

A Real Pain in the *Back*

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Introduction:

Congenital spinal disorders in cattle can have many different etiologies. Such etiologies include genetic, infectious, toxic, nutritional, and embryologic defects. This paper focuses on congenital structural abnormalities that cause malformations of the vertebral column. A few bovine congenital structural spinal issues include short spine lethal, atlanto-occipital fusion, kyphosis, lordosis, scoliosis, torticollis and many more. Many of these defects can occur by themselves or in association with other organ systems. Since research in congenital vertebral malformations is limited in large animal species, pathophysiology and treatment is extrapolated from small animal and human studies.

History and Presentation:

Otis Fleenor, an approximately 6-month-old Scottish Highlander bull calf, presented to Mississippi State University College of Veterinary Medicine Food Animal Services on April 21, 2020 for a hunched back, tail flagging and mild weakness in the hindlimbs. The owners noted that Otis had been “off” and hunched up for approximately 10 days. He had progressively gotten weaker in the hind end over the past several days. Otis was still nursing at the time of presentation, and was accompanied by his dam, Maggie. He and Maggie are kept in a field with other calves his size as well as adult cattle. Occasionally the calves are noted to “rough house” in the pasture.

A physical examination was performed. On presentation Otis was bright, alert and responsive. He weighed 165kg with a body condition score of 6/9. His vital parameters were within normal limits. He had a temperature of 101.4 F. His heart auscultated with a rate of 115 beats per minute and no murmurs or arrhythmias were detected. His lungs auscultated normally with no crackles or wheezes. His respiration rate was 48 breaths per minute. His hydration

appeared to be normal with pink, moist mucous membranes. His capillary refill time was less than 2 seconds indicating adequate perfusion. A limited neurological exam was performed due to the nature of the species. Otis' mentation was alert and responsive and all cranial nerves were considered normal. He walked with a kyphotic posture in the lumbar region with his tailhead held to the left. He was ambulatory with moderate proprioceptive ataxia as his hindlimbs were noted to cross over midline when walking as can be seen here in this image. He seemed to have normal hoof placement in both his thoracic and pelvic limbs with no knuckling during proprioceptive testing. No pain was noted on spinal palpation however, a kyphosis of the spine could be appreciated on palpation.

Diagnostic Approach and Considerations:

Following Otis' physical and neurologic examinations, a complete blood count and chemistry were performed which revealed no significant clinical abnormalities. Given the history of "tail-flagging" and "hunched posture" a brief abdominal ultrasound was performed to ensure no bladder issues were evident to indicate urolithiasis. No abnormalities were noted. Next, a single right lateral thoracolumbar spinal radiograph showed focal kyphosis at the level of L2 and L3. The L2 and L3 vertebrae were wedge shaped and foreshortened in a cranial to caudal direction. The end plates were also not present at this site. The L2-3 intervertebral disc space was narrowed.

Based on the radiographic findings combined with his clinical signs of paraparesis and pelvic limb ataxia the next step was to perform a CT of his spine to further determine the level of spinal cord involvement. The CT confirmed the structural abnormalities noted on the radiograph. In addition, the spinal cord was flattened dorsoventrally at this site. Also, the previously identified narrowing of the L1-2 intervertebral disc space was identified. Given the history,

physical and neurologic examinations, and diagnostic findings, our differentials for Otis' included congenital vertebral malformation or previous trauma. Infectious and degenerative lesions were considered but no evidence of these conditions was identified on exam or any diagnostics. Therefore, the etiology of Otis' condition was likely congenital or trauma-related. Unfortunately, the lesion could not be further characterized based on radiographic modalities so both etiologies had to be considered when developing his treatment plan.

Treatment and Management:

A bilateral articular facet arthrodesis was performed at L1-2, L2-3 and L3-4. This procedure intended to allow the ventral segments of the vertebrae to continue to grow while halting growth of the dorsal vertebra to prevent worsening kyphosis.

On April 28, 2020 Otis was taken to surgery. The surgical procedure went as follows. The patient was placed in sternal recumbency under general anesthesia. Next, the patient was clipped and aseptically prepped. Using palpation, T13 to L4 were identified and paramedian incisions were made bilaterally. Periosteal elevators and mayo scissors were used to dissect down to the facets. The attachments to the facets were cauterized and broken down with Mayo scissors. Hemorrhage was controlled with bipolar cautery. A small amount of muscle was removed to facilitate access to the facets. The anatomy was atypical, consistent with the radiographs. Using the radiographs and CT images as a guide, the L2-3 facets were easily identified when compared to the L1-2 and L3-4 facets. A Hall Burr Drill with a small round burr was used to drill through the zygapophyseal joints to remove the articular surfaces at L1-L2, L2-L3 and L3-4. A drill bit was used to create a pathway for the cerclage wire through the right L3-L4. This was completed with some difficulty. Due to the inability to drill through the facets safely and appropriately, a cerclage wire was not placed on the right L1-L2 or L2-L3 facets. The

cerclage wires were not attempted on the left due to difficulty/risk. The sites were copiously lavaged. Synthetic bone matrix (Consil) was placed in the previously drilled locations within the facets. The fascial layer was closed with 1 PDS in a combination of cruciate and single suture pattern bilaterally. The subcutaneous tissue was closed with 1 PDS in a simple continuous pattern. The skin was closed bilaterally with 1 Braunamid in a ford interlocking pattern. Otis recovered from anesthesia uneventfully.

In the days following surgery Otis was monitored for any neurologic disturbances including worsening ataxia, neuropathies, or myopathies. Otis' ataxia seemed to worsen at times. He also had difficulty rising. This was thought to be due to pain and inflammation of his surgery site resulting in soreness. Otis was kept separate from his dam but was allowed to nurse for 15-30 minutes twice daily. He also given calf starter, alfalfa, and collard greens to ensure he received adequate nutrition. For pain management a combination of morphine, meloxicam, and gabapentin was used. The morphine was switched to butorphanol a few days following surgery. Throughout Otis' recovery he had the most difficulty with ataxia, knuckling and a narrow-based stance when ambulating. Many modalities of physical therapy were used to assist in improving these signs.

On May 4, 2020, Otis began rehabilitation. For the pain and soreness associated with the incision, the incision line was lasered with a Class 3b Cold Laser Therapy at a dosage of 2 J/cm² for a total of 160 Joules per incision site and the immediate surrounding area. It was applied using a pulsed mode of 20 mHz. Otis' physical rehabilitation plan consisted of balance, proprioceptive, muscle strengthening exercises to assist in improving his ability to ambulate.

Proprioception exercises of mild side to side weight shifts were initiated to help with balance and muscle building. The use of a 1 in high balance pad under each hind foot was

incorporated into the weight shift routine. Due to Otis' continued difficulty rising and standing on his own, a supporting sling and walker were used to assist Otis in walking. The sling provides support of the animal's hind limbs but allows for normal ambulation. To help improve his narrow-based stance and periodic limb crossing, a Theraband was placed around the pastern region of each hind leg to help facilitate normal hoof placement when walking with the sling and the walker. On May 28th, recheck radiographs were taken due to the ataxia. No significant changes were noted since the previous films. On May 29th, a 15 foot PVC pipe with a 4 inch diameter was added to Otis's walking routine. He was asked to walk the length of the pipe with a front hoof and hind hoof on either side of the pipe to encourage him to track wider in the hind limbs. Otis' rehabilitation plan was continued at home with his owners after discharge.

Pathophysiology:

Malformation of the vertebra causing kyphosis and secondary spinal cord compression is the ultimate diagnosis in this case. A trauma-related injury resulting in this confirmation could not be ruled out, but for the purpose of this paper, congenital abnormalities resulting in similar presentations will be discussed. Due to limited research on congenital vertebral malformations in the bovine species specifically, pathophysiology was extrapolated from small animal and human literature.

In cases of congenital vertebral abnormalities, the malformation is present at birth, but clinical signs may not occur until a few months of age. In many cases the defect goes unnoticed unless incidentally seen on thoracic or abdominal radiographs. In a rare number of cases, as the animal grows, onset of clinical signs develops due to vertebral malformation causing kyphosis to develop at the site. Typically, clinical signs do not develop until a few months of age when the

kyphosis causes a compressive myelopathy in the spinal cord resulting in clinical signs of ataxia, pelvic limb paresis, incontinence, etc.^{1,3}

There are several types of vertebral deformities that can occur throughout the vertebral column.

In small animals, several reported abnormalities include butterfly vertebra, various wedge-shaped vertebra, transitional vertebra, and block vertebra^{3,10}. Often these findings can be incidental but, like in Otis' case, they can occasionally be the underlying cause of clinical signs.

Defects that occur in the embryonic period are a result of abnormalities of the neural tube and can occur in conjunction with other organ systems due to simultaneous development⁸.

Deformities originating in the fetal period are associated with abnormal segmentation, ossification and chondrification of the vertebra^{3,8}. There are multiple centers of ossification within a vertebra. Any insult to the active cartilage proliferation during this period could result in a malformation^{3,6}.

The etiology of congenital vertebral anomalies often goes unexplained. Any disruption along the pathway of embryologic and fetal development can result in vertebral abnormalities³. Causes of disruption could vary, but include genetic malformations, nutritional deficiencies, infectious or toxic insults⁴. The most common genetic vertebral malformation in cattle is reported in Holsteins^{4,7,8}. Nutritional deficiencies, such as maternal diets deficient in vitamin D have also been shown to increase the development of spinal deformities in offspring⁹. Infectious causes of spinal malformation typically present with more than one defect and in association with other organ systems. One study looking at virus-induced congenital malformations, several viruses (BVDV, Schmallenburg virus, blue tongue virus, Akabane virus) were associated with various musculoskeletal and central nervous system defects. Defects included hydranencephaly, cerebellar hypoplasia, kyphosis, lordosis, scoliosis, torticollis and arthrogryposis. Infection with

these viruses at a certain period in the gestation period results in clinical presentation. A theory as to why these defects develop is due to a loss of neurons by the viral infection which causes an imbalance in fetal muscular activity.²

A specific underlying cause for vertebral malformations is usually undetermined. In cattle there are various tests available to determine potential causes, but the diagnosis typically remains open, like in this case.

Case Outcome:

Following discharge from the hospital, Otis' physical therapy was continued by his owners. He was kept in a stall and monitored closely. He was taken on daily walks to graze. His theraband therapy was also continued to help with improvement of his narrow-based stance.

Today Otis lives in a pasture with 2 older cows. His hunched posture is greatly improving and he is able to freely roam the pasture on his own. His hindlimb ataxia and paresis is hardly evident. Overall, Otis made tremendous improvements and seems to be living a great quality of life. What truly made this case have an awesome outcome was the dedication of not only the clinicians and students involved in his care and recovery, but also the dedication of owners following his discharge.

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