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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Cervical Physeal dysplasia and subchondral synovial cyst in a case of compressive myelopathy in a six-year-old Warmblood gelding

Miguel Carrillo Parraguez[1], Brian Murphy[2], Nick Huggons[3] Francisco R. Carvallo[4*]

[1]Escuela de Medicina Veterinaria, Facultad de Ciencias de la Vida, Universidad Andrés Bello, Chile
[2]Department of Pathology, Microbiology, and Immunology, School of Veterinary Medicine, University of California – Davis, Davis, CA.
[3]San Luis Rey Equine Hospital. Bonsall, CA 92003-7106
[4]Department of Biomedical Sciences and Pathobiology, Virginia Maryland College of Veterinary Medicine, Blacksburg, VA.

[*]**Corresponding author:** Fcarvallo@vt.edu

Clinical history:

A 6-year-old, 604 kg. History of progressive incoordination, difficulty to jump and ataxia. Euthanized with barbituric overdose.

Macroscopic findings:

At necropsy, the animal had good postmortem conservation state and appropriate deposits of adipose tissue. Findings in internal organs were associated with euthanasia procedure, with splenomegaly and pulmonary congestion. A parasagittal section of the cervical and thoracic column was made (Fig. 1).

**The Diagnostic Exercises are an initiative of the Latin Comparative Pathology Group (LCPG), the Latin American subdivision of The Davis-Thompson Foundation (DTF). These exercises are contributed by members and non-members from any country of residence. Consider submitting an exercise! A final document containing this material with answers and a brief discussion will be posted on the DTF website: https://davisthompsonfoundation.org/diagnostic-exercise/*

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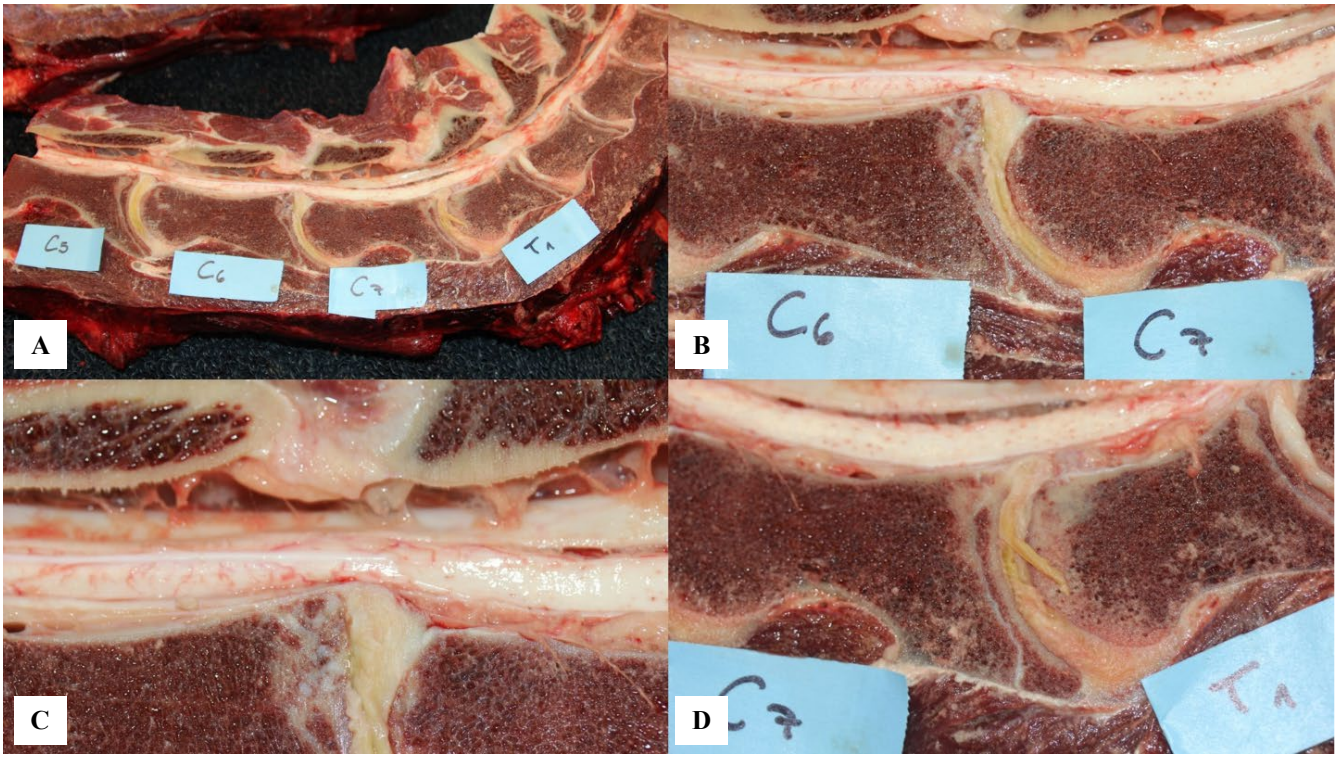

Figure 1. Parasagittal section of the cervical and thoracic column. Figure 1a. Spinal column, vertebrae C5 to T3**. Figure 1b, S**pinal cord at the C6-C7. Figure 1c. Spinal cord, cervical region. **Figure 1d.** Spinal cord, cervical region. Intervertebral disc of C7-T1.

Follow-up questions:

- *Macroscopic description.*
- *Histologic description.*
- *Morphologic diagnosis.*

ANSWERS

Macroscopic description (Fig. 1):

The caudodorsal portion of the body of cervical (C) vertebra 6 (C6) had an osseous protuberance that focally compressed the meninges (dura-mater) and spinal cord. This protuberance was composed of multifocal to coalescing, well demarcated white areas that measured 0.2 to 0.3 cm diameter, located in the physis and epiphysis. Additionally, the intervertebral discs of C5-C5, C6-C7 and C7-T1 were severely degenerate (grades 4 to 5, classification scheme 0-5) (Bergmann et al., 2018), with a yellow fibrillary pulpous nucleus, focal areas of mineralization, a longitudinal cleft and a dorsal protuberance of the fibrous annulus, feature that was more prominent at the C7-T1 intervertebral space. All articular facets in this segment had increased synovial fluid and mild degenerative changes of the articular surfaces, characterized by cartilage erosion and few osteophytes at the margins of the joints.

Histologic description (Figs. 2-7):

Cervical vertebral, physis and epiphysis: Multifocally between the bone trabeculae, there are disorganized islands of hyaline cartilage that measure 0.5 to 2 mm diameter. Most of these islands follow the same trabecular bone path, joining each other and occasionally projecting into the medullary cavities, partially or totally obliterating them. In some areas to the periphery of islands there is a transition zone from the basophilic cartilaginous matrix into the eosinophilic bone matrix, which is characterized by a lower cell density and the presence of ossification lacunae. To the interior of the cartilage islands, there are homogenous groups of chondrocytes in resting, proliferative or hypertrophic stages without a particular organization pattern, which are imbedded in a heterogenous inter territorial matrix with fibrillary projections arranged in multiple crisscrossed patterns (dysplasia). In addition, there is a 3.5 x 1.5 mm, focal well-defined, cavitated cyst-like lesion in the subchondral bone. The cyst is lined internally by one or more layers of cells ranging from spindle-shaped to polygonal

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(synoviocytes) and externally by variable amounts of fibrous tissue that multifocally merges with other islands of dysplastic cartilage.

Morphologic diagnosis:

- Compressive myelopathy due to epiphyseal dysplasia of the caudo dorsal aspect of C6.
- Subchondral focal synovial cyst.
- Intervertebral disc degeneration (grade 4-5) of intervertebral spaces C5-C6, C6-C7 and C7-T1

Comments

The term "Wobbler syndrome" was first used in 1938 to describe some clinical neurological abnormalities in the equine ambulation (Nout et al., 2003). The cervical progressive myelopathy is considered as the main cause of Wobbler syndrome in horses (Levine et al., 2007). In horses with spinal compression in which intervertebral disc disease have been reported, it is suggested that protrusion have less clinical relevance in contrast with well documented description of discopathy reported in dogs and humans with clinical disease (Bergmann et al., 2018). Cervical vertebral compressive myelopathy can originate from alteration of one or multiple structures, so to determine its specific pathogenesis is difficult. Currently two main hypotheses have been proposed to explain the lesion mechanism. The development hypothesis proposes that an undetermined alteration (probably nutritional or genetic) interferes with the maturation of cartilage and bone, which produces cervical vertebral malformation. The biomechanics hypothesis proposes that abnormal mechanical strengths and tensions (such as repeated trauma or inappropriate articular conformation due to a prior lesion) in the cervical column induce reactive structural vertebral changes, which produces stenosis of the medullary canal (Janes et al., 2015). Among the different causes of focal or multifocal spinal compression are atlanto occipital malformations, traumatic damage, vertebral fracture, vertebral neoplasia, discospondilitis, discospondylosis, intervertebral disc protrusion and arachnoid diverticula (Nout et al., 2003).

In the present case, in addition to the macroscopic degenerative changes of the intervertebral disc numerous disorganized islands of epiphyseal cartilage were noted in the caudodorsal aspect of the C6 vertebra, with areas of disorganized endochondral ossification, which was interpreted as physeal dysplasia. In order to explain its possible origin is important to mention that vertebral growth plates close later in the life of the horse, especially in Warmblood horses (around 6 years of age) and that the physes connected to the caudal epiphyses of the cervical vertebrae are the last to close (Dr. Jennifer Janes, University of Kentucky, personal communication). This could mean that the physeal dysplasia can be considered as a reactive developmental alteration, in this case probably due the articular instability secondary to the intervertebral disc disease. During growth and even after the physes closes, vertebrae respond to biomechanical forces of the neck following the Wolff's law associated with mechanic regulation (Janes et al., 2015). The purely morphological diagnosis of epiphyseal dysplasia formulated in this case

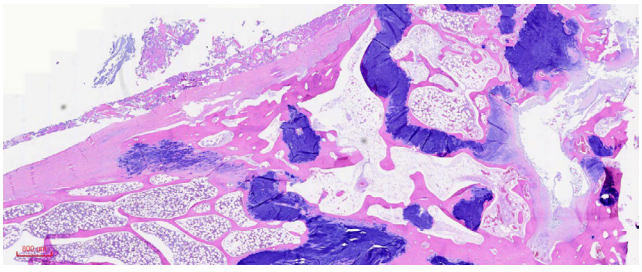

Figure 2. Histologic section of the dorsocaudal aspect of C6. There are multiple disorganized islands of hyaline cartilage that follow a similar bone pathway as present in the epiphysis. In some areas to the periphery of islands there is a transition zone from the basophilic cartilaginous matrix into the eosinophilic bone matrix, which is characterized by a lower cell density and the presence of ossification lacunae. To the interior of the cartilage islands, there are homogenous groups of chondrocytes in resting, proliferative or hypertrophic stages without a particular organization pattern, which are imbedded in a heterogenous inter territorial matrix with fibrillary projections arranged in multiple crisscrossed patterns (dysplasia).

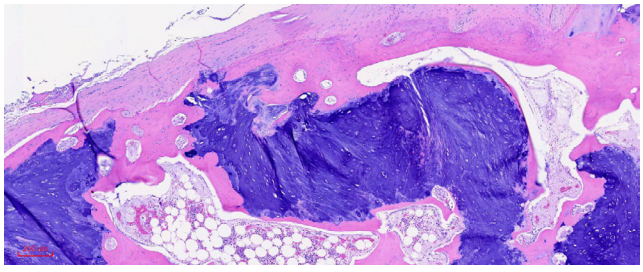

Figure 3. Histologic section of the dorsocaudal aspect of C6. There are multiple disorganized islands of hyaline cartilage that follow a similar bone pathway as present in the epiphysis. In some areas to the periphery of islands there is a transition zone from the basophilic cartilaginous matrix into the eosinophilic bone matrix, which is characterized by a lower cell density and the presence of ossification lacunae. To the interior of the cartilage islands, there are homogenous groups of chondrocytes in resting, proliferative or hypertrophic stages without a particular organization pattern, which are imbedded in a heterogenous inter territorial matrix with fibrillary projections arranged in multiple crisscrossed patterns (dysplasia).

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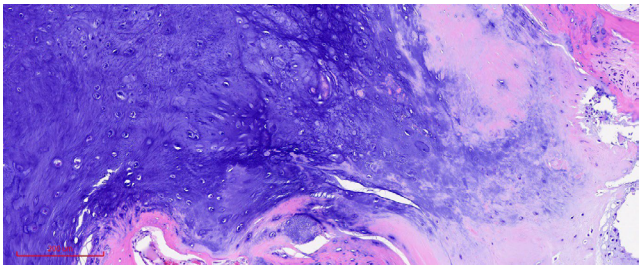

Figure 4. **Histologic section of the dorsocaudal aspect of C6**. There are multiple disorganized islands of hyaline cartilage that follow a similar bone pathway as present in the epiphysis. In some areas to the periphery of islands there is a transition zone from the basophilic cartilaginous matrix into the eosinophilic bone matrix, which is characterized by a lower cell density and the presence of ossification lacunae. To the interior of the cartilage islands, there are homogenous groups of chondrocytes in resting, proliferative or hypertrophic stages without a particular organization pattern, which are imbedded in a heterogenous inter territorial matrix with fibrillary projections arranged in multiple crisscrossed patterns (dysplasia).

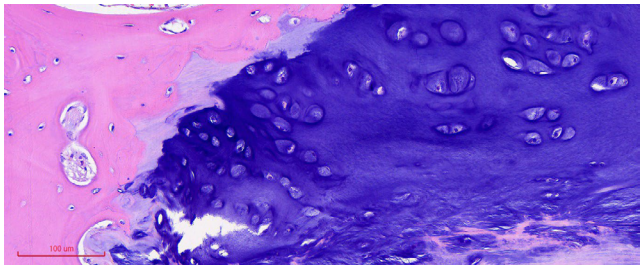

Figure 5: Histologic section of the dorsocaudal aspect of C6. There are multiple disorganized islands of hyaline cartilage that follow a similar bone pathway as present in the epiphysis. In some areas to the periphery of islands there is a transition zone from the basophilic cartilaginous matrix into the eosinophilic bone matrix, which is characterized by a lower cell density and the presence of ossification lacunae. To the interior of the cartilage islands, there are homogenous groups of chondrocytes in resting, proliferative or hypertrophic stages without a particular organization pattern, which are imbedded in a heterogenous inter territorial matrix with fibrillary projections arranged in multiple crisscrossed patterns (dysplasia).

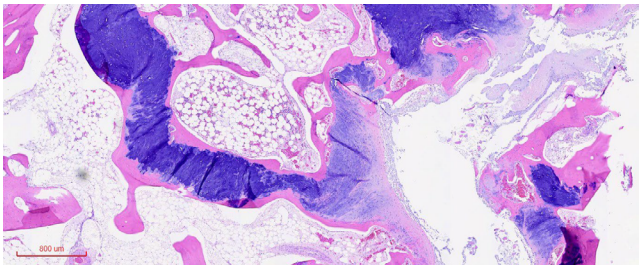

Figure 6. **Subchondral synovial cyst**. The cyst is lined internally by one or more layers of cells ranging from spindle-shaped to polygonal (synoviocytes), occasionally covering papillary projections of cartilaginous matrix, and externally by variable amounts of fibrous tissue that multifocally merges with other islands of dysplastic cartilage.

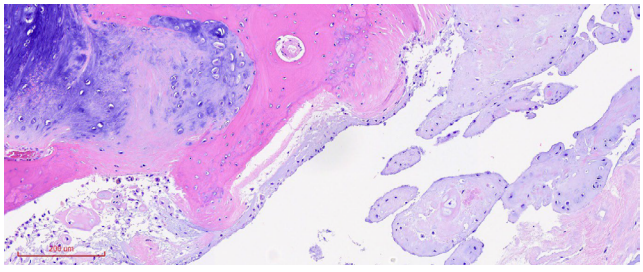

Figure 7. **Subchondral synovial cyst**. The cyst is lined internally by one or more layers of cells ranging from spindle-shaped to polygonal (synoviocytes), occasionally covering papillary projections of cartilaginous matrix, and externally by variable amounts of fibrous tissue that multifocally merges with other islands of dysplastic cartilage.

summarizes the alterations in the organization of cartilage growth and its ossification process. The term osteochondrosis as a differential diagnosis for this case, implies a lytic lesion of the cartilage that was not observed, accompanied by reactive changes of the subchondral bone. In particular, the importance of these findings can be highlighted as a possible state of growth disorder prior to osteochondrosis, with epiphyseal dysplasia being a primary lesion that would lead to osteochondrosis and its stages, given the necessary stimuli. This idea of dyschondroplasia and subsequent osteochondrosis has been suggested before, although early cases where there is purely growth disorder in cartilage have not been reported (Jeffcott 1997; Jeffcott & Henson 1998; Beck et al 2002). This case is not consistent with other lytic or inflammatory processes, such as osteomyelitis or a focal infarction. No evidence of vascular changes, inflammatory infiltrate, necrosis, or sequestration was found during histopathologic examination.

A case of compressive myelopathy secondary to focal vertebral dysplasia secondary to focal necrosis that compromised the diaphysis, and metaphysis was described in a foal. In that case, a vascular origin of the alteration is proposed, which would be explained by polyphasic ischemic events that induced changes in the growth cartilage and bones that compose the vertebra (Yang et al., 2018). Necrosis likely originated from an ischemic event, specifically associated with irrigation from the nutritious vertebral

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artery was supported by the location of the lesion (restricted to irrigation areas, diaphysis and physis). The epiphysis, irrigated by epiphyseal arteries, were intact. Nevertheless, the interpretation of the selective distribution of these lesions must be further investigated because the arterial irrigation of the vertebral components varies according to the species (Wirth et al., 2002; Craig et al., 2016). Regarding the subchondral synovial cyst, the they are defined as a periarticular synovium-lined structures filled with synovial fluid. It is proposed that the pathogenesis of these lesions is a joint capsule herniation due to increased intra-articular pressure (Craig et al., 2016). In the present case, we speculate that this focal lesion is a reactive developmental alteration secondary to the intervertebral disc disease.

References

1. Beck C, Middleton D, Maclean A Lavelle R. Osteochondrosis of the second cervical vertebra of a horse. Equine Vet J 2002;34(2): 210–2.
2. Bergknut N, Rutges JP, Kranenburg HJ, Smolders LA, Hagman R, Smidt HJ, Lagerstedt AS, Penning LC, Voorhout G, Hazewinkel HA, Grinwis GC, Creemers LB, Meij BP, Dhert WJ. The dog as an animal model for intervertebral disc degeneration? Spine 2012; 37(5):351–8.
3. Bergmann W, Bergknut N, Veraa S. Gröne A, Vernooij H, Wijnberg ID, Back W, Grinwis GCM. Intervertebral disc degeneration in Warmblood horses: Morphology, grading and distribution of lesions. Vet Pathol 2018; 55(3): 442–52.
4. Yang C, Weisbrode S, Yardley J, Schroeder E, Premanandan C. Metaphyseal and Diaphyseal Dysplasia of the Third Cervical Vertebra Secondary to Physeal Necrosis in a Quarter Horse Foal. J Comp Path 2018; 163, 38–41.
5. Craig LE, Dittmer KE, Thompson. Bones and joints. In: Maxie MG, ed. Jubb, Kennedy, and Palmer's Pathology of Domestic Animals. 6th ed. Vol. 1. St. Louis, MO: Elsevier; 2016. p. 17–163.
6. Janes JG, Garrett KS, McQuerry KJ, Waddell S, Voor MJ, Reed SM, Williams NM, MacLeod JN. Cervical Vertebral Lesions in Equine Stenotic Myelopathy. Vet Pathol. 2015; 52(5) 919–927.
7. Jeffcott, LB & Henson FMD. Studies on growth cartilage in the horse and their application to aetiopathogenesis of dyschondroplasia (osteochondrosis). Vet J 1998; 156,177–92.
8. Jeffcott LB.. Osteochondrosis in horses. In Pract 1997; 19, 64-71.
9. Levine JM, Adam E, MacKay RJ, Walker MA, Frederick JD, Cohen ND. Confirmed and presumptive cervical vertebral compressive myelopathy in older horses: a retrospective study (1992-2004). J Vet Intern Med 2007; 21: 812–19.
10. Nout YS & SM Reed. Cervical vertebral stenotic myelopathy. Equine Vet Edu 2003; 15: (4)212–23.