Abstract

Early recognition of the clinical signs of septic shock is key to a quick diagnosis and life-saving treatment for septic patients. This case report documents the critical role of the veterinary nurse in identifying patients with signs of septic shock, including the vital changes associated with different stages of shock. After diagnosis of sepsis via cytologic, biochemical, and physical evaluation, treatment includes both symptomatic care and surgical intervention. The article will also review stabilization, ultrasound-guided free fluid sampling, placement of advanced vascular access catheters, monitoring the critical patient under anesthesia, and the use of vasopressors in hypotensive patients.
Veterinary nurses play a crucial role in early recognition and treatment of patients in septic shock. The ability to recognize critical vital signs, identify and sample cavitary effusion, place advanced vascular access lines, and run high-level anesthesia is essential to the treatment and recovery of these patients. This case report reviews the presentation, treatment, and recovery of a patient with septic shock secondary to a foreign body perforating the duodenum.

**HISTORY**

Leo, a 6-year-old, neutered male Siberian husky weighing 34 kg, presented to the emergency department for lethargy, pale gums, and shallow breathing. He had a 24-hour history of decreased appetite prior to presentation and had ingested and vomited a sock earlier in the week.

**INITIAL ASSESSMENT**

Upon examination, Leo was dull but responsive. He had a normal respiratory rate and effort. Upon auscultation, he was tachycardic with a heart rate (HR) of 140 beats/minute (bpm) with no heart murmur. Mucous membranes were pale with a prolonged capillary refill time (CRT) of 3 seconds. He had a mildly decreased body temperature of 37.4 °C (99.4 °F). His abdomen was mildly distended and painful upon palpation. A point-of-care ultrasound revealed mild echogenic peritoneal effusion in the cranial abdomen. Doppler systolic blood pressure was 60 mm Hg.

An IV catheter was placed and blood was collected for a complete blood count (CBC), serum biochemical profile, blood glucose, and packed cell volume/total protein (PCV/TP) measurements. Blood glucose was 62 mg/dL (reference range,

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**Take-Home Points**

- Clinical signs of septic shock are different depending on the stage (hyperdynamic versus hypodynamic).
- Free abdominal fluid will appear anechoic (black) on ultrasound and should be sampled in patients displaying signs of septic shock.
- Effusion samples should be evaluated cytologically for degenerate neutrophils and intracellular bacteria.
- Critical patients undergoing anesthesia should have multiple venous access sites, preferentially central venous access in the form of a central line or peripherally inserted central catheter.
- Early administration of vasopressor therapy can increase cardiac output, reducing the need for large-volume fluid bolusing.
80 to 180 mg/dL) and PCV was 60% with a TP of 8.2 g/dL. Clinicopathologic abnormalities included mild hyponatremia and hypochloremia, moderate azotemia and hyperphosphatemia, and mildly elevated alanine aminotransferase.

A 10 mL/kg IV lactated Ringer’s solution (LRS) fluid bolus was administered over 15 minutes. A diagnostic ultrasound-guided abdominocentesis revealed serosanguinous, flocculent effusion with a PCV of 5% and a TP of 3.2 g/dL. Microscopic cytologic examination revealed an inflammatory cell population with degenerate neutrophils and rare intracellular cocci.

TREATMENT

Leo was admitted to the hospital for abdominal exploratory surgery. He was continued on the LRS at 4 mL/kg/h with 2.5% dextrose. He was administered ampicillin/sulbactam 50 mg/kg IV, enrofloxacin 15 mg/kg diluted 1:5 with sterile water IV, maropitant 1 mg/kg IV, and pantoprazole 1 mg/kg IV during stabilization. A recheck Doppler blood pressure was 80 mm Hg. Using the modified Seldinger technique (BOX 1), a 7-French, triple-lumen, 60-cm peripherally inserted central catheter (PICC) was placed in the right lateral saphenous vein. A 20-gauge catheter was placed and secured in the left dorsal pedal artery.

Leo was induced for surgery with a 5 µg/kg fentanyl bolus, midazolam 0.1 mg/kg IV, and alfaxalone 1 mg/kg IV titrated to effect for endotracheal intubation. He was intubated and placed on 2 L/min oxygen flow and 0.5% isoflurane gas. Throughout induction Leo’s direct mean arterial blood pressure (MAP) was around 50 mm Hg; therefore, fluids were increased to 1 L/h. When the hypotension proved unresponsive to fluid therapy, a dilute norepinephrine constant rate infusion (CRI) was started at 0.2 µg/kg/min, isoflurane was turned off, and fentanyl was increased to 10 µg/kg/h.

Exploratory laparotomy revealed a linear cloth foreign body anchored in the stomach with a large duodenal perforation. A gastrostomy was performed to remove the foreign material, and the duodenal perforation was repaired with suture and a serosal patch. Arterial hypotension persisted; therefore, the norepinephrine CRI was increased to 0.4 µg/kg/min and fentanyl was decreased back to 5 µg/kg/h, after which MAP improved to 60 to 70 mm Hg. Leo had an episode of bradycardia (HR ~40 bpm) that improved after administration of a half-dose of atropine (0.02 mg/kg IV). A nasogastric tube was placed through the left nostril intraoperatively.

Leo recovered from surgery uneventfully with normal postoperative vitals, excluding dull mentation and hypothermia (34.8 °C [94.6 °F]). His fluids were decreased to 3 mL/kg/h and he remained on the norepinephrine CRI at 0.4 µg/kg/min with a direct MAP of 80 mm Hg. His blood glucose was 104 mg/dL on 2.5% dextrose supplementation. His recheck PCV and TP were 45% and 3.2 g/dL, respectively. A double unit of DEA (dog erythrocyte antigen) 1.1 negative fresh frozen canine plasma was administered over 4 hours. Leo was maintained on electrocardiography (ECG) monitoring and continuous direct MAP monitoring overnight.

An indwelling Foley urinary catheter was placed during recovery. Urinary catheter care was performed every 8 hours and urinary output was quantified every 4 hours. The nasogastric tube was aspirated and quantified every 4 hours. Recumbency care and passive

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**Seldinger Central Line Placement**

1. Measure the length from insertion site to desired termination.
2. Aseptically prep and drape the chosen site. Don sterile gloves.
3. Insert catheter into chosen vessel and remove stylet.
4. Insert guidewire until the remaining wire is shorter than the sterile drape. Remove catheter. Never let go of the wire.
5. Insert and remove dilator(s) in order of ascending size.
6. Insert catheter tip over guidewire and feed guidewire out of the vessel into the catheter until the tip can be visualized protruding from 1 lumen. Taking care to grab the extruding wire, feed catheter into the vessel to the measured length and remove the wire.
7. Remove intraluminal air and flush lumens.
8. Suture in place.
range of motion were performed every 4 hours. Central line luminal maintenance was performed every 4 hours, and bandaging was changed daily.

Over the next 24 hours Leo was normotensive and he was successfully weaned off his vasopressor support. He was hospitalized for another 3 days with continuation of the treatments listed above, with the addition of nasogastric tube feedings of increasing amounts daily up to resting energy requirement. Daily blood analysis revealed improvement in all clinicopathologic abnormalities; however, mild azotemia was persistent and a mild anemia developed (suspected dilutional). Leo required nasogastric tube feedings throughout his hospital stay and never began voluntary oral intake. He was discharged on the fourth day after admittance for continued care at home and an at-home food trial.

OUTCOME
Leo started eating at home and continued to improve overall. He returned for regular rechecks for several weeks. His azotemia persisted, with his final recheck creatinine at 2.1 mg/dL (reference range, 0.5 to 1.8 mg/dL) before he transitioned to follow-up with his primary care veterinarian.

DISCUSSION
Recognizing Sepsis
Sepsis is a leading cause of mortality in veterinary medicine and is associated with a poor to grave prognosis. Early recognition, intensive supportive care, and treatment of the underlying source of infection are crucial to increase chances of survival. Patients show signs of systemic inflammatory response syndrome (SIRS) and likely 1 or more types of shock. Distributive shock is common on presentation.

In dogs, clinical signs of distributive shock are caused by a hyperdynamic state of vasodilation and include tachycardia, pyrexia, and hyperemic mucous membranes. This hyperdynamic state is caused by an exaggerated immune response to cytokines released during the body’s response to a bacterial infection, which leads to loss of the ability to balance homeostatic mediators. If this state is left unaddressed, decompensated hypodynamic shock develops. Clinical signs of this stage of sepsis in dogs include tachycardia, hypothermia, depressed mentation, pale mucous membranes, and a prolonged CRT. The body enters a phase of severe septic shock when arterial hypotension is present despite fluid resuscitation. Persistent arterial hypotension leads to organ dysfunction and eventually to multiple organ failure.

In dogs, the stages of SIRS and sepsis may be diagnosed based on several criteria. A CBC, serum biochemical profile, coagulation times, blood lactate, and blood gas may be assessed. CBC findings consistent with SIRS and sepsis include leukocytosis or leukopenia from overstimulated immune response, hemoconcentration from possible volume depletion, and thrombocytopenia from consumption and disseminated intravascular coagulation. Common biochemical abnormalities include azotemia from acute kidney injury, hyperbilirubinemia from cholestasis, hypoglycemia due to increased glucose use or impaired gluconeogenesis, hypoalbuminemia due to decreased synthesis and destructive catabolism, and hyperlactatemia from hypoperfusion (severe hyperlactatemia is associated with poorer prognosis). Coagulation times may be prolonged due to increased use and decreased production of clotting factors. ECG and cardiac ultrasonography may reveal arrhythmias, decreased cardiac contractility, and ventricular hypovolemia. Patients with severe septic shock have a persistent MAP of <60 mm Hg despite fluid resuscitation.

In this case, Leo presented with clinical signs consistent with decompensated hypodynamic shock (pale mucous membranes, prolonged CRT, decreased responsiveness) and laboratory results consistent with SIRS. His altered mentation, mild hypoglycemia and hypotension, and presence of peritoneal effusion immediately raised concern for potential bacterial sepsis.

Triage Ultrasonography and Free Fluid Cytology
Point-of-care ultrasonography can aid in the timely identification of life-saving treatments. Recognition of effusion in either the pleural or peritoneal cavity in combination with vital signs consistent with septic shock should create a high suspicion for septic effusion. A diagnostic abdominocentesis should be performed, followed by quick cytologic assessment of the effusion. The emergency room veterinary nurse should be comfortable performing a free fluid evaluation as part of the triage process.

In this case, peritoneal effusion was recognized in the cranial abdomen. The area was clipped and aseptically
prepped, and a 22-gauge, 1.5-inch needle attached to a 6-mL syringe was introduced into the fluid pocket with ultrasound guidance to obtain a sample. A cytopsin was performed, and the sample was cytologically examined using the line-prep technique. Swift collection of a peritoneal effusion sample and cytologic assessment for the presence of intracellular bacteria (FIGURE 2) were key to the quick decision for emergency surgery to remove the source of sepsis.

Advanced Vascular Access
Early placement of a central sampling catheter in critical patients aids in serial blood collection and administration of vasopressors or high-osmolality solutions such as high-concentration dextrose. In this case, a multilumen PICC was placed instead of a jugular central line based on the risk for the development of a coagulopathy and the author’s preference in large dogs (FIGURE 3).

Invasive blood pressure monitoring via an arterial catheter is considered the gold standard for monitoring blood pressure. For hypotensive critical patients, arterial catheters can provide lifesaving real-time blood pressure updates without the technical time required for noninvasive blood pressure monitoring. Arterial catheter placement is technically challenging, particularly in hypotensive patients with difficult-to-palpate pulse pressures (FIGURE 4). Timely flushing with heparinized saline may help prevent clotting of the catheter during patient transport.

In this case, the area of the left dorsal pedal artery was clipped and aseptically prepped. The pulse was sterilely palpated with the author’s nondominant hand, and the catheter was placed, taped, and flushed with heparinized saline. The catheter was connected to the arterial blood pressure monitoring transducer, which
was zeroed and kept at the level of the heart. A bag of 0.9% NaCl was heparinized to 1 U/mL and placed in a pressure bag inflated to 300 mm Hg to deliver 3 mL/h.

**Critical Patient Anesthesia**

Leo was prepped for surgery prior to induction and induced in the operating room to decrease anesthetic time. Alfaxalone was chosen as an induction agent based on its studied reduced cardiovascular and baroreceptor depression. Fentanyl was chosen for pain relief based on its titratability, allowing a decrease in anesthetic gases and thus cardiopulmonary compromise.

Leo was persistently hypotensive during the pre- and perioperative periods, with no improvement after fluid resuscitation; therefore, vasopressor support in the form of a norepinephrine CRI was initiated. Norepinephrine was chosen to increase cardiac output and organ perfusion. The CRI was diluted to match the dose rate (0.2 µg/kg/min = 2 mL/h) for ease of administration.

Leo’s episode of bradycardia may have been vagally mediated and was responsive to anticholinergics. In the event of cardiopulmonary arrest, the author was prepared with epinephrine, atropine, and naloxone as well as flumazenil to reverse the fentanyl and midazolam.

**SUMMARY**

The successful outcome of this case depended on the quick recognition of peritoneal effusion and treatment of septic shock. Collection of a peritoneal effusion sample and identification of intracellular bacteria allowed for efficient diagnosis and led to quick removal of the septic source. Swift placement of advanced vascular access catheters allowed for serial blood sampling and direct arterial blood pressure monitoring. Early administration of vasopressors enabled the patient’s hemodynamic stabilization. Anesthesia was multimodal and well balanced to reduce cardiopulmonary compromise. Treatment was successful and the patient survived to discharge; however, remaining azotemia was likely secondary to acute kidney injury from arterial hypotension. **TVN**

**References**


**FIGURE 4.** Arterial catheter in place.

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Jennifer has worked in emergency and critical care for 7 years. She achieved her VTS in 2021. She is currently working as a critical care veterinary nurse and clinical supervisor at MedVet in Salt Lake City, Utah. Her interests include ultrasonography, triage, critical patient anesthesia, and advanced vascular access. She is the proud mother of 4 cats and spends her free time enjoying the outdoors.