“The Boy Who Lived”

A Case Report of an Intracranial Extradural Hematoma in a Feline

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Introduction:

Felines commonly present on an emergency basis for a history of a traumatic event, including animal attacks, falls, or being hit by a car. Traumatic brain injuries are defined as either physiologic or structural damage to the brain caused by an outside source, and these injuries secondary to head trauma occur with relative frequency (10). These injuries can consist of skull fractures, extraparenchymal hemorrhage, or contusions of the brain parenchyma. Although head trauma does not always result in a traumatic brain injury, prompt diagnosis and aggressive treatment of patients with traumatic brain injury is vital for a positive outcome, as treatment of these injuries is time-sensitive.

History and Presentation:

Malfoy is an approximately 12 year-old male castrated domestic shorthair cat who presented to MSU Neurology Service on October 22, 2018 for evaluation of being laterally recumbent and stuporous. He was found on October 16, 2018 lying on the ground and unresponsive. He had urinated on himself, and it appeared that he had been recently paddling. Malfoy was reportedly completely normal earlier that day. He spends time both indoors and outdoors with a large amount of land to roam freely. He is commonly seen to eat lizards, snails, birds, and other wildlife. At the time of presentation, he was also reported to have a 6-month history of weight loss and decreased appetite.

Malfoy was immediately presented to his primary veterinarian after being found laterally recumbent outside. He was agonal breathing on arrival and was successfully resuscitated with doxapram. He was also noted to have marked bilateral mucopurulent nasal discharge. Complete blood count and chemistry panel were unremarkable. Due to Malfoy's profoundly abnormal

mentation, magnetic resonance imaging (MRI) was performed at Carriage Hills Animal Clinic. The MRI revealed a large smoothly marginated extradural mass with rim contrast enhancement compressing the left cerebrum. Tests for feline leukemia virus, feline immunodeficiency virus, Cryptococcus, and Neospora were all negative. Toxoplasma IgG was elevated (consistent with chronic exposure) but IgM was negative. Malfoy was treated with prednisolone and Clavamox, and an esophageal feeding tube was placed on October 19th to facilitate feeding due to his mentation. He had at least one witnessed seizure on October 20th while hospitalized with the primary veterinarian, which was treated successfully with diazepam. With concern for a frontal meningioma or other neoplasm on his MRI, Malfoy was then referred to MSU for further treatment.

On presentation to the MSU Veterinary Specialty Center, Malfoy was laterally recumbent and stuporous with cervical ventroflexion. He had significant bilateral mucopurulent nasal discharge with depigmentation and ulceration of the nasal planum and absent airflow through the right nare. Green to black diarrhea was also noted on his hind end. He was bradycardic (HR 140 beats per minute) and had increased respiratory effort with loud stertor. His respiratory rate (36 breaths per minute) and temperature (100.3*F) were within normal limits. Crackles were auscultated bilaterally but were more severe in the right lung fields. Cardiac auscultation was normal with no murmur or arrhythmia heard, and his femoral pulses were strong bilaterally. He was not painful on abdominal palpation, and all palpable lymph nodes were a normal size..

A comprehensive neurological exam was performed. Malfoy was stuporous and non-ambulatory tetraparetic. Menace response, visual tracking, and palpebral reflex were absent in both eyes, and both nictitating membranes were elevated. He had normal pupillary light reflexes in both eyes but decreased nasocortical sensation on the right side. Physiologic nystagmus was

decreased in both eyes, and postural reactions were absent in all 4 limbs. All segmental reflexes were normal, and he had no cutaneous trunci reflex. These findings were considered consistent with a multifocal intracranial neurolocalization.

A complete blood count (CBC) and serum chemistry were performed. The CBC showed a mild thrombocytopenia but was otherwise unremarkable. The serum chemistry revealed a moderate hypernatremia, mild hyperchloridemia, mildly elevated ALT, mild hyperglycemia, mild hypoalbuminemia, and a mildly elevated CK. A urinalysis was within normal limits. Malfoy then underwent contrast computed tomography (CT) of the head, which revealed a fluid or soft tissue opaque mass within the right nasal passage that was likely invading into the left nasal cavity, as well as destruction of the nasal turbinates. Hyperostosis of the right frontal bone was also appreciated, which could be either congenital or caused by trauma. Within the dorsolateral aspect of the left frontal lobe, confluent with the calvarium, there was a heterogeneous soft tissue dense mass measuring 11 x 15 x 13 mm. This mass demonstrated minimal, heterogenous contast enhancement and was causing compression of the left lateral ventricle. This intracranial mass was considered consistent with the suspected meningioma seen previously on MRI. A thoracic CT scan was also performed to further investigate the bilateral crackles auscultated, which showed no evidence of nodular pulmonary metastatic neoplasia. Abdominal radiographs showed diffuse dilation and gas within the gastrointestinal tract, most likely due to aerophagia, as well an incidental trichobezoar within the stomach. An abdominal ultrasound was also performed and revealed a diffusely nodular liver, material within the gallbladder, changes suggestive of pancreatitis, a small amount of free abdominal fluid, and debris within the urinary bladder. Fine needle aspirates of the liver showed no atypical cells or etiologic agents, and analysis of a sample of the free abdominal fluid was consistent with a

transudate with no atypical cells or etiologic agents. Cytology of the mucopurulent nasal discharge showed neutrophilic inflammation with numerous degenerate neutrophils and low numbers of macrophages. No atypical cell population or etiologic agents were found.

Malfoy was presumptively diagnosed with an intracranial meningioma based on the lesion's MRI and CT characteristics. Due to Malfoy's substantial neurologic deficits and the duration of clinical signs, he was not an ideal surgical candidate. After thoroughly discussing concerns for significant anesthetic and surgical and risks, Malfoy's owners elected to proceed with a craniectomy to debulk his suspected meningioma. Prior to surgery, he was blood typed as type A, and a coagulation panel was within normal limits. Malfoy underwent a left rostrotentorial craniectomy on October 24th. Upon entering the calvarium, a dark red to black extradural mass was visualized compressing the left frontal lobe. Samples of the mass were collected for culture, cytology, and histopathology. The remainder of the mass was removed with suction, neurosurgery spears, and gentle traction. Blind nasal biopsies were also performed under general anesthesia, and the nasal cavity was thoroughly flushed with sterile saline. Malfoy did well under anesthesia, and his recovery was uneventful.

Histopathology of Malfoy's intracranial mass was consistent with a hematoma, and culture was negative. Given the peracute onset of Malfoy's neurological signs, this intracranial hematoma was considered most likely to be secondary to head trauma, although none was witnessed. Histopathology of the nasal biopsies showed severe suppurative and lymphoplasmacytic rhinitis with ulceration and intraepithelial blisters. Culture of the nasal tissue grew *Bordetella bronchiseptica* and *Stenotrophomonas maltophilia*.

Pathophysiology:

Traumatic brain injuries are described conceptually in terms of both primary and secondary injury. Primary injuries occur at the time of the initial traumatic event due to the direct mechanical damage of intracranial structures. Primary injuries may include hematomas, both epidural and subdural, skull fractures, and traumatic axonal injury. Secondary injuries have a delayed onset and can be due to inflammation, production of reactive oxygen species, or other biochemical changes that occur as a result of the primary injury. Systemic factors such as hypotension and hypoxia, as well as intracranial factors such as seizures, edema, or hemorrhage, can exacerbate secondary brain injury. Therapeutic intervention is directed at preventing or minimizing the effects of secondary brain injuries (5, 9, 10, 13).

There are three anatomic components that comprise the intracranial contents: the brain parenchyma, cerebral blood flow, and cerebrospinal fluid. Because the cranial vault is a rigid structure, when one compartment increases in volume, the other two must decrease to maintain appropriate intracranial pressure. The brain's ability to adapt to changing intracranial volume and pressure is described by the Monroe-Kellie doctrine (12). With head trauma, there is often an increase in intracranial volume due to the presence of edema, hemorrhage, or skull fractures. The brain has autoregulatory mechanisms to accommodate mild changes in volume without consequence, called intracranial compliance. However, severe or acute changes in intracranial volume can cause a concomitant increase in intracranial pressure (10). This results in a decrease in the cerebral blood flow due to vasoconstriction, which can lead to cerebral hypoxia and potentiation of secondary injury to the brain parenchyma. The Cushing reflex, or CNS ischemic response, may be seen secondary to an acute or severe increase in intracranial pressure. Cerebral blood flow is decreased due to increased intracranial pressure, leading to decreased carbon dioxide (CO_2) removal from the tissues. Hypercapnia is detected by the vasomotor center and

stimulates the sympathetic nervous system, resulting in an increase in systemic blood pressure to improve the cerebral perfusion pressure (10, 13). Baroreceptors in the aortic arch and carotid body detect the resulting hypertension, leading to reflex bradycardia (1, 14). An irregular respiratory pattern, Cheyne-Stokes respiration, may also be seen, and is described as a pattern of hyperventilation followed by a period of apnea (12, 13). Together, systemic hypertension, bradycardia, and Cheyne-Stokes respiration comprise the Cushing Triad. When present, rapid intervention is necessary to prevent brain herniation, of which there are four possible types: falcine, transtentorial, foramenal, and calvarial (12, 13).

Intracranial hemorrhage can occur as a result of damage to blood vessels either within the intracranial space or the brain parenchyma itself. Hemorrhage can occur in the subarachnoid, subdural, and/or epidural spaces (5, 8, 13). Epidural hemorrhages are most commonly due to damage to meningeal arteries, after which blood accumulates rapidly and causes acute clinical signs (12). The location of the bleed in relation to the brain determines the specific nature of clinical signs, depending on which neural structures are damaged or compressed. Hemorrhage may occur immediately after the initial injury or up to 48 hours post-injury and may continue to expand for 24 hours (3, 8). Hematomas can cause direct damage to the brain parenchyma; additionally, edema forms around the hematoma, causing further damage and inflammation. The presence of blood is also proinflammatory (12). Though there is limited data on intracranial hemorrhage in felines, one study found that 96% of dogs and cats with traumatic brain injuries had evidence of intracranial hemorrhage. Based on the limited data available, epidural hematomas appear to be the least common of the three types (4, 8).

Diagnostic Approach:

When presented with a patient with neurologic signs secondary to known or suspected trauma, it is imperative to thoroughly examine the animal for other systemic injuries to identifying any additional life-threatening trauma. The literature reports that up to 60% of humans presenting with head trauma also have injuries to other body systems (9). When head trauma is present, maintaining normal oxygenation and blood pressure to reduce secondary brain injury is imperative. Blood pressure, electrocardiography, and respiratory pattern should be monitored for evidence of the Cushing reflex. Once the patient is systemically stabilized, a full neurologic examination should be performed prior to administration of analgesia in order to most accurately assess the neurologic status. When possible, the full examination should include assessment of mental status, cranial nerve function, proprioceptive reactions, spinal reflexes, gait, and nociception, and special care should be taken to observe pupil size and eye position (6, 9, 13). Care should be taken to minimize patient manipulation if trauma to the vertebral column is suspected; in these cases, neurological assessment may be more limited for the patient's safety.

The modified Glasgow coma scale (MGCS) is used to predict prognosis and monitor progress as treatment is implemented. There is little information regarding use of the scale in felines, though it is clinically helpful and can be an objective tool for evaluating trauma patients. The small animal coma scale (SACS) is also used and has been adapted from the MGCS to better fit veterinary patients. The MGSC measures three categories: level of consciousness, motor function, and brainstem reflexes. Lower scores in each category are indicative of more severe the neurologic disease, and thus are associated with a worse prognosis. Patients can be re-evaluated every 30 minutes to monitor for response to therapy. Level of consciousness is an important component of grading head trauma and can be classified as normal, depressed, obtunded, stuporous, or comatose. Stuporous animals are unconscious but react to noxious

stimuli, and comatose animals do not respond to any stimulus (6, 9, 13). Altered levels of consciousness indicates dysfunction of the cerebral cortex and/or brainstem reticular activating system, with comatose animals often having global cerebral damage or severe brainstem damage. Motor function may help localize a brain injury if a decerebrate or decerebellate posture is seen. Brainstem reflexes are assessed by evaluating pupil size, shape, and responsiveness to light. It is also important to rule out primary ocular injury as a potential cause of any abnormalities seen. Miotic pupils indicate cerebral injury, while mydriatic pupils indicate brain herniation and requires immediate intervention. A decreased or absent oculocephalic reflex indicates a brainstem lesion and is important to assess, even in an unconscious animal (9).

Prior to pursuing advanced imaging, thoracic and abdominal radiographs should be performed to evaluate other systemic injuries (10). Computed tomography (CT) is often the initial imaging method of choice for head trauma patients, particularly in an emergency setting as image acquisition is rapid and does not require the patient to be anesthetized (6, 9). Though MRI is more sensitive in detecting subtle parenchymal damage or intracranial hemorrhage, it requires general anesthesia due to much longer image acquisition time compared to CT (8). Patients who have recently undergone trauma are often not suitable anesthetic candidates. CT is also helpful for evaluating the rest of the body for other injuries, and it is superior for evaluation of bone pathology compared to MRI. CT and MRI are both adequate modalities to help determine whether or not surgery is indicated in patients with traumatic brain injuries.

Treatment and Management:

The first steps in treating a patient with a traumatic brain injury are to address any life-threatening systemic or extracranial abnormalities, particularly hypovolemia, hypotension, and hypoxemia, as these play a large role in the development and propagation of secondary brain

injury (5, 9, 13). Fluid therapy to correct hypovolemia should be one of the first steps in treating the extracranial disease involved in a traumatic brain injury. Isotonic crystalloids should be administered first, and colloids may be added if additional pressor support is needed. Dextrose should be avoided, as hyperglycemia is associated with worsened outcomes with traumatic brain injuries (9).

Oxygen supplementation should be provided if the pulse oxygenation (SpO_2) is less than 95%. Mechanical ventilation should also be considered if the patient cannot maintain a $PaCO_2$ less than 45 mmHg (9, 13). The patient's head should be elevated to 30 degrees to maximize venous drainage and arterial delivery and further decrease intracranial pressure (10). If a seizure occurs, anti-epileptic treatment should be initiated. Levetiracetam is suggested, as it does not significantly depress mentation and thus allows for better serial neurologic evaluation. Studies in the human literature have shown that prophylactic anti-epileptic therapy may help to reduce the incidence of early seizures (occurring during the first seven days after injury) but do not appear to aid in the prevention of late seizures (occurring greater than seven days after the injury) (9, 10). Corticosteroids are contraindicated in patients with traumatic brain injuries, as they contribute to hyperglycemia and have been associated with increased morbidity and mortality (5).

Following initial systemic stabilization, hyperosmolar therapy should be administered to help decrease intracranial pressure. Mannitol is an osmotic diuretic that increases cerebral blood flow, reduces cerebral edema, and acts as a free radical scavenger, however patients must be euvolemic at the time of administration (5, 9, 10). There has been concern in the past about using mannitol in patients with intracranial hemorrhage, however there is currently no evidence supporting this concern (10). Hypertonic saline may be used in hypovolemic patients to cause

volume expansion, though it should be cautiously used in hyponatremic animals. It also has positive inotropic effects (5, 9, 10).

Therapeutic hypothermia has been used in treatment of humans with traumatic brain injuries with increased intracranial pressure or in status epilepticus. Studies have shown that cooling the body to 90 to 95 degrees F is associated with increased survival and improved neurologic outcomes. Hypothermia can help mitigate the development of secondary brain injury by decreasing cerebral metabolism, reducing inflammation, and decreasing the production of excitatory neurotransmitters. It may also help to reduce seizure activity. Risks of therapeutic hypothermia include arrhythmias, coagulopathies, hypovolemia, and an increased susceptibility to infection (11). One report described the use of therapeutic hypothermia in a canine patient with a traumatic brain injury with a successful outcome (7).

Decompressive surgery is indicated when a space-occupying mass, such as an extra-axial hematoma, or depressed skull fracture is present in order to reduce intracranial pressure. Surgery should also be considered in patients who do not respond to medical therapy. In human medicine, surgery is a mainstay of therapy for many traumatic brain injuries, but it has been uncommonly used in veterinary patients. Because CT is becoming more readily available in veterinary medicine, the need for decompressive surgery is becoming increasingly recognized and utilized (5, 6, 9, 10).

Nursing care for all patients with traumatic brain injury is imperative. Adequate analgesia is necessary, and opioids are the medication of choice as they have limited cardiovascular effects. The most important concern is whether or not the gag reflex is intact so that the patient can protect its airway and avoid aspiration pneumonia. Patients should be evaluated regularly for adequate pain control and the ability to swallow (9, 10, 13).

Gastrointestinal protectants are commonly used in these patients to reduce the likelihood of forming gastric ulcers, and omeprazole also has the added benefit of reducing cerebrospinal fluid production in dogs through a poorly understood mechanism (13). Nutritional support should also be provided to these patients early in the course of treatment. Enteral nutrition is preferred, though feeding tubes are commonly placed to facilitate feeding of patients who are unable to swallow. One human study showed that traumatic brain injury patients receiving early enteral nutrition had reduced infection rates by 55% (13).

Patients with traumatic brain injury are often recumbent for several days, necessitating intensive nursing care to avoid associated complications. They must have thick, clean, dry bedding and require turning at least every four hours to prevent the formation of pressure sores. Bladders should be evaluated every six hours while recumbent and expressed as needed. Eyes should be lubricated and evaluated frequently for formation of corneal ulcerations. Passive range of motion exercises should be performed several times daily, as patients with prolonged recumbency are at significant risk for muscle atrophy and limb contractures (5).

Prognosis for patients with traumatic brain injury is extremely variable, and there is very little information regarding prognostic indicators in animals. Though the modified Glasgow coma scale is able to give an idea of severity, it has not been validated for measuring prognosis in cats. It is known that being comatose, decerebrate posture, and clinical evidence suggestive of brain herniation carry a guarded to poor prognosis (13). Animals are able to handle losing cerebral function fairly well, though concurrent injuries must also be taken into account for prognostication. Each patient must be evaluated individually, and patients who receive early and aggressive treatment are more likely to have a better outcome than those who do not.

Case Outcome:

Malfoy recovered uneventfully after surgery. He was treated with mannitol post-operatively to help decrease intracranial pressure, and his head was elevated at approximately 30 degrees. He was initially maintained on a remifentanil CRI, prednisolone, Clavamox, Cerenia, pantoprazole, and levetiracetam. The morning after surgery, Malfoy had improved from stuporous to obtunded and was remarkably more alert than before surgery. He was ambulatory, though he was moderately tetraparetic and ataxic and made wide circles to the left. His postural reactions were normal in the left thoracic and pelvic limbs but absent on the right side. His muscle tone was still normal to increased, and his cutaneous trunci reflex remained absent. Malofy continued to improve throughout the day and was transitioned from remifentanil to buccal buprenorphine. His ataxia slowly improved, he began grooming himself, urinating in the litter box, and eating on his own, so the Cerenia and pantoprazole were discontinued. He was eating well but unable to drink water on his own, so water was provided though his esophageal feeding tube. Over the next several days in the hospital, Malfoy's neurologic status continued to improve daily, and his nasal discharge completely resolved. On October 29th, day 8 of his hospitalization, Malfoy was discharged. At that time he was bright, alert, and strongly ambulatory, though he remained mildly ataxic and occasionally circled to the left. His postural reactions remained delayed on the right side but normal on the left, and his cutaneous trunci reflex returned to normal. His menace response, palpebral reflex, and visual tracking remained absent bilaterally, though he did appear to be regaining some vision while being observed to navigate a room. He did not return for a follow-up visit, but he did present to his primary veterinarian two weeks after discharge from MSU for removal of his esophageal feeding tube, as he was eating and drinking well on his own. Malofy's owners reported that he was doing very well at home, and they appreciated continuous improvement in his vision.

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