Echo at the Disco

by

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Class of 2022

Clinicopathologic Conference

January 14, 2022

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Introduction:

Discospondylitis is an infectious inflammatory neurologic disease that originates at two adjacent vertebral endplates and secondarily affects the associated intervertebral disc.[12,20] The condition was discovered in humans around 400_{BC}, but the first canine case was not reported until the 20th century.[20] Middle aged, male, purebred, large breed dogs are predisposed, and risk factors include previous spinal surgery, immunosuppression, and comorbidities.[3,20] Historically, the most common isolate is coagulase-positive *Staphylococcus*, but other etiologies reported include *Escherichia coli*, *Brucella canis*, and *Aspergillus*.[3,6,9] Infection is typically acquired by hematogenous spread, and the most common site infected is L7-S1 disc space.[6,12,20] Other reported mechanisms and locations of infection include direct contamination and foreign body migration (eg grass awn) to the thoracolumbar or, less commonly, cervical spine.[6,20] Discospondylitis can be focal or multifocal and result in a variety of clinical presentations ranging from acute to progressive clinical signs with mild to severe neurologic deficits. Systemically, affected dogs often experience a combination of nonspecific signs such as hyperesthesia near the lesion, anorexia, pyrexia, weight loss, and reluctance to move.[3,6,12,20] Neurologic localization is specific to the spinal cord segments affected by the condition.

Accurate diagnosis and appropriate treatment of discospondylitis requires diagnostic imaging and isolation of the causative organism.[3,6,12,16] Pathognomonic radiographic changes are a collapsed disc space with an aggressive lytic and proliferative bony lesion at the affected vertebral endplates and intervertebral disc.[6,16,18,20] Because radiographic evidence of disease lags behind clinical signs, computed tomography (CT) and magnetic resonance imaging (MRI) are often required to detect early pathology and spinal cord compression.[4,12,16,20] Urine or blood

culture are commonly performed but frequently non-diagnostic.[2,17] Due to public health concerns, *Brucella* infection must be ruled out, especially in intact males.[9,10]

Medical management consists of long-term antibiotic therapy guided by culture and sensitivity results when available. Because of culture low yield and likelihood of *Staphylococcal* involvement, first generation cephalosporins are commonly utilized empirically.[6,20] Surgical decompression of the spinal cord is warranted if severe neurologic deficits are present or compressive lesions, such as spinal epidural empyema, are identified on CT/MRI.[1,6,11,20] Monitoring for resolution of disease requires serial spinal radiographs at one-to-two-month intervals regardless of clinical improvement.[3,6,13,16,18] Prognosis is generally favorable but varies depending on neurologic deficit severity, response to therapy, and etiology with fungal agents having a worse prognosis.[6,20]

History and Presentation:

On September 1, 2021, an approximately 7-month-old, male intact German Shepherd presented to Mississippi State University College of Veterinary Medicine Emergency Department for a gradual onset of paraplegia. Pertinent history included a two-week duration of lethargy, hyporexia, persistent pyrexia, progressive spinal pain, and an acute, self-limiting episode of forelimb lameness one month prior with no evidence of trauma or injury. During the initial workup by the primary veterinarian on August 14, 2021, the patient was febrile with a temperature of 104^{o}F, but his physical exam and bloodwork were unremarkable. He was given a dose of meloxicam and discharged with cefazolin, doxycycline, and carprofen. Four days following discharge, the patient's signs had progressed to include hindlimb ataxia and reluctance to flex the head and neck. On physical exam, he was had severe cervical hyperesthesia and a

fever of 103ºF. Apart from a mild anemia (RBC 5.12 x 10^3/ul [reference range: 5.83 – 9.01]), no clinically significant abnormalities were appreciated on his complete blood count and chemistry panel. Cervical radiographs revealed no overt abnormalities. He was hospitalized for monitoring and given a dose of dexamethasone subcutaneously. He improved overnight and was discharged with a course of prednisone. One week later, the patient became acutely paraplegic and was referred to MSU-CVM for further neurologic evaluation.

On presentation to MSU-CVM Emergency Department, the patient weighed 23.8 kilograms with a body condition score of 4/9. He was anxious but alert and responsive. On cardiothoracic auscultation, he was mildly tachycardic with a heartrate of 140 beats per minute and no murmurs or arrythmias were appreciated. His lungs auscultated normally with no crackles or wheezes, and he was panting. His rectal temperature was 103.1ºF. His mucous membranes were pink and tacky with a capillary refill time of less than two seconds. His peripheral lymph nodes palpated soft and symmetrical bilaterally. On neurologic exam, he was mentally appropriate with intact cranial nerves. He was paraplegic with intact nociception in both pelvic limbs and tail. Proprioceptive placement was normal in both thoracic limbs but absent in both pelvic limbs. Hyperesthesia was appreciated multifocally along his vertebral column. His flexor-withdrawal and segmental spinal reflexes were intact. His neuroanatomical localization was T3-L3 myelopathy.

Diagnostic Approach:

The patient was admitted by the emergency department and a thorough triage examination was conducted. No free fluid was visualized on abdominal or thoracic FAST scan, and he had a normal sinus rhythm on electrocardiogram. He was normotensive with oscillometric

blood pressure readings at 116/102 (106), 85/75 (78), 114/58 (76) mmHg. He was adequately oxygenating at 96% pulse oximetry. A venous blood sample was submitted for complete blood count (CBC) and biochemistry, and urine was submitted for urinalysis and culture. CBC abnormalities included mild anemia (RBC 5.44 x 10^6/ul [reference range: 5.60 – 7.90]), mild lymphopenia (lymphocytes 993.6 /ul [reference range: 1100.0 – 4800.0]), moderate thrombocytopenia (platelets 71 x 10^3/ul [reference range: 159 – 455]) and moderate hyperproteinemia (plasma proteins 8.9 g/dl [reference range: 6.0 – 7.5]). Manual platelet count confirmed thrombocytopenia. Chemistry abnormalities included mild hyperphosphatemia (phosphorus 8.9 g/dl [reference range: 2.5 – 6.8]), which was attributed to normal puppy growth, and mildly elevated creatinine kinase (CK 795.0 U/L [10 – 200]). Urinalysis was within normal limits. Urine culture and blood *Brucella* serology were negative. Based on the patient's history, signalment, presentation, and neurological status, he was transferred to the Neurology Service for further evaluation.

An intravenous catheter was placed, and an emergency computed tomography was performed to evaluate the integrity of his spine and further characterize the lesion causing neurologic deficits. CT revealed an aggressive bony lesion with sclerosis and moth-eaten lysis of the endplates between thoracic vertebrae five (T5) and six (T6). Additionally, there was a moderate amount of heterogenous contrast enhancing material surrounding T5 and T6, which extended into the ventral aspect of the spinal canal. The spinal cord was focally dorsally deviated and severely narrowed above T5-T6. Finally, several intra-abdominal lymph nodes appeared moderately enlarged. The CT was most consistent with discospondylitis with extradural compression from suspect empyema and associated reactive lymphadenopathy. The patient was hospitalized overnight on intravenous medications including Plasmalyte, methadone, maropitant,

pantoprazole, and cefazolin. Based on the diagnostic imaging findings and neurological status of the patient, surgery was recommended for spinal cord decompression and to obtain samples of the lesion for culture.

Treatment:

On September 2, 2021, the patient underwent a left-sided hemilaminectomy at the level of T5-T6. Under general anesthesia, the patient was clipped, aseptically prepped, and placed in sternal recumbency. His dorsal thoracic spine was draped, and an approximately 10-centimeter skin incision was made along dorsal midline from the spinous processes of T4 to T8 using electrocautery. The muscles and fascial planes were dissected to reveal the spinous processes of T4 to T7. A hemilaminectomy was performed at the level of T5 and T6 using a high-speed surgical drill, 3-millimeter round burr, and Kerrison ronguers. Within the spinal canal at T5-T6 and adhered to the dura, there was a moderate amount of irregularly marginated, white, firm material ranging in size from 1-3 millimeters. The spinal cord was decompressed by removing the extradural material with gentle traction using forceps and various probes. Samples were submitted for cytology, culture, and histopathological evaluation. The surgical site was flushed with 0.9% sterile saline, and a porcine small intestine submucosal patch was placed over the exposed spinal cord followed by an autogenous fat graft. A routine three-layer closure was performed.

The patient recovered from general anesthesia uneventfully. Postoperatively, he was maintained on intravenous fluids, fentanyl constant rate infusion, pantoprazole, and cefazolin. Eighteen hours after surgery, his neurological status had improved to non-ambulatory paraparetic with persistent proprioceptive deficits bilaterally in the hindlimbs. He was struggling to

voluntarily urinate, so bethanechol and diazepam were added to his treatment regimen. The patient's pyrexia resolved, and neurologic deficits gradually improved. Three days postoperatively, he was eating, drinking, urinating, and defecating normally. Intravenous therapies were discontinued, and he was transitioned to oral gabapentin and Tylenol 4. He was moved into the general hospital wards for physical therapy and further monitoring.

Case Outcome:

The cytology, culture, and histopathology for the extradural material were nondiagnostic or revealed no growth. The biopsy results were consistent with osseous metaplasia composed of a mixture of fibrous connective tissue, cartilage, and woven bone. Negative cytology, surgical site culture, and urine culture were attributed to recent history of multi-antibiotic administration. Blood cultures were not performed due to cost and suspected low yield. With no causative agent identified, the patient was treated empirically with a long-term course of cephalexin. At the time of discharge, he was ambulatory paraparetic requiring minimal support with mild proprioceptive deficits bilaterally in the hindlimbs. Activity restriction and physical therapy instructions were provided to facilitate surgical site healing and gradually promote recovery to unassisted ambulation. At recheck examination two weeks following discharge, the patient showed dramatic improvement as he was able to walk unassisted with mild paraparesis and proprioceptive ataxia. No pain was elicited on spinal column palpation, and his physical exam was within normal limits. Three months postoperatively, the owner reported that the patient continues to make slow improvements and can now run, climb stairs, and stand on his hindlimbs.

Pathophysiology:

The pathophysiology of discospondylitis is controversial and complex in both human and veterinary medicine. Hematogenous is the most common route of infection, whether suspected or confirmed, and several theories exist regarding organism inoculation.[6,12,20,21] The majority of theories are based on foundational knowledge of the spine's anatomy and blood supply. Canine spinal cord and vertebrae blood supply is provided by spinal branches from the vertebral, intercostal, or lumbar arteries.[20] These arteries give rise to the ventral spinal artery, which runs along the ventral median fissure in the vertebral canal. The ventral spinal artery branches into the segmental artery and interosseous arteries within the vertebral body ending in vascular loops situated at each vertebral epiphysis.[12] The loop structure increases the surface area for nutrient exchange at the interface between the endplate and the avascular intervertebral disc.[3] The venous drainage system for the capillary loops is by way of the valveless vertebral venous system on the ventral aspect of the vertebral canal.[20] The basivertebral vein, which runs through the vertebral body, also anastomoses with the venous plexus. The proposed mechanism for hematogenous seeded discospondylitis is that these vascular loops and valveless plexuses slow circulation in high pressure scenarios, thereby promoting infectious organism accumulation.[12,16,20] Because of the intimate relationship between the endplate and intervertebral disc, progression of disease is thought to be by direct diffusion.[20] Infection may spread from the following sources: urogenital tract, direct skin inoculation, dental pathology, grass awn migration, or endocarditis.[12,20] Comorbidities that increase the risk of human infection include diabetes mellitus, hypothyroidism, urogenital infections, and respiratory tract infections. [3,6,20,21] Similar concurrent diseases are reported in dogs with discospondylitis.[3] *Staphylococcus* and *Escherichia coli* are the most common gram-positive and gram-negative isolates, respectively.[3,6,20] The most common fungal genus is *Aspergillus*.[6,20] Brucellosis is a significant public health concern as zoonotic

transmission can cause human abortion, fever, endocarditis, and arthritis.[9,10,20] Any disc space can be affected, but L7-S1 is the most common site of infection.[20]

Spinal epidural empyema (SEE) is a severe sequela of discospondylitis that involves a septic, suppurative process within the epidural space causing spinal cord compression.[6,14,19] Congestion within the abdominal and epidural venous plexuses promotes reflux and exchange of infectious and inflammatory mediators causing spread of disease.[19] Unlike discospondylitis, SEE most commonly occurs in the thoracolumbar area.[1,14,19] This can be explained by two factors: the epidural space in this region is larger allowing for accumulation of infectious material, and the pro-inflammatory epidural fat in this location is more abundant.[19] Spinal cord damage occurs by direct compression from infectious material, thrombosis, ischemia, or inflammation from toxins and inflammatory mediators.[7, 19] The well-documented "classic triad" of human SEE symptoms includes fever, back pain, neurological symptoms.[5,11] Because these are nonspecific, only 13% of humans meet the criteria for the triad on initial assessment.[5] The triad has not been defined in canine SEE, but similar nonspecific clinical signs are noted including weight loss, anorexia, depression, fever, and spinal pain.[14] Human risk factors for developing SEE are diabetes mellitus, intravenous drug use, multifocal sepsis, and immunosuppression.[1,11] No veterinary risk factors have been confirmed.

Discussion:

Spinal epidural empyema secondary to progression of discospondylitis has rarely been reported in veterinary medicine. Although no etiologic agent or septic process were identified in this case, the history, clinical signs, and imaging findings were highly suggestive of the disease. Other differentials for thoracolumbar myelopathy include vertebral malformation,

myelitis/meningitis, multiple cartilaginous exostosis, intervertebral disc extrusion-protrusion, degenerative myelopathy, fibrocartilaginous embolism, and neoplasia.[8,20] Given the patients age and clinical presentation, primary consideration was given to infectious/inflammatory etiologies such as bacterial, fungal, or tick-borne agents causing discospondylitis with empyema. Vertebral physitis and osteomyelitis were also considered due to the parallels with discospondylitis in signalment and presentation. Primary diagnostic imaging findings with vertebral physitis include widening of the caudal vertebral physis with focal lysis and loss of detail of the surrounding metaphasis and epiphysis.[6,12,16] With osteomyelitis, a lytic and proliferative aggressive bony lesion is centered on the vertebral body.[12,20] In contrast to discospondylitis, the integrity of the caudal endplate and intervertebral disc space is preserved with physitis and osteomyelitis.[12,20] The lesion identified on CT in this case was centered on the endplates.

Progressive T3-L3 myelopathy with rapidly declining neurologic status, severe pain, and persistent fever warranted immediate imaging. CT was elected due to availability, time constraints, the emergency status of the patient's condition, and the need for possible surgical planning. The aggressive bony lesion centered on the vertebral endplate with surrounding extradural contrast enhancing material was confirmatory for a diagnosis of discospondylitis with SEE. Historically, discospondylitis has been a radiographic diagnosis, revealing a collapsed intervertebral disc space, endplate lysis, sclerosis, and possible vertebral subluxation.[3,6,18,20] Unfortunately, radiographs are insensitive as the changes are often not visible until two weeks after disease onset.[6,16,21] Additionally, atypical findings such as disc space narrowing without the classic lytic appearance are often present in juvenile dogs with discospondylitis.[13] Therefore, advanced imaging is preferred to identify subtle endplate erosion and paravertebral soft tissue swelling early in the course of disease, especially in young dogs.[12,13,16,20]

MRI is the modality of choice in human medicine for diagnosis of discospondylitis.[4,20] Like CT and radiography, characteristic findings include narrowed intervertebral disc space and endplate cortical erosion.[4,6,16,20] The endplates are typically T1 hypointense and T2 hyperintense.[4,6,16,20] With chronicity, the endplates become T2 hypointense.[4] Adjacent soft tissues are often contrast enhancing and hyperintense on short tau inversion recovery (STIR) and T2-weighted images.[4,6,12,16] Less commonly used diagnostic tools include scintigraphy, ultrasound, and fluoroscopy-guided fine need aspirate.[12,16,20] A retrospective cross-sectional study conducted by Emery, et al. in 2017 concluded that CT is a reasonable first-line imaging modality to identify canine thoracolumbar myelopathy lesions.[8] In this report, additional imaging was required in older patients, afterhours imaging, lack of surgical lesion identification on CT, and non-dachshund breeds. Although CT is insensitive compared to MRI, it is an appropriate option in many patients presenting with thoracolumbar myelopathy, including the patient in this case.

MRI is also the gold standard for SEE diagnosis because of the high sensitivity and ability to thoroughly assess the extent of the lesion, particularly changes to the soft tissue structures.[1,7,19,21] SEE is classically contrast enhancing, T1 hypointense, and T2 and STIR hyperintense.[6,7,16,19] Paraspinal musculature varies in appearance based on route of infection and ranges from T2 hyperintense to T1 hyperintense.[7,19] Spinal cord gray matter is usually T2 hyperintense.[6,7] Subjective assessment of the basivertebral vein may reveal alterations in size.[7] Postcontrast T1-weighted images show one of two patterns: peripheral enhancement around homogenous non-enhancing tissue or diffuse enhancement of epidural fat.[7,16] In human medicine, diffuse enhancement is associated with a better prognosis, but this correlation has not

been defined in veterinary medicine.[7,19] With MRI, lesions may be visualized as early as one week from disease onset in human medicine.[19]

Complete diagnostic workup for etiologic agent identification in discospondylitis and SEE includes blood and urine cultures.[6,10,20] In a retrospective cross-sectional study performed at North Carolina State University College of Veterinary Medicine, the validity of canine urine versus blood cultures were assessed based on hospital sample submissions over five years.[2] Two hundred ninety-five parallel blood and urine cultures were submitted, of which only 14 had concordant growth. These results prove that urine culture is not a replacement for blood culture in identifying systemic infections. Urosepsis was the only exception, as previously diagnosed urogenital disease had 100% concordance in urine and blood culture results. The report also concluded that overall organism identification was lacking with only 17% and 24% positive blood and urine cultures, respectively. These results agree with recently published data in which only 20% of blood cultures were positive in hospitalized dogs.[17] Therefore, blood and urine cultures should be used as adjunctive tests in discospondylitis and SEE with imaging being the primary diagnostic.

Bloodwork abnormalities in discospondylitis with associated SEE commonly include leukocytosis, neutrophilia, and hyperglobulinemia.[6,14,19,20] In human medicine, C-reactive protein (CRP) assay is used as a marker of inflammation in various diseases, specifically osteomyelitis and discospondylitis.[19] A retrospective medical record analysis was performed to investigate the correlation of CRP with canine discospondylitis.[21] Trub, et al. concluded that CRP is a more sensitive biomarker than fever and leukocytosis in diagnosis of discospondylitis, and it is typically elevated in early stages of disease. Additionally, CRP may be useful in excluding intervertebral disc disease as CRP levels should be normal with IVDD. Elevated CRP was not

associated with the presence of empyema, but it may be prognostic in predicting failure of medical management. This data illustrates that CRP should be considered as an additional diagnostic when discospondylitis and SEE are suspected.

SEE is most often a surgical emergency for decompression of the spinal cord.[1,5,6,14,19] However, medical versus surgical management of SEE has recently been explored in human medicine.[1,19] Acceptable reasons to opt for medical management includes presence of significant comorbidities, lack of spinal cord compression, or greater than seventy-two hours of complete paralysis.[1] A recent human case report described a CT-guided percutaneous fine needle aspiration of SEE and concluded that this may be a reasonable alternative to surgery if SEE is diagnosed with no neurologic deficits or the patient is a poor anesthetic candidate.[19] Regardless of medical versus surgical treatment, recovery is highly dependent upon severity of deficits and duration of treatment delay.[1,14,19] A specific prognosis has yet to be defined for canine discospondylitis and SEE but is typically contingent upon severity of signs/neurological status and ability to identify the organism.[6,15,19] Infection with *Aspergillus* has been associated with a poorer prognosis.[6,] There is a possibility for persistence of neurologic deficits especially if treatment is delayed or clinical signs are severe.[20]

Regardless of treatment regimen, most patients require long-term physical therapy, serial imaging, and antibiotics.[3,17,20] Clinical improvement is marked by resolution of fever, return of appetite, and improvement of neurologic deficits and can occur as soon as one to two weeks after initiation of antibiotics.[6,18,20] Serial radiographs are used to track vertebral structural healing with bridging and sclerosis, which may take up to 40-80 weeks.[3,6,16] Shamir, et al. published a retrospective cross-sectional study in *Veterinary Radiology and Ultrasound* describing the recovery progress of dogs with discospondylitis.[18] They concluded that clinical and radiographic

recovery accurately correlated in affected dogs that were less than one year old. However, a significant three to nine week lag in radiographic recovery was observed in older dogs. Although this delay in healing did not correlate with severity of clinical signs, duration of disease, or lesion location, it is clinically relevant when assessing older patients recovering from discospondylitis. Based on these results, antibiotic efficacy should not solely rely on radiographic findings but also include clinical progress.[3,18] It is still recommended, however, that the duration of antibiotic therapy be based on radiographic resolution.[3,18]

In conclusion, discospondylitis should be considered in dogs with spinal pain, neurologic deficits, and the described diagnostic imaging findings. Failure to respond to antibiotic therapy and/or rapidly deteriorating neurological status should raise concern for an atypical pathogen/improper antibiotic selection or concurrent SEE.

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