

Under Pressure:

A compressive spinal cord tumor in a canine patient

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Introduction

Compressive spinal cord neoplasia can cause significant neurological deficits and pain in dogs. Overall spinal cord tumors are uncommon, with the most common signalment being middle-aged to older, large breed dogs.² The most common type of primary neoplasm affecting the spinal cord of dogs are meningiomas, but sarcomas and carcinomas can also occur.^{1,5} Tumors affecting the spinal cord can involve the spinal cord, meninges, nerve roots, and the surrounding tissues. Spinal cord masses are classified based on the area of the spinal cord from which they arise. Extradural tumors are located outside of the dura. Intradural/extramedullary tumors originate from within the dura but remain outside of the spinal cord. Finally, intramedullary tumors originate from within the spinal cord itself. Additionally, spinal cord tumors are classified as primary (arising from the central nervous system) or secondary (a result of metastasis) . Primary spinal cord tumors are more commonly reported than secondary tumors.³ Classifying spinal cord tumors can help identify the most likely tumor types as well as affect treatment and prognosis.¹

Malignant spinal cord compression occurs as tumors grow within the spinal canal. Clinical signs associated with spinal cord compression vary depending on the location of the lesion and the rate of tumor growth. Signs can include, ataxia, paresis, plegia, postural deficits, and hyperaesthesia. These signs are usually chronic and slowly progressive.^{3,6} Lesions can be localized based on a neurological exam, but clinical signs cannot differentiate spinal cord masses from other compressive diseases. Advanced imaging, such as CT or MRI is needed to diagnose spinal cord tumors. When both imaging modalities are available, MRI is preferred to CT. MRI provides superior contrast resolution and a higher sensitivity for detecting intradural tumors when compared to CT.⁴

History and Presentation

Bella is an eight year old spayed female Yorkshire Terrier who presented to the MSU-CVM Veterinary Specialty Center on 11/20/2019 for evaluation of a cervical tumor found on referral CT. Bella began showing clinical signs of hiding and decreased activity in late September. These signs continued to worsen through October and she became increasingly painful in her cervical region. She was prescribed prednisone (1mg/kg) by her primary care veterinarian. In early November, she began to show signs of ataxia, pain, lethargy, weakness, anorexia, and a right sided head tilt. On 11/15/19 Bella's signs became significantly worse and she was taken to a specialty center where a CT scan and CSF tap were performed. She was subsequently referred to MSU-CVM for a neurology consultation. At the time of referral, her medications included tramadol, hydrocodone, prednisone, robaxin, pepcid, and CBD oil.

On presentation, Bella was bright, alert, and responsive. Her body condition score was 5 out of 9 (4 being ideal) with a weight of 4.2kgs. She had a temperature of 101.2 degrees Fahrenheit. She was normocardic with a heart rate of 128 with no murmurs or arrhythmias heard on auscultation of the heart. Her heart and lungs auscultated normally, and she was panting. She had significant dental disease with no evidence of masses or debris. Her lymph nodes palpated soft and symmetrical. There was no debris or discharge of the eyes, ears, or nose. No pain was elicited on abdominal palpation. A capillary refill time of less than two seconds was obtained and her gums were pink and moist.

On neurological examination, Bella displayed obvious signs of pain. She was ambulatory tetraparetic. She displayed anisocoria, with the left pupil being smaller than the right. The remaining of her cranial nerve examination was within normal limits. On postural reaction

evaluation, she had absent proprioceptive placing in the left forelimb, and diminished placing on the left hindlimb. Her postural reactions on the right side were normal. On segmental reflex examination, she had normal integrity of the sensory and motor components of all reflex arches. On cutaneous trunci examination, she had normal contraction of the cutaneous trunci muscles bilaterally. She had normal muscle tone and no evidence of atrophy on palpation. Severe pain was elicited on cervical palpation. The lesion was localized to C1-C5, with possible multifocal involvement.

Diagnostic Approach

A CT scan performed prior to referral revealed a moderate to severely compressive intradural extramedullary mass at the level of C1-C2. A CSF tap revealed an elevated nucleated cell count of 112 cells/uL (0-4 cells/uL). Upon arrival to MSU-CVM, full staging was performed that included thoracic radiographs, abdominal radiographs, and an abdominal ultrasound. Aspirates of the spleen and liver were obtained for cytology. No evidence of metastasis was noted and it was determined that Bella had a primary spinal tumor.

Next, an MRI was performed to further evaluate the mass for surgical planning. The MRI revealed a large mass extending from the cranial aspect of C1 to the caudal aspect of C2. At its widest point, the mass filled about 90% of the spinal canal. The mass extended through the C1-2 intervertebral foramen on the left and extended outside the left vertebral body of C2. This mass was classified as intradural/extramedullary.

Pathophysiology

Compressive spinal cord tumors cause direct distortion to the structure of the spinal cord tissue, as well as vascular and metabolic changes. As these tumors grow, they cause disruption of blood flow and ischemia of the spinal cord. Ischemia of the cord leads to complex biochemical

changes within the affected cells. Free radical production causes cell death and production of inflammatory cytokines.⁶ Spinal cord tumors cause direct damage by neoplastic infiltration, as well as indirect damage via compression and ischemia. Direct damage to the spinal cord is irreversible, even following decompressive surgery.⁴ After decompression, inflammatory cells such as lymphocytes and macrophages infiltrate the damaged area. Next, fibroblasts lay down scar tissue and local astrocytes proliferate. Damaged spinal cord axons regrow to reach the edges of the scar tissue. Although axons cannot be regenerated, if enough axons remain intact, acceptable motor function can be achieved.^{1,6} Immediately following decompression, many patients show decreased proprioception and motor function. This is due to reperfusion injury and iatrogenic damage to the spinal cord during surgery. Reperfusion injury occurs when blood supply returns to tissue after a period of ischemia, resulting in inflammation and oxidative damage. This is usually temporary, and returning function can be noted by three days post-operatively.⁴ Additionally, hyperalgesia is a common postoperative complication that occurs due to changes in neuronal sensitivity. These changes occur at both the level of peripheral receptor (peripheral sensitization) and at sites in central nervous system (central sensitization).⁵

Damage to the ascending and descending white matter tracts results in proprioceptive and motor deficits. Due to redundancy in these tracts, a large portion of the spinal cord must be affected before motor function is significantly impaired.⁵ In the cranial cervical spine, damage to the spinal cord results in upper motor neuron deficits to all four limbs. This manifests as tetraparesis or tetraplegia, normal to increased reflexes, and normal to increased muscle tone in all four limbs. Respiratory compromise can also occur with lesions affecting the C3-C5 vertebrae. These signs can help localize the lesion to the C1-C5 spinal cord segment.⁶

Treatment and Management

Treatment of spinal cord neoplasia includes a combination of surgery, radiation and chemotherapy to slow the rate of local tumor growth and metastasis.^{2,6} Due to anatomical constraints, complete tumor resection is usually not attainable, but surgical debulking can relieve pain, improve clinical signs and prolong life. Additionally, surgery allows for biopsies to be obtained for a definitive diagnosis. Identifying the tumor type allows for a more tailored treatment as well as a better understanding of prognosis.⁶

In Bella's case, a left sided hemilaminectomy was performed at the level of C1-C2. The extra-medullary portion of the mass was visualized to the left of C2 and was debulked with a combination of cautery, blunt and sharp dissection. Samples of the extradural mass were submitted for histopathology. Kerrison rongeurs were used to create a window in the vertebrae. The bone removed was noted to be fragile and thin. The dura was visualized and noted to have an abnormal appearance with a dark, mottled surface and visible blood vessels. Next, a duratomy was performed. Upon incision of the dura, the mass mildly extruded out of the confines of the dura. The dura was submitted for histopathology. The mass was carefully dissected with a combination of cautery and blunt dissection. Samples of the intradural mass were submitted for biopsy. Ventral to the mass, normal looking spinal cord could be identified. The site was copiously lavaged with sterile saline and was routinely closed. Bella recovered from anesthesia uneventfully.

Case Outcome

Following surgery, Bella was non-abulatory tetraparetic, with her left side being more affected. She was maintained on a fentanyl constant rate infusion for pain control. Three days following surgery, Bella began to attempt to walk and was able to ambulate unassisted five days post-operatively. Biopsies of the mass were submitted for histopathology, which revealed an

anaplastic sarcoma. The cells were extremely undifferentiated, which made it difficult to determine their origin. The tumor appeared aggressive with high numbers of mitotic figures and invasion into surrounding tissues. Additional stains were sent out to further characterize the tumor. The additional stains were also compatible with anaplastic sarcoma. Radiation therapy was recommended due to the aggressive nature of this tumor.

Bella was discharged 5 days after her surgery. At the time of discharge, she was ambulatory tetraparetic with pain that was moderately controlled. Because the tumor was not completely resectable, Bella began a definitive radiation protocol at Auburn University two weeks following surgery. She represented to MSU-CVM on January 15, 2020, seven weeks postoperatively for a recheck appointment with the Oncology and Neurology departments. At this time her condition had significantly declined. She was non-ambulatory and exhibited cranial nerve II deficits. It was suspected that the tumor had metastasized to the forebrain. At this time, humane euthanasia was recommended due to Bella's poor quality of life.

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