Physitis & a Fracture in an English Bulldog

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History

Jax, a 10-month-old intact male English bulldog, was presented for acute onset of a pelvic limb gait abnormality. Jax was adopted at 6 months of age with no prior medical history reported. Three weeks after adoption, he was presented to the neurology service for a 5-day history of scuffing and dragging both pelvic limb paws when walking (see ***Gait Abnormality Video***). There was no history of trauma or discomfort since adoption. The primary veterinarian performed a routine serum chemistry profile and CBC prior to referral; all results were within normal limits.

Physical & Neurologic Examination

On examination, Jax was bright, alert, and responsive. Results of cranial nerve examination were normal. Moderate general proprioceptive ataxia and paresis were present in both pelvic limbs. Evaluation of postural reactions revealed delayed proprioceptive positioning in both pelvic limbs; in addition, all segmental reflexes were intact. No hyperpathia was noted on palpation of the vertebral column. Temperature was 102.4ºF (39.1ºC). The physical examination was otherwise normal.

Based on the neurologic examination, an anatomic diagnosis of T3-L3 myelopathy was made. Differential diagnoses included trauma, infection,

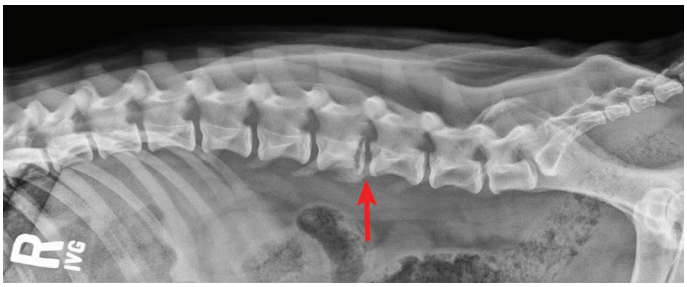
GAIT ABNORMALITY VIDEO

To view a video of Jax's gait abnormality on presentation, visit **cliniciansbrief.com/physitis-fracture**

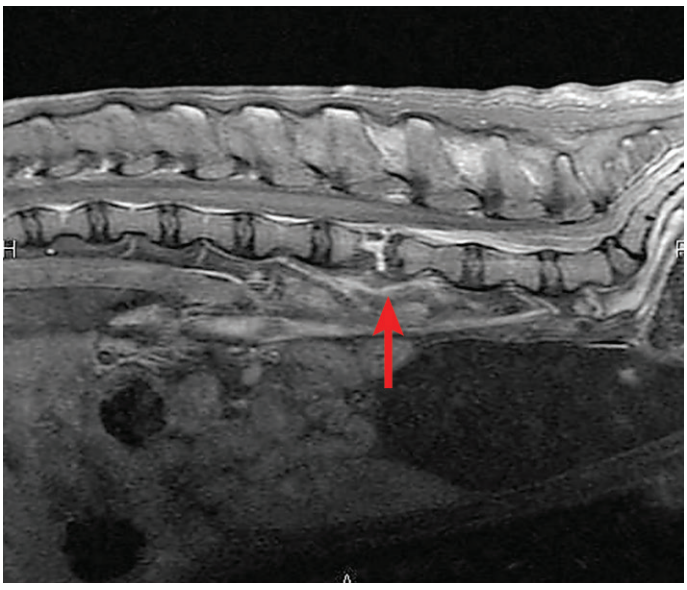
inflammation, neoplasia, and a congenital vertebral malformation. A degenerative process (eg, storage disease) was also considered.

Diagnostics

Imaging of the thoracolumbar vertebral column was performed. Survey radiographs of the vertebral column were obtained because of the possibility of trauma, infection, neoplasia, or a congenital vertebral malformation. For general practitioners, it should be noted that survey radiographs are usually not indicated when intervertebral disk disease is suspected. Although radiographs may show mineralized disks, they are not reliable because they are unable to definitively diagnose which disk is causing the problem and are unable to show the extent of any spinal cord compression. If intervertebral disk disease is the primary consideration, a practitioner should attempt to conserve finances for advanced, confirmatory imaging such as MRI or, in some instances, CT.



▲ **FIGURE 1** Lateral radiograph of the patient's vertebral column. Narrowing of the L4-L5 space, reduction of the L4-L5 foramen, and an irregular lucent cleft at the level of the caudal endplate of L4 can be seen (***arrow***).



▲ **FIGURE 2** Sagittal T1+ contrast MRI revealing indistinct periosteum with irregular physeal bone at the level of L5, with strong enhancement of the L4 caudal endplate and associated epidural space (***arrow***)

A lateral radiograph of Jax's vertebral column revealed narrowing of the L4-L5 intervertebral disk space, reduction of the L4-L5 intervertebral foramen, and an irregular lucent cleft at the level of the caudal endplate of L4 (***Figure 1***). These findings were most consistent with physitis (see ***Discussion***) and an associated end plate fracture.

CT was performed to evaluate bony compartment involvement. The 3 compartments of the vertebral column include dorsal, middle, and ventral compartments. The dorsal compartment is composed of the spinous process, the vertebral arch, the articular process, and the associated ligaments. The middle compartment includes everything ventral to the articular process, including the ventral floor of the vertebral canal and the dorsal portion of the intervertebral disk. The ventral compartment includes the remaining portion of the intervertebral disk and the vertebral body.[1] Two compartments were found to be involved: the dorsal vertebral body (ie, middle compartment) and the remaining vertebral body (ie, ventral compartment).

An MRI was performed and revealed indistinct periosteum with irregular physeal bone and no obvious sign of trauma at the level of L5 (***Figure 2***). The caudal endplate of L4 and the associated epidural space became strongly enhanced after administration of gadolinium contrast.

Urinalysis, urine culture and susceptibility, and *Brucella canis* titer results were within normal limits.

Potential causes for Jax's physitis and fracture included a traumatic fracture that occurred before adoption and progressed to secondary physitis or a bony reaction secondary to a fracture. However, the most likely cause in this case—based on signalment, author experience, and a similar prior case report[2]—is active physitis that occurred secondary to infection elsewhere in the body with a subsequent pathologic fracture.

Treatment

Cephalexin was prescribed at 22 mg/kg q12h for 8 months to treat physitis. An antibiotic regimen lasting 6 to 8 months is generally recommended in cases of vertebral infections because of the possible nature of these infections (see ***Discussion***).

Typically, if 2 out of 3 compartments are involved in a vertebral column fracture, as in this case, surgical stabilization is recommended. However, the high suspicion of infection made this dog a poor candidate. Strict rest was emphasized to aid fracture healing.[1]

Discussion

Physitis refers to inflammation and lysis of the caudal physeal zone of one or more vertebrae.[3] This does not include inflammation of the intervertebral disk, which would be referred to as *diskospondylitis*.[3] Diskospondylitis also affects both endplates.[3] In cases of physitis and most other vertebral infections, the primary cause or infectious agent is usually not identified.[2] It has been hypothesized that physitis arises from hematogenous spread of an infection elsewhere in the body.[2] Common sources of a primary infection include the urogenital tract, skin, heart, and oral cavity.[2] Infections associated with physitis are most commonly thought to result from the urogenital system when the lumbar vertebrae are involved.[2]

Knowledge of the vascular system of the vertebrae and vertebral column is necessary to understand how primary infection elsewhere in the body may result in physitis (see ***Vascular System***).

Because of the location of the lesion in this case, it was hypothesized that the patient may have had an occult urogenital infection (eg, prostatis) that reached the lumbar vertebrae due to retrograde blood flow through the

VASCULAR SYSTEM

Within the vertebral bodies, venous blood drains through the basivertebral vein. This venous structure (usually paired) occurs in the midbody of the vertebra in an osseous canal. Blood drains dorsally to enter the ventral internal vertebral venous plexus. The ventral internal vertebral venous plexus, also a paired structure, courses from the cranial cavity caudally throughout the vertebral canal. Venous blood from the ventral internal vertebral plexus drains into the intervertebral veins, which course along with spinal nerves out of the vertebral column. In the 5th through 7th lumbar vertebrae, the intervertebral veins continue as paired lumbar veins. At each vertebra, L5 through L7, the paired lumbar veins anastomose with each other and enter the dorsal aspect of the caudal vena cava, which lies immediately ventral to the ventral surface of the lumbar region of the vertebral column. This entire venous system is a collection of valveless vessels. Blood may follow in either direction based on the pressure in the system. The caudal abdominal organs drain into the common iliac vein, which becomes the caudal vena cava.[4] Consequently, there is a close relationship with venous drainage of the lumbar vertebrae and caudal abdominal organs (eg, the urogenital tract).[2,4,5]

With increases in abdominal pressure, there can be a reversal of venous blood flow whereby blood from the caudal vena cava flows retrograde into the basivertebral veins. This retrograde venous flow can transport bacteria and other potentially infectious organisms into the vertebral bodies.[2]

For a helpful diagram of the vascular system, see ***Suggested Reading***, next page.

unique vascular system as previously described.

This hypothesis is based on various research in the human literature showing evidence of prostatic metastasis to the lumbar vertebrae through similar mechanisms[6,7] as well as on an author's previous case in which an occult pyelonephritis was identified following postmortem examination.

Research on causative organisms for vertebral infections such as physitis is limited. In diskospondylitis, the most common organism isolated is *Staphylococcus* spp.[2] A primary causative organism has not been isolated for physitis. However, *Escherichia coli*, *Serratia* spp, *Streptococcus* spp, and *Staphylococcus* spp have been identified in patients with uncomplicated physitis.[2] Urine culture and serology for *Brucella canis* are strongly recommended in all patients. Although there is no clear evidence linking brucellosis to vertebral infections, its zoonotic potential and localization in the urogenital tract make it an imperative rule-out. Although expensive and often negative, blood cultures should also be considered.

Typically, an antibiotic with efficacy against gram-positive organisms (specifically *Staphylococcus* spp), such as amoxicillin–clavulanic acid or cephalexin, is recommended. If extensive neurologic deficits are involved, combination therapy with enrofloxacin is recommended to increase the antimicrobial spectrum. Antibiotics should be continued for at least 6 to 8 months or several months past resolution of clinical signs. Pain can be treated with gabapentin or tramadol. If the patient does not respond to pain medication alone, an NSAID (eg, carprofen) may be considered.

In this case, surgical debridement and/or stabilization were not pursued because of the increased risk for implant failure. If a patient does not respond to medical management, surgery may be indicated to debride the region and obtain samples for culture. In these cases, the benefits of surgical intervention are thought to outweigh the risks.

Outcome

Jax remained on an 8-month course of cephalexin and strict rest. His neurologic signs resolved, antibiotics were discontinued, and his clinical signs never recurred.

References

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Suggested Reading

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