving the primary care facility), the dog was recumbent with pale mucous membranes. The heart rate was 206 beats/min with bounding pulses; heart sounds were quiet. Respiratory rate was 40 breaths/min, and rectal temperature was 36.6°C (98°F). An abdominal fluid wave was detected by use of ballottement. A second IV catheter was placed in the right cephalic vein, and a

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sample of venous blood was obtained for assessment of PCV, blood TP and electrolyte concentrations, PT, and aPTT; blood gas analysis; CBC; serum biochemical analyses; and blood typing. Abdominal ultrasonography (performed by use of a portable machine) revealed marked peritoneal effusion; a sample of hemorrhagic fluid was removed via aspiration. At the initial evaluation, the dog's PCV was 24% and blood TP concentration was 1.2 g/dL; analysis of the abdominal fluid revealed that the fluid PCV was 20% and TP concentration was 1.2 g/dL. Results of point-of-care coagulation testing<sup>b</sup> indicated that PT was 21 seconds (reference range, 12 to 17 seconds) and aPTT was 108 seconds (reference range, 71 to 102 seconds). The worsening anemia, hypoproteinemia, and prolonged coagulation times were consistent with massive blood loss and a mild dilutional coagulopathy. Blood gas and electrolyte abnormalities<sup>c</sup> included hyponatremia (141.1 mmol/L; reference range, 147.0 to 154.0 mmol/L), hyperglycemia (120 mg/dL; reference range, 82 to 117 mg/dL), and hyperlactemia (6.5 mmol/L; reference range, 0.3 to 1.0 mmol/L). Ionized calcium concentration in blood was low (1.09 mmol/L; reference range, 1.17 to 1.38 mmol/L). These results were considered consistent with hemorrhage and associated hypovolemic shock.

Crystalloid fluid therapy was continued, and transfusion with packed RBCs was begun as the operating room staff was notified about the urgent need to perform an exploratory celiotomy for control of hemorrhage. On the basis of the severe hemoperitoneum and the close anatomic proximity of the aorta to the urinary bladder, an aortic laceration was suspected, although a large venous laceration could not be excluded. Two units of packed RBCs (18 mL/kg [8.2 mL/lb]) and 1 unit (9 mL/kg [4.1 mL/lb]) of FFP were transfused within 20 minutes. Because of worsening peripheral pulse quality and evidence of cardiovascular collapse, administration of lactated Ringer's solution was continued at 90 mL/kg/h (41 mL/lb/h). Other resuscitative techniques, including hypotensive resuscitation with early operative intervention, or administration of other types of fluids (eg, hypertonic saline or hetastarch solutions) could have been selected.

The dog was premedicated with hydromorphone (0.035 mg/kg [0.016 mg/lb], IV) and midazolam (0.2 mg/kg [0.09 mg/lb], IV), and anesthesia was induced via administration of propofol (2 mg/kg [0.9 mg/lb]). During surgery, mechanical ventilation was applied and anesthesia was maintained with isoflurane (0.75% to 1.5%) and bolus administration of hydromorphone (0.02 mg/kg [0.009 mg/lb], IV) intermittently. The dog's physical status was classified as 4-E (where E indicates emergency), according to the scheme adopted by the American Society of Anesthesiologists.<sup>1</sup>

A ventral midline laparotomy was performed, and 2.2 L of blood was suctioned from the peritoneal cavity. The first 1.2 L of blood was collected aseptically in an autotransfusion device<sup>d</sup> for later use. A large blood clot was seen in the caudal portion of the abdomen, with evidence of continuing bleeding dorsal to the blood clot. The abdominal aorta was located and confirmed to be the source of the bleeding. On inspection, a small pinprick and a 2-mm laceration were observed in the

ventral surface of the aorta immediately dorsal to the urinary bladder; both defects were associated with severe hemorrhage. The aorta was occluded via application of digital pressure cranial to the defects, and the puncture and laceration were each oversewn with 4-0 polypropylene suture<sup>e</sup> and packed with a cellulose-based hemostatic material.<sup>f</sup> Occlusion of the aorta and application of digital pressure on the repair sites were maintained for a further 5 minutes, after which time the hemostatic material was removed, the aortic occlusion was released, and the repair sites were inspected for hemorrhage. The aorta was intermittently occluded for a total of 25 minutes during surgery. No further hemorrhage was evident, and the abdomen was closed in a routine manner. During surgery, the dog was administered physiologic saline (0.9% NaCl) solution (10 mL/kg/h [4.5 mL/lb/h]) along with 2 units of packed RBCs, 2 units of FFP, and autotransfusion of 1.2 L of blood that had been removed from the abdomen. Intraoperative point-of-care clinicopathologic analyses revealed that the dog's PCV was 19%, TP concentration was 2.2 g/dL, sodium concentration was 140.3 mmol/L, ionized calcium concentration was 0.85 mmol/L, and lactate concentration was 1.8 mmol/L. Because of progressive hypocalcemia, calcium gluconate (68 mg/kg [30.9 mg/lb]) was administered IV over a 5-minute period. The worsening hypocalcemia was thought to be associated with the transfusion of citrated blood products, including FFP, packed RBCs, and the autotransfused blood. Recovery from anesthesia was uneventful. The volume of blood products used was approximately 3 L (5 units of packed RBCs, 3 units of FFP, and 1.2 L of autotransfused blood).

Immediately after surgery, results of point-of-care clinicopathologic testing indicated improvement in the dog's condition; PCV was 23%, TP concentration was 3 g/dL, ionized calcium concentration was 0.97 mmol/L, lactate concentration was 1.7 mmol/L, glucose concentration was 100 mg/dL, and sodium concentration was 141 mmol/L. The PT and aPTT values were within reference limits. Heart rate was 77 to 126 beats/min, and systolic arterial blood pressure was 180 mm Hg (reference range, 90 to 140 mm Hg).<sup>2</sup> Administration of cefazolin (22 mg/kg [10 mg/lb], IV, q 8 h) and hydromorphone (0.05 mg/kg [0.023 mg/lb], IV, q 4 h) was commenced, and fluid therapy was changed to lactated Ringer's solution with 20 mEq of potassium chloride/L (3.8 mL/kg/h [1.73 mL/lb/h]). In addition, the dog received another dose of calcium gluconate IV. Intensive care monitoring included continuous ECG and assessment of urine output, blood pressure, and respiratory rate and effort; blood pressure rapidly returned to reference range, and all other monitored variables remained within reference limits during the postoperative period. The dog regurgitated some brown fluid during the first night following surgery.

The following morning, point-of-care clinicopathologic analyses revealed further improvement in the dog's condition; PCV was 28%, TP concentration was 3.4 g/dL, sodium concentration was 144.1 mmol/L, lactate concentration was 0.6 mmol/L, ionized calcium concentration was 1.26 mmol/L, and blood glucose concentration was 105 mg/dL. On physical examina-

tion, the only major abnormality was moderate to severe swelling of both hind limbs; signs of discomfort were evident on palpation of the hind limbs or when the dog attempted to stand. Administration of hydromorphone (for analgesia) and cefazolin (for treatment of a presumed urinary tract infection) was continued. To treat the suspected reperfusion injury, the dog received SAMe (23 mg/kg [10.5 mg/lb]) orally once daily. Because of regurgitation, administration of famotidine (0.7 mg/kg [0.32 mg/lb], IV, q 24 h) and omeprazole (0.7 mg/kg, PO, q 24 h) was initiated for treatment of possible esophagitis or gastrointestinal ulceration. All of the treatments were continued throughout the duration of hospitalization. At this time, the dog was markedly improved and signs of the hind limb discomfort and swelling were decreased; the dog was able to walk outside and eat without additional episodes of regurgitation.

The dog was discharged from the hospital on the third day after surgery. Bacterial culture of urine performed at the primary veterinary care facility yielded *Enterococcus* spp that were susceptible to several commonly used antimicrobials. The owners were instructed to administer potentiated amoxicillin (12.9 mg/kg [5.86 mg/lb], PO, q 12 h) for 14 days, tramadol (3.5 mg/kg [1.59 mg/lb], PO, q 8 h) for 3 to 5 days, and SAMe (23 mg/kg, PO, q 24 h) for 3 to 5 days. At the time of discharge from the hospital, the dog's PCV was 33% and TP concentration was 5.2 g/dL. The dog continued to do well but had an episode of vomiting and reduced appetite 2 days after discharge; treatment with potentiated amoxicillin was discontinued, and on the basis of the antimicrobial susceptibility test results, administration of enrofloxacin (9.4 mg/kg [4.3 mg/lb], PO, q 24 h) was commenced. Administration of famotidine and omeprazole was reinstated at the previous dosages. The dog continued to make a full recovery without further complications from this time point. At the time of suture removal, 10 days after surgery, the dog was clinically normal; PCV was 42%, and TP concentration was 6.0 g/dL. Bacterial culture of a free-catch sample of urine was performed and yielded negative results. Administration of all medications was discontinued.

## Discussion

This case report illustrates a rare and life-threatening complication of an everyday procedure. Cystocentesis is a widely used technique for obtaining urine samples for urinalysis and is specifically warranted when samples for bacterial culture are desired. To the authors' knowledge, this is the first report of aortic laceration as a complication of this technique in dogs. It must be remembered that cystocentesis is not a completely benign procedure and that special precautions, including the use of adequate manual or chemical restraint, should be taken with dogs that are difficult to restrain or that resist the procedure. Ultrasonographic guidance may be useful to ensure adequate urine volume in the urinary bladder prior to attempts at cystocentesis and to direct needle placement. Reported complications of cystocentesis include aspiration of intestinal tract contents, transient hematuria, and hemorrhage from the urinary bladder mucosa.<sup>3</sup> As with all procedures, care should

be taken to master safe and appropriate techniques and to immediately discontinue any procedure that might result in patient harm. This dog survived the cystocentesis-related complication because of prompt recognition of the problem and initiation of treatment by the primary care veterinarian, along with rapid emergency medical and surgical intervention to provide cardiovascular support and correct the underlying cause of the bleeding.

Reports of the management of aortic injuries in dogs are limited. To our knowledge, there are 2 case reports of rupture of the thoracic aorta in dogs. In 1 dog, the rupture was the result of esophageal foreign body complications<sup>4</sup>; for the other dog, it was hypothesized that the injury occurred when the dog was running at the limit of its chain.<sup>5</sup> In both of those cases, the rupture was fatal. In humans, traumatic injury to the descending aorta most commonly results from vehicular trauma and is associated with a 24-hour fatality rate of 99%.<sup>6</sup> In the dog of this report, success was, in large part, attributable to the relatively small size of the aortic injuries.

The 2 major complications that developed in the dog of this report were electrolyte disturbances and hind limb swelling and discomfort. The hyponatremia detected on arrival at the hospital was most likely a result of blood loss and abdominal effusion. As part of the treatment protocol, the dog received massive transfusion of blood products; a total of 5 units of packed RBCs, 3 units of FFP, and 1.2 L of autotransfused blood were administered. A dog's blood volume is approximately 90 mL/kg (equivalent to 2.6 L of total blood volume). For the dog of this report, the transfusion volume administered represented approximately 115% of its blood volume; thus, it fulfills the criteria for massive transfusion. Autotransfusion of blood from the abdomen was particularly appealing because of the lack of suspected contamination with bacteria, urine, or neoplastic cells, which is potentially common in regard to other causes of hemoperitoneum. A decrease in blood ionized calcium concentration following massive transfusion (as detected in the dog of this report) has been reported.<sup>7</sup> Potential complications of massive transfusions include electrolyte abnormalities (eg, alterations in circulating sodium, magnesium, and potassium concentrations), coagulation disturbances, acute lung injury, vomiting, and facial edema as well as hemolytic and nonhemolytic transfusion reactions.<sup>7</sup>

In the dog of this report, the hind limb swelling and signs of pain were interesting complications. The abdominal aorta was intermittently occluded with digital pressure for a period of approximately 25 minutes during surgery while the defects in the aorta were located, assessed, and repaired. We hypothesize that the muscle swelling and signs of pain after surgery were most likely results of reperfusion injury in the hind limbs following establishment of normal blood flow through the aorta once the occlusion was released. Other possible causes for the development of hind limb edema included reduced venous or lymphatic drainage because of thromboembolic disease or recumbency or systemic conditions such as hypoproteinemia, although these systemic diseases would be expected to affect all limbs. The devel-

opment of reperfusion injury was unexpected because of the relatively short period of aortic occlusion. Ischemia leads to metabolism (and rapid depletion) of ATP, the metabolic product of which is hypoxanthine. In the presence of oxygen, hypoxanthine is converted to xanthine and then to uric acid. In an ischemic state, influx of calcium into cells causes conversion of xanthine dehydrogenase to xanthine oxidase, which cannot convert hypoxanthine in the absence of oxygen. This ultimately leads to accumulation of both xanthine oxidase and hypoxanthine. At the moment of reperfusion, the large amount of xanthine oxidase present begins to convert the large concentration of hypoxanthine to urate and superoxide anions; this effect is seen initially at the interface of the endothelium and blood. The superoxide anions are then converted to hydrogen peroxide and ultimately to hydroxyl radicals. The generation of reactive oxygen species occurs very quickly after reperfusion, usually within the first 30 seconds.<sup>8</sup> We speculate that, in the dog of this report, apparent reperfusion injury developed despite aortic occlusion of short duration because the aorta was intermittently occluded, potentially allowing the effects of several small ischemia-reperfusion insults to summate.

Currently, treatment of reperfusion injury is controversial. Various strategies have been proposed, many of which are effective only if they are administered before the reperfusion insult occurs. Treatments include administration of calcium channel blockers, allopurinol, glutathione, and vitamins E and C, which all block the formation of reactive oxygen species. A potentially more clinically useful approach would be to treat patients in which signs of ischemia-reperfusion injury have developed because scavenging reactive oxygen species have already formed. Some of the proposed treatments (iron chelation and administration of dimethylsulfoxide, nitric oxide, or 21-aminosteroids) appear to be most efficacious if they are given before the ischemic insult.<sup>9</sup> Lidocaine has also been proposed as a possible treatment for reperfusion injury.<sup>10</sup> The dog of this report was treated with SAME because of its ability to reduce oxidative damage in liver disease in rat experimental situations, the widespread acceptance of its use as an antioxidant in canine liver disease, and its widespread availability.<sup>11,12</sup> However, there is no evidence in the literature that SAME is an effective treatment for ischemia-reperfusion injury, and the authors are uncertain whether its use contributed to

the dog's recovery. Despite the potential for development of surgery-related complications, such as reperfusion injury, this case highlights the need for rapid surgical intervention in veterinary patients with intracavitary bleeding that do not respond quickly and adequately to more conservative management.

- a. Azostix, Siemans Medical Solutions Diagnostics, Tarrytown, NY.
- b. SCA 2000 veterinary coagulation analyzer, Synbiotics Corp, San Diego, Calif.
- c. STAT Profile Xpress blood gas analyzer, NOVA Biomedical, Waltham, Mass.
- d. Autovac, Boehringer Laboratories Inc, Norristown, Pa.
- e. Surgipro, Covidien, Synteture, Mansfield, Mass.
- f. Surgicell, Ethicon Inc, Somerville, NJ.

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