Spinal Trauma Drama

Management of Acute Spinal Cord injury in a Dog

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Introduction

Management of acute spinal cord injury can be quite challenging; however, it is not impossible. Currently, there is no standard treatment for patients with acute spinal cord injury, therefore each patient needs to be individually assessed and treatment should be tailored to meet their specific needs. It is critical for veterinarians to understand the basics of acute spinal cord injury in order to initiate appropriate therapy. Acute spinal cord trauma is characterized by alterations of spinal cord architecture and homeostatic mechanisms and usually results in edema, hemorrhage, ischemia, and loss of function. The spinal cord is composed of a centralized grey matter surrounded by white matter. The central grey matter houses the nerve cell bodies and therefore has a higher metabolic rate and greater demand for glucose and oxygen than the surrounding white matter. These greater demands within the grey matter therefore require an increased blood supply compared to the surrounding white matter. It is reported that the blood supply within the grey matter exceeds that of the white matter by 5:1. (1, 5) The characteristics of the grey matter make it much more susceptible to injury. (5)

Causes of acute spinal cord injury include vehicular injury, falls, bite wounds from dog fights, missile injury from gun shot wounds, Hanson type 1 intervertebral disk disease, and fibrocartilaginous embolism. The most common of these causes is vehicular injuries with one study reporting 88% of spinal cord injury cases observed were due to vehicular trauma. The other 12% in that study were from falls and dog fights. (1, 2)

History and Presentation

Junie Bakhaus is an 8 year-old, spayed female mixed breed dog who presented to MSU-CVM Emergency Service on May 28, 2017. It was reported that on previous night, May 27, 2017, the owner left on a boat around midnight. When owner returned to the dock a few hours later, Junie could not be found. Her owners searched for her around the lake all night, and finally found her around noon on

Sunday lying in the water near the shore. She had a large deep wound over her hind end, presumably due to trauma from a boat propeller. The wound was covered in mud and debris, and owner believed they could visualize a separated spinal column. At that time, Junie was taken to an emergency veterinary hospital in Tuscaloosa, AL.

Upon presentation to the rDVM, a physical exam revealed a large 6 inch deep and 7 to 8 inch long wound noted just cranial to the tail. Junie had pale mucus membranes, with a capillary refill time (CRT) of 3 seconds. Thoracic auscultation was within normal limits, but she was tachycardic and had a slight increase in respiratory effort. Deep pain was present in all four limbs, but was negative in the tail. She also had no anal tone. Her cutaneous trunci reflex was present from the cranial edge of the wound forward. Junie was noted to be shivering, so heating pad was placed on top of her. After successful catheter placement, 1 liter of 0.9% sodium chloride was bollused followed by a second liter with 45 milliliters of lidocaine added. Junie also received buprenorphine and Baytril intravenously (IV). The wound was flushed and initially debrided and tissue samples were taken for culture. Due to the severity of her injuries, Junie was referred to MSU-CVM for further evaluation and treatment. Her wound was then packed with lap sponges and horizontal mattress sutures were placed to secure her wound prior to transport.

Junie presented to MSU-CVM Emergency Service at approximately 9 p.m. Sunday night, May 28, 2017. Upon presentation, Junie was secured to back board in right lateral recumbency to prevent any further trauma. At this time, she was quiet, alert, and responsive. Her temperature and pulse were within normal limits; however, she was tachypneic with a respiratory rate of 120 breaths per minute. Her mucus membranes were pale/pink and tacky. Her CRT was approximately 2 seconds which showed mild improvement. She was estimated to be 6-8% dehydrated. Non-invasive blood pressure showed a systolic pressure of 138 mmHg, a diastolic pressure of 78 mmHg, and a mean arterial pressure of 98

mmHg. There was a large, approximately 30 cm full thickness wound positioned transversely over the dorsal pelvic region, with 4 vertical mattress sutures in place to appose the skin edges. Lap sponges were visible beneath the skin, packed into wound. There was a palpable step that felt consistent with soft tissue swelling over the cranial lumbar spine, but no superficial wound was noted in this area. There was also a smaller, approximately 2 cm, full thickness wound through skin and muscle over the distolateral aspect of left humerus. There were no palpable long bone fractures in that area. During the initial stabilization, Junie had intermittent ventricular premature contractions (VPCs), but did not have any dropped pulses.

Clinicians then performed a thorough neurologic exam. Because Junie was secured to backboard in right lateral recumbency, her gait and postural reactions were not assessed, but strong voluntary motor activity was observed in all 4 limbs during recumbency. All cranial nerves were assessed and found to be normal. Withdrawal reflex in all 4 limbs was intact. Triceps, biceps, and extensor carpi radialis reflexes were intact left forelimb. Patellar, gastrocnemius, sciatic, and cranial tibial reflexes were intact left pelvic limb. Right forelimb and pelvic limb spinal reflexes were unable to be fully assessed due to the patient being strapped to backboard in right lateral recumbency. Anal tone was absent and the anus was gaping open. Perineal reflex was also absent. She was deep pain negative in her tail, but reacted when her tail was lifted dorsally. Cutaneous sensation was absent caudal to the level of the cranial edge of the wound.

A Complete Blood Count (CBC) and serum chemistry were performed late that night to look for any hematologic or electrolyte derangements. The CBC showed she had a low PCV, high number of segmented neutrophils, and low numbers of lymphocytes. This was most likely due to a stress leukogram. Her serum chemistry panel showed a moderate hyponatremia, a mildly low CO2 and low anion gap, a moderate hyperglycemia, a mild to moderate hypoproteinemia and hypoalbuminemia, a

mild hypocalcemia, a mild to moderately low calculated osmolality, a moderate hypomagnesemia, and a high CK.

Due to Junie's traumatic history and neurologic deficits, multiple radiographs and a CT study were performed in order to determine the extent of Junie's spinal cord involvement to better assess prognosis and treatment options. Based on physical and neurologic examination along with diagnostic imaging, Junie was diagnosed with acute spinal cord injury, soft tissue damage with foreign bodies present, and pneumoperitoneum.

Pathophysiology

The pathophysiology of acute spinal cord injury is divided into primary and secondary injury. The initial traumatic event is categorized as primary injury and is described as physical disruption of the cell membranes of neuronal and glial cells within the spinal cord that occurs as a direct result of the traumatic event. Physical disruption of the spinal cord architecture results in hemorrhage, herniated disk material, and displaced or fractured vertebrae. The 6 forces involved in primary injury include compression, concussion, contusion, shearing, laceration, and distraction. (2, 3, 4) Compression is due to an abnormal amount of pressure placed on the spinal cord. Compression affects spinal cord perfusion by occluding venous drainage and preventing arterial flow. Concussion occurs when there is a rapid period of acceleration followed by deceleration by some external force. Contusions occur due to vascular hemorrhage within the spinal cord. Shearing forces occur when the parallel plane of the spinal cord is forced in a perpendicular direction. Laceration refers to the physical tearing of the spinal cord tissue and distraction is due to abnormal stretching within the axial plane of the spinal cord. (4)

Secondary injury is quite extensive and encompasses all biochemical process that occur after the initial traumatic event. Factors that contribute to secondary injury include loss of autoregulatory mechanisms, excessive release of excitatory neurotransmitters, increases in intracellular calcium,

production of reactive oxygen species, and the inflammatory process that occurs in response to acute spinal cord injury. (3, 5) The spinal cord has autoregulatory mechanisms in place to compensate for changes in systemic blood flow in order to maintain perfusion. Spinal cord injury results in a loss of these autoregulatory mechanisms which results in the inability to maintain perfusion and leads to ischemia. Spinal cord injury patients also experience systemic hypotension which further compounds ischemia. The progressive decline of perfusion in the grey matter will cause ischemic infarction and subsequent necrosis within the first 24 hours. Therefore, it is critical to monitor systemic blood pressure in these patients and support with appropriate IV fluids as needed to maintain perfusion. (5, 7)

One of the primary excitatory neurotransmitters of the central nervous system is glutamate. Glutamate is directly excitotoxic to the spinal cord and extracellular increases in glutamate leads to production of reactive oxygen species. Excessive release of glutamate occurs due to damaged neuron leakage and depolarization induced release. In normal circumstances, astrocytes will reuptake glutamate via ATP; however, during spinal cord injury, the ischemic and hypoxic conditions result in an inadequate amount of ATP. Activation of the glutamate receptors causes an influx of sodium into cells. However, with depletion of ATP, the sodium-potassium-ATPase pump is unable to pump sodium out of the cell resulting in sodium accumulation and cytotoxic edema. (3)

Increases in intracellular sodium also activates the sodium-calcium ion exchanger which leads to increasing intracellular calcium levels. Intracellular calcium level are furthermore increased via the release of calcium from intracellular organelles and decreased calcium efflux of the ATP dependent pumps. High levels of intracellular calcium lead to the production of reactive oxygen species, apoptosis, and programmed cell death. (3, 6, 7) Reactive oxygen species cause direct cell membrane damage to glial, neuronal, and endothelial cells via lipid peroxidation. Reactive oxygen species also inhibit mitochondrial respiration which perpetuates ischemia. Studies show that peak production time of

reactive oxygen species occurs within the first 12 hours of injury and levels remain elevated for at least a week. The inflammatory process that occurs results in neutrophilic infiltration and release of inflammatory mediators. Neutrophilic inflammation causes parenchymal damage via the release of proteolytic enzymes. Inflammatory mediators such as tumor-necrosis-factor and interleukin-1β will increased permeability of the spinal cord barrier thereby facilitating neutrophilic inflammation. (2, 6, 7)

Diagnostic Approach/Considerations

Before any diagnostic procedures are initiated, all acute spinal cord injury cases should be appropriately triaged and stabilized with any life threatening injuries addressed first. Initial stabilization of the patient takes priority over primary spinal cord injury since hypotension and hypoxemia can further contribute to secondary injury. Therefore, it is critically important to monitor and maintain adequate systemic blood pressure and oxygen delivery. (5) Immobilization is a key factor in initial stabilization if vertebral fractures are suspected to prevent any further primary injury. After stabilization, a thorough neurologic examination should be performed and should include assessment of mentation, gait (when possible), cranial nerve function, postural reactions, spinal reflexes, and nociception in the limbs. (3) If a spinal fracture is suspected and the patient is immobilized, a full neurologic examination may not be possible and can be abbreviated. It is important to remember that the initial neurologic examination should be performed before administration of pain medications in order to accurately assess neurologic function. (2)

Current literature describes 3 accepted scoring systems that can be used when assessing patients with spinal cord injury and includes The Modified Frankel Score, The 14-Point Motor Score, and The Texas Spinal Cord Injury Score. The modified Frankel Score was derived from a human scoring system and has been used in veterinary medicine to evaluate the pelvic limbs in patients experiencing thoracolumbar spinal injury. This scoring method can also be used to evaluate all 4 limbs. In contrast,

The 14 Point Motor Score only assess functionality of the pelvic limbs. The Texas Spinal Cord Injury Score is the most recently developed method and evaluates each limb individually for gait, postural reactions, and nociception. (3, 14)

Any trauma related patient should have thoracic and abdominal radiographs performed in order to rule out pulmonary contusions, pneumothorax, pneumoperitoneum or any other possible trauma related injuries. If other imaging modalities such as computed tomography (CT) or magnetic resonance imaging (MRI) cannot be performed, radiography of the spine can be used to detect abnormalities; however, radiography alone has a relatively low sensitivity for detection of vertebral fractures with one study reporting a 72% sensitivity rate for vertebral fracture detection and a 77% sensitivity rate for detection of subluxations. Orthogonal radiographs should be performed in cases for which CT or MRI are not an option, and the entire spinal column should be imaged since approximately 20% of patients that present with spinal trauma have multiple sites of injury. Due to low sensitivity, vertebral fractures or luxation cannot be ruled out in normal appearing radiographs. (4)

If vertebral fracture or luxation is suspected, CT should be performed as it is the superior imaging modality for bone. CT can also be advantageous in that it is relatively fast compared to MRI; therefore, sedation can be used and general anesthesia is not required. (4) CT can be combined with myelography in order to increase sensitivity. MRI is superior for evaluation soft tissue structures which include spinal cord parenchyma, intervertebral disks, and spinal nerve roots. It is advantageous in that it has the ability multiple planes. It is also more costly compared to CT and is relatively time consuming thereby requiring general anesthesia. (4)

Treatment and Management

Surgical intervention is targeted at the effects of the primary injury such as relieving compression of the spinal cord, while medical treatment is targeted at limiting the amount of damage

due to secondary injury. Since secondary injury begins immediately after primary injury occurs, medical management to address the secondary injury should be initiated as soon as possible. (3, 5)

The use of corticosteroids has been highly controversial in spinal cord injury patients. The rationale of administration of a steroid relates to the free radical scavenging properties, anti-inflammatory effects, and preservation of blood flow. The most researched corticosteroid to date is methylprednisolone; multiple human and animal studies have shown minimal to no benefit when given within the first 8 hours of injury. Adverse effects of high dose corticosteroids include increased risk of gastrointestinal ulceration, immunosuppression with increased risk of sepsis, delayed wound healing, and compromised renal perfusion in hypovolemic patients. Due to these adverse effects and lack of significant benefits, newer literature is not recommending the use of corticosteroids in acute spinal cord injury patients. (3, 4, 8)

Vitamin E and selenium have been studied due to their natural antioxidant effects. Antioxidative benefits have been reported in experimental models; however, these products require a lengthy time period of administration in order to reach therapeutic concentrations. This limits their clinical effectiveness for spinal cord injury, especially during the acute phase; therefore, vitamin E and selenium are not routinely used for spinal cord injury patients (3, 4)

Since increasing intracellular levels of calcium greatly contributes to secondary injury, the role of calcium channel antagonists has been studied as a potential treatment option. However, there are currently no clinically proven treatment options available at this time. Human studies using calcium channel antagonists have failed to show a benefit. Also, the complications and side effects of calcium channel antagonists involving cardiac muscle and vascular tone limits their potential and clinical use. (1, 2)

More recently, polyethylene glycol (not to be confused with ethylene glycol) has been studied due to its ability to fuse membranes. Polyethylene glycol is a fusogen, or a hydrophobic polymer, that targets damaged membranes and attempts to restore the damaged area. Polyethylene glycol has surfactant properties that allow it to help repair the damaged membranes and prevent further ion leakage. (9) A phase 1 clinical trial using polyethylene glycol has been completed in dogs with paralysis which were deep pain negative. This study reported that 60% of those patients recovered function; however, efficacy of this treatment strategy still needs to be established as that study was not blinded. (2, 4)

One interesting study reports transplanting olfactory glial cells in dogs with spinal injury. In this study, glial cells were taken from an olfactory bulb and transplanted to the site of injury via dorsal laminectomy. This study showed promising results with 7 out of 8 patients with no motor function, did have some improvement. There was also one patient in that study who was deep pain negative and regained sensation in that limb. The benefits reported warrant further research. (3)

Veterinarians should be able to determine if a patient warrants surgical treatment based on exam findings and imaging. Indications for surgical stabilization include minimal voluntary motor function or complete paralysis, clinical or radiographic evidence of instability, and progression of neurologic signs despite appropriate non surgical methods. The three compartment theory allows assessment of spinal fractures and whether or not surgical intervention is required. The three compartments include a dorsal, middle, and ventral compartment. The dorsal compartment includes articular processes, laminae, pedicles, and spinous processes. The middle compartment includes dorsal longitudinal ligament, dorsal annulus fibrosis, and the dorsal aspect of the vertebral body. The ventral compartment includes the remaining vertebral body, the lateral and ventral aspects of the annulus fibrosis, the nucleus pulposus, and the ventral longitudinal ligament. Surgery is indicated if there is

disruption of more than one compartment, or if the ventral compartment alone compromises stability. If only the dorsal or the middle compartment is compromised, surgery may not be necessary and external coaptation can be utilized. (5) The most commonly utilized surgical options include a hemilaminectomy or a dorsal laminectomy for decompression, open reduction and internal fixation, or external fixation for vertebral fractures.

Whether or not surgery is elected, a major component of treatment in patients with spinal cord injury is supportive care. Appropriate analgesic therapy is essential for proper supportive care since uncontrolled pain can have detrimental cardiovascular and gastrointestinal effects such as tachycardia, hypertension, increased cardiac workload, vomiting, nausea and ileus. It is important to monitor the patient's pain level throughout the course of treatment to ensure adequate analgesia is being provided.

It is also important to provide high quality bedding with thick blankets and soft surfaces to prevent pressure ulceration as many of these patients are recumbent. Other potential complications related to recumbency include urine scalding, fecal soiling, limb contraction and aspiration pneumonia. Patients that are recumbent should be turned at least every 4 hours to aid in prevention of congestion and atelectasis of the dependent lung lobes. Recumbent patients are also at risk for muscle atrophy and limb contracture due to disuse. Physical therapy exercises such as passive range of motion can help reduce these risks. Performing passive range of motion exercises in these patients can help prevent limb atrophy or contracture. (3, 4)

Nutritional support is vital to the recovery of these patients. Their increase in catecholamine and cortisol levels can place them in a catabolic state. Human spinal cord injury studies have found that patients undergo hypermetabolic state; therefore, early feeding is encouraged. Studies have not been performed in animals to document this; however, we know that injured or sick animals are at risk of malnutrition due to their refusal to eat. Of course, enteral nutrition is preferred over parenteral

nutrition since this method preserves the gastrointestinal function. Nasogastric or nasoesophageal tubes may be utilized if patients continue to refuse to eat. (3)

Another important factor when managing patients with acute spinal cord injury is appropriate bladder care. Urinary dysfunction is common in these patients which requires special attention and supportive measures to prevent complications such as bladder distention, bladder atony, urinary tract infections, and urine scalding. Patients with bladder dysfunction may require manual expression, intermittent catheterization, or indwelling urinary catheterization to facilitate micturition. (3, 10)

The prognosis is highly dependent on the type and extend of the injury. Also, the prognosis is ultimately based on the patient, and not diagnostic images obtained. It is important when assessing these patients, not to solely rely on imaging, but to perform a thorough neurologic exam throughout therapy to evaluate their response. Studies show that dogs that are deep pain negative after 24 hours have only a 5-10% chance of regaining function. This statistic again demonstrates the importance of starting treatment as soon as possible. Patients who can sense deep pain in the digits have a much better prognosis. (1, 3, 5)

Case Outcome

After treatment plan and costs were discussed with Junie's owners, Junie was rushed into emergency surgery where a celiotomy was performed. The abdomen was explored to identify any abnormalities. A hematoma was found in the left retroperitoneal space, but was left to be reabsorbed by the body since no active bleeding or body wall perforation was found. After no other abnormalities were found, the abdomen was then closed and Junie was repositioned in sternal recumbency for a tail amputation. The large wound just cranial to the tail head was thoroughly debrided using Metzenbaum scissors and a #10 blade. The caudal vertebrae were dissected free from the surrounding tissues using Metzenbaum scissors and monopolar electrocautery until the tail was pulled through the caudal

incision. A vacuum assisted closure system was placed in standard fashion within the wound site. A VAC system is essentially a piece of sterile foam that is placed into the wound site and is connected to a vacuum that is constantly applying negative pressure to the wound area. The negative pressure has been found to decrease tissue edema and remove excess fluid produced from the wound, thereby facilitating a more favorable wound environment. (12, 13) The vacuum can also remove bacteria from the wound that may be present within the wound site. In addition, the applied negative pressure promotes increased blood flow resulting in a more rapid formation of granulation tissue. Vacuum-assisted bandages can be left on for 48-72 hours resulting in decreased wound management time and costs as opposed to having to perform daily bandage changes. (11)

After surgery was complete, Unasyn, Baytril, and metronidazole were started IV to provide four quadrant antibiotic coverage and address any potential infection within Junie's wound. Morphine and ketamine were administered as constant rate infusions (CRI) to provide adequate analgesia and were discontinued three days post operatively. At that time, oral Tynelol 4 was initiated for pain control. Junie was exhibiting multiple premature ventricular contractions (PVC) during and after surgery; therefore, a lidocaine CRI was given. Two days post operatively, Junie was exhibiting a normal sinus rhythm and no more PVCs were seen, therefore the lidocaine CRI was discontinued.

Four days post operatively, the VAC system was removed under sedation to assess healing. Overall, the wound size had significantly decreased and there was new granulation tissue present. There was still a moderate amount of dead space present so the VAC system was replaced within the wound. Seven days post op, the VAC system was changed again and a culture of the wound site was taken. Results of the culture showed growth of *Citrobacter freundii, Aeromonas hydrophila,* and *Streptococcus canis*, all of which were not completely susceptible to our chosen antibiotics. Due to these results, Baytril, metronidazole, and Unasyn were discontinued, and cefpodoxime was initiated. That same day,

Junie's urinary catheter was removed to assess whether or not she could urinate on her own. She was prescribed bethanecol and prazosin to facilitate urination and manual expression of her bladder was performed multiple times per day. Ten days post operatively, a third VAC change was performed. It was noted that the dead space had not decreased and there was no new granulation tissue present. Therefore, the wound was thoroughly lavaged and a new VAC system was placed on top of her wound. Another culture was taken at that time. The following day, Junie was showing signs of SLUD and was hypersalivating and had severe diarrhea; therefore, bethanecol was discontinued. Throughout the day, hypersalivation decreased and her diarrhea began resolving. Also that day, our culture results taken during the previous VAC change were completed and showed heavy growth of *Enterobacter cloacae, Pseudomonas fluorescens,* and *Enterococcus faecalis* that were resistant to multiple drugs. At that time, Junie was started on amikacin to address this issue. A couple days of later, a leak was noted in the VAC system. At that time, the VAC was removed, her wound was debrided and flushed, and a Jackson Pratt (JP) drain was placed. Each following day, fluid produced from the wound was decreasing. Two days later, the drain was ultimately removed and Junie's wound was closed. Her wound continued to heal, and on June 21, 2018, after spending 25 days in the hospital, Junie was discharged!

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