

SIGNALMENT

Frankie, one-year-old male Australian Shepherd; approximately 50#; MR #606184

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 CO₂ (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 CO₂ 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.

