

## CASE REPORTS

The following two examples are for you to use as a guide while writing your case reports.

### Remember the formatting requirements:

The reports **must** be typed and **double spaced**. Case reports **must not exceed five pages each**. Case reports not meeting these requirements will be rejected. The following manuscript form should be used: **Times New Roman** font; margins justified and 0.5" top, bottom, and sides; top right of each page: **CASE REPORT # \_\_\_, APPLICANT NAME, PAGE #**, font size 10, all caps, bold; **Headings** capitalized, font size 10, bold; body font size 10.

**SIGNALMENT:**

Bailee, 3 year old Staffordshire Terrier (FS), weight 21kg, Date \_\_\_\_\_, Case log # \_\_\_\_\_

**HISTORY:**

Bailee was referred to the Ontario Veterinary Hospital for evaluation of possible septic peritonitis. She had undergone an exploratory laparotomy and multiple enterotomies six days previous to remove a linear foreign body from the jejunum. Prior to that, she had a two week history of intermittent vomiting and diarrhea.

**INITIAL PHYSICAL EXAM:**

Upon presentation, Bailee appeared very depressed. She was pyrexia (39.7C), a little tachycardic (120bpm), respiratory rate 40, with very injected and tacky mucous membranes. Dorsal pedal pulses palpable, but non-invasive BP measurements revealed hypotension (115/53, MAP 69). Dehydration was estimated at 10%. A grade II/VI systolic heart murmur was noted. Lung sounds were normal. Her abdomen was distended and a fluid wave was palpable. Serosanguinous fluid was seen dripping from the abdominal incision site. EKG revealed a normal sinus rhythm.

**INITIAL INTERVENTIONS:**

I placed two cephalic, 18G 2" IV catheters. A 2L bolus of P-148 was initiated and given over the next hour. A 300 ml bolus of Pentastarch was given as an initial intervention for the hypotension. Antibiotics were started (enrofloxacin 5mg/kg). I drew the entry blood for our ICU's quick assessment tests, in addition to a CBC. An abdominal tap was performed by the veterinarian. It revealed many degenerative neutrophils and both intracellular and extracellular bacteria (rods and cocci). An immediate exploratory laparotomy was planned.

**LAB RESULTS:**

Laboratory tests revealed an elevated PCV (55%) and moderate hypoproteinemia (TS 4.6/L). Considering her hypovolemic state, her true PCV/TS were assumed to be considerably lower. Glucose was low normal at 3.7mmol/L. Her blood urea nitrogen stick (Azostix) was elevated at 30-40mg/dl. Electrolytes showed a hyponatremia (129mmol/L) and a decreased chloride (100mmol/L), potassium was normal. Blood gases revealed a metabolic acidosis with little or no respiratory compensation (pH 7.330, PaCO<sub>2</sub> 36.4, HCO<sub>3</sub> 18.1, ABE -5.6). Her activated clotting time (ACT) was prolonged at 135 seconds. I placed a urinary catheter and Bailee was taken to surgery.

**SURGICAL INTERVENTION:**

The exploratory laparotomy revealed a large volume of serosanguinous fluid with particulate matter. All three previous enterotomy sites had dehisced. A markedly inflamed and edematous pancreas was seen. Cultures and tissue samples were

taken. Two enterotomy sites were debrided and repaired. Eight inches of jejunum around the last enterotomy site was dissected and an anastomosis performed. The abdomen was lavaged with large amounts of sterile saline, but due to the severity of the septic peritonitis, the abdomen was left open to facilitate drainage and a sterile abdominal bandage was applied.

**PATIENT MANAGEMENT CONCERNS:**

- Hypotension; due to decreased oncotic pressure secondary to protein loss into the abdomen, and/or hypovolemia due to third spacing into the abdomen, and/or vasodilation related to sepsis (visible by Bailee's already injected mucous membranes)
- Pain management; involving both the post-operative requirements and the potential for severe pain from the concurrent pancreatitis
- Decreased renal perfusion; an issue due to the potential for hypotension and hypovolemia (if MAP is <60, then renal perfusion can be compromised)
- DIC (Disseminated Intravascular Coagulation), a concern in any severe trauma or disease state due to the large inflammatory response, and Bailee's elevated ACT
- Rapid correction of the hyponatremia: if the Na is increased faster than 0.5mEq/hr, there is danger of neurological signs secondary to the loss of cellular equilibrium in the brain. Na moves quickly into the cells and water moves too quickly out, the cell shrinks resulting in possible lethargy, seizures, and coma
- Adequate nursing care; due to the recumbent nature of the patient and the known potential for extreme fluid loss in the abdomen, watching for seepage from the abdominal bandage, and closely monitoring this patient's ins and outs is vital

**IMMEDIATE POST-OPERATIVE INTERVENTIONS:**

A nasal cannula was placed by the ICU nurse and oxygen was delivered at 2L/min, as during surgery Bailee's oxygenation was poor. The first of two FFP transfusions over the next 8 hours was started (following standard test dose protocol and monitoring for reaction). Her post-operative PCV was 32%, with a TS of 3.0g/L. Bailee was very hypotensive (52/35, MAP 40). A dopamine CRI was started at an inotropic dose of 5ug/kg/min, this was eventually increased to 10ug/kg/min, needed to maintain her Map over 60.

Over the next 8 hours, urine production was poor, just reaching 1ml/kg/hr with sp. G 1.040-1.050. Crystalloid therapy remained P-148, at 3-4 times maintenance rates. Several 150ml boluses were given to increase urine production and to keep up her blood pressure. A 20ml/hr CRI of Pentastarch was also maintained. In addition to the FFP, this was given to try and maintain her oncotic pressure and keep fluids in the intravascular space.

Oxymorphone IV (0.05mg/kg) was given frequently for pain management. Temperature, heart rate and respiration were monitored constantly, all remained elevated. Continuous EKG showed a normal sinus rhythm. Her post-operative ACT was significantly greater (>4 min.). Bailee continued to have a metabolic acidosis. Her electrolytes were slowly improving and by 12 hours post presentation, her sodium was 134mmol/L.

Her bandage was monitored for signs of seepage, which would make a bandage change necessary. If the outer layers of the bandage get wet, they can 'wick' bacteria into the open abdomen. Clindamycin (10mg/kg) was added to her antibiotic regime.

#### **FURTHER INTERVENTIONS:**

Into the second day, Bailee became more stable. Blood pressure improved (MAP 100), and the dopamine CRI was slowly weaned off. Urine production improved to normal limits. The metabolic acidosis had resolved. The fluids were changed to 0.9% NaCl with 20 mEq/L KCl to further improve her slight hyponatremia. Fluids remained at four times maintenance rates, but potassium administration did not exceed 0.5mEq/kg/hr. Arterial blood gases showed good oxygenation, and the oxygen administration was decreased to 1L/min.

Her ACT had increased slightly (140 sec). The veterinarian decided she should receive two additional FFP transfusions through the day, to treat increasing ACT and the growing concern of DIC. Heparin therapy was also initiated to prevent possible DIC. Her total solids were maintained from around 3.2 to 3.4g/L. A human albumen transfusion was started, but after a few hours of administration, Bailee developed significant facial swelling. I stopped the transfusion, notified the veterinarian, and she was treated with diphenhydramine IV (2mg/kg). The swelling subsequently improved. Her PCV decreased by 8%, and a packed RBC transfusion was given. The PCV subsequently increased to 24%.

I assisted with the placement of a double lumen jugular catheter (MILA). Partial parenteral nutrition was then initiated. Due to the pancreatitis and the state of her GI tract, it would be a while before Bailee would be able to have any oral nutrition. Oxymorphone continued to be administered as needed (q4hr), and bandage changes occurred almost every 8 hours. She continued to lose copious amounts of fluid into her abdomen.

All vitals continued to be monitored frequently (she was at risk for fluid overload and subsequent pulmonary edema, due to the hypoproteinemia and high fluid rate). All remained within normal limits. Fluid ins and outs were closely monitored and appeared balanced. Recumbent patient care was initiated. The patient was moved from right to left lateral recumbency to decrease the risk of lung atelectasis and peripheral edema. Physiotherapy was initiated. All catheters were checked and maintained to reduce the risk of infection.

**CONTINUED CARE:** Over the next 3-6 days, Bailee continued to improve. On the third day she went back to surgery for a laparotomy and lavage and on the sixth day this was repeated and the abdomen closed. Post-operative blood pressures remained stable. She continued to receive a number of FFP transfusions during this time to keep up with the protein losses and because of her pancreatitis. Vital stats remained within normal limits. Laboratory values remained stable, including the ACT, which was within normal limits on the fifth day. Urine production was good and her fluid rate was decreased slowly. Bailee's attitude and demeanor were improving rapidly.

By the sixth day, blood work revealed normal pancreatic values and that evening she was started on oral food. Small amounts of an intestinal formulation (very easily digestible) were offered and accepted readily. Once her oral intake met her caloric requirements, the PPN was discontinued. The urinary catheter was pulled. She developed frequent episodes of diarrhea, but this was attributed to a number of factors; gastrointestinal irritation and motility problems secondary to the resolving septic abdomen and the reintroduction of solid food. This slowly improved. Bailee was discharged home 11 days after presentation to the unit.

**SIGNALMENT**

Frankie, one-year-old male Australian Shepherd; approximately 50#; Date: \_\_\_\_\_ Case log # \_\_\_\_\_

**HISTORY**

Frankie presented to the referring veterinarian at 0900; one half hour after being kicked by a horse. The owners did not know what part of his body had been kicked. Frankie was found 10 minutes later, unresponsive and gasping for breath. A cephalic IV catheter was placed and approximately 1500 mls LRS and 500mg prednisolone sodium succinate was given. An attempt was made to lower the dog's temperature (106.4F) by cool water rinsing. Oxygen therapy by mask was also delivered until time of transfer to our emergency service at 1300hrs.

**INITIAL PHYSICAL EXAMINATION**

Frankie presented to the ICU laterally recumbent. Upon my triage exam, I found Frankie to be unresponsive, hypoventilating, and hyperthermic (104F). He was cyanotic, tremoring with muscle fasciculations, and both pupils were miotic and fixed (negative pupillary light response). Frankie was tachycardic at 170bpm, and I heard considerable upper airway noise on inspiration. His expiratory efforts were quite diminished giving a gasping effect to every breath. No external signs of trauma were present aside from scleral hemorrhages OU.

**INTERVENTIONS**

I applied pulse oximetry and continuous ECG immediately. A SpO2 of 89% indicated Frankie needed oxygen supplementation; and according to his presenting signs of head trauma, we also needed to lower his CO2 in an effort to reduce intracranial pressure. Given the patient's stuporous mentation, intubation was possible without any sedation or anesthesia; although it did prove to be quite difficult due to a large amount of laryngeal swelling present. Once intubated, bloody fluid refluxed up through the endotracheal tube indicating pulmonary edema, most likely of neurogenic origin. It is believed that neurogenic pulmonary edema can result from head trauma or secondary brain injury. It could also result from the direct thoracic trauma. Careful suctioning was done to clear the airway. I was cautious due to a danger of increasing intracranial pressure if Frankie coughed. A vagal response of decreasing heart rate and possible cardiac arrhythmias might also occur with careless suctioning.. An arterial blood gas/electrolytes revealed high CO2 and O2 (on 100% O2), and acidosis (CO2 58.9mmHg, O2 500mmHg, and pH 7.148).

Frankie was placed in sternal recumbency (to fully ventilate his lungs equally) with his head at approximately a 20 degree angle (to help decrease intracranial pressure ICP). Hyperventilation was also used to try to decrease ICP. The cool metal table and cold packs around Frankie helped to decrease his temperature. Intravenous fluids were not pushed due to both the pulmonary edema and the neurologic signs. The ECG tracing remained stable with a normal sinus rhythm, but still slightly tachycardic (150bpm).

I administered furosemide IV within the first 15 minutes, followed by a mannitol infusion over 30 minutes. These were given to treat the increased ICP. Frankie was beginning to tremor more and was exhibiting opisthotonus, so diazepam was given IV to control seizures. A neurology consult advised full radiographs, CT scan, and careful use of mannitol. That advice (regarding mannitol) is due to a contraindication of this drug administered in the presence of intracranial hemorrhage. Mannitol may leak out into the brain at the site of the hemorrhage and actually pull more fluid into that specific area. A routine CBC, serum chemistry, and repeat arterial blood gas were submitted. A CT scan of the skull, along with cervical spine and thoracic radiographs were requested. I placed an indwelling urinary catheter to monitor urine output and assist in cleanliness.

The thoracic radiographs showed no further evidence of pulmonary edema; but did reveal a moderate interstitial pattern compatible with hypoinflation. Frankie's cervical radiographs showed no abnormalities. The skull radiographs revealed multiple fractures of the right parietal and right temporal bones. The CT scan of the skull further defined the fractures as including the occipital bone. Ventral displacement of the occipital bone segments, along with medial displacement of the parietal and temporal bone segments were visualized. Obscured visualization of the right ventricle and displacement of the midline falx to the left indicated the high degree of inflammation present. Also, increased densities in the ventral cerebrum were indicative of acute hemorrhages. Debate followed among the neurologists concerning surgery to remove segment of bone in order to alleviate ICP. The owners requested a few hours to thoroughly consider the option.

Two and one half hours after admission, Frankie was returned to ICU to be placed on a ventilator. Since admission, the patient had been manually hyperventilated on 100% oxygen. I set up the BEAR 2 Adult Volume ventilator with the humidifier chamber (temperature controlled), and the color-coded lines (to ease set up). No sedation or anesthesia was necessary due to the patient's mentation (now comatose). Frankie no longer vocalized in response to any kind of stimulus.

The goal in setting the ventilator would be to lower his CO<sub>2</sub> (again, to decrease the intracranial pressure), and maintain the pliability of his lungs by full inflation. After the initial settings, adjustments were made according to arterial blood gas results taken at q30 minutes. By means of hyperventilation, the CO<sub>2</sub> was kept between 27-30mmHg. The ventilator settings were as follows: simultaneous intermittent mandatory ventilations (SIMV) mode; tidal volume @ 10mls/kg; normal pressure limit at 20ml; O PEEP (none planned until lungs show noncompliance); 40% oxygen.

One hour after the start of ventilation, Frankie began to have seizure activity. Diazepam was administered again, followed by a loading dose of phenobarbital IV. I placed a double lumen catheter in the right saphenous vein in preparation for total parenteral nutrition (TPN) administration. I did not use a jugular vein due to risk of increasing ICP. A jejunostomy tube would be another option for nutrition during Frankie's hypermetabolic state (due to head trauma). This would have to be placed surgically, if he went

for cranial decompression. Both a PEG tube and an NG tube are inappropriate due to his mentation (risk of vomiting and aspiration). An attempt to place an arterial catheter was unsuccessful, so doppler blood pressures were monitored until an arterial line could be placed. Frankie was started on a minimal IV infusion of hetastarch along with his LRS (colloid/crystalloid combination) to maintain his fluid volume and blood pressure. His diastolic pressures remained at 80-85mmHg. Minimal adjustments to the endotracheal tube were made in an attempt to avoid any stimulus that would cause an increase in ICP (example: cough). I made slight changes in cuff inflation, and very slight changes in position, every hour. Saline flushes and suctioning were scheduled for BID only.

### **OUTCOME**

Six hours after ventilation support, Frankie's body temperature and heart rate began to increase. His rectal temperature was >106F, and his heart rate reached 180bpm. Frankie's pupils remained fixed (no pupillary light response), but were now dilated. Generalized muscle rigidity was beginning to appear. Neurological signs of this nature indicate cerebral herniation or very severe brain stem damage. There was no real chance for this patient to recover from the amount of trauma and secondary injury (edema and hemorrhage) to the brain. After a discussion with the owners by telephone, Frankie was euthanized. No necropsy was performed.