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SPONTANEOUSLY ARISING DISEASE

Metaphyeal and Diaphyseal Dysplasia of the Third Cervical Vertebra Secondary to Physeal Necrosis in a Quarter Horse Foal

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Summary

Ischaemia-induced physeal injury has not been described previously in the horse. A 1-month-old Quarter horse foal was submitted for necropsy examination due to an acute onset of ataxia followed by a 4-week history of progressive decline. Focal narrowing of the spinal canal due to ventral compression by the rotation of the cranial aspect of the third cervical vertebra (C3) was observed. The metaphysis and diaphysis of C3 were markedly shortened and white–tan in colour. Microscopically, there was complete loss of the dorsal compact bone of C3 and replacement of 80% of the physis that runs parallel to the vertebral canal with fibrous tissue and thickened viable trabecular bone. Both cranial and caudal physes of C3 showed widespread bands of coagulative necrosis of the hypertrophic and calcifying zones. Marked bone marrow hypoplasia with slight fibrosis was observed in the metaphyses and diaphysis. There was no evidence of fracture or inflammation. The epiphyses were microscopically unremarkable. It was hypothesized that a regional transient incomplete and possibly multiphasic ischaemia involving the nutrient artery caused necrosis of the physes, resulting in dysplasia of the bone. Ischaemic injury to the physis should be considered in the pathogenesis of focal bone dysplasia in horses.

Keywords: bone dysplasia; horse; physeal necrosis; vertebra

A 1-month-old Quarter horse filly was evaluated by The Ohio State University Veterinary Medical Center Equine Field Services for acute onset of lethargy, inability to stand and a 1-week history of watery diarrhoea. Two days prior to presentation, the foal reportedly fell over and was unable to rise. On initial examination at the farm, the foal was laterally recumbent, dull and had stupor-like mentation. There was elevation of the heart rate (100 beats/min), respiratory rate (48 breaths/min) and rectal temperature (39.4°C). Cardiothoracic auscultation was within normal limits. The oral mucous membranes appeared pink and moist with a capillary refill time of <2 sec. Borborygmus was mildly increased in all quadrants. The external umbilical structures were within normal limits and no evidence of external trauma was observed. The foal was treated with intravenous isotonic fluids prior to referral. Further diagnostics were performed at The Ohio State University Gallbreath Equine Center. The neurological assessment of the foal was limited due to her inability to stand and ambulate. Cranial nerve examination was within normal limits; however, the foal did have decreased anal tone with mildly decreased tail tone. Spinal reflexes were deemed to be normal in all four limbs. The foal was assisted to stand and was able to bear weight on all limbs, but required a significant amount of support and balance to remain standing, leading to an interpretation of spinal ataxia and neuromuscular weakness. The dull mentation persisted despite significant attempts at stimulation and the foal was unable to support her head for more than several seconds at a time. The neuroanatomical lesion was localized to the cerebrum and the cervical spinal cord segment.

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Thoracic and abdominal ultrasonography revealed no significant abnormal findings. Haematological abnormalities included an elevated white blood cell count (12.7×10^9/l; reference interval [RI]: $4.1–9.7 \times 10^9$/l) with an increase in segmented neutrophils (9.7×10^9/l; RI: $2.4–7 \times 10^9$/l) and no band neutrophils. The remainder of the haematological findings, serum biochemical profile and serum ammonia concentration were all within normal limits. Urinalysis was unremarkable, with a normal hyposthenuric specific gravity of 1.004. The owner declined further diagnostics and elected medical management.

The foal was administered supportive care for 24 h and was then discharged from the hospital back to the farm with continuance of medical management. The foal regained normal neurological mentation; however, the spinal ataxia and neuromuscular weakness remained and the foal further declined to become completely recumbent over the following month. Due to normalization of the cerebral signs and the inability of the foal to right itself and stand, cervical trauma with potential cervical vertebral body fracture was suspected. The foal was humanely destroyed due to the poor prognosis.

At necropsy examination, there was severe focal narrowing of the spinal canal due to ventral compression by the clockwise rotated third cervical vertebra (C3) in sagittal sections of the vertebral column (Fig. 1). The metaphyses and diaphysis of C3 were markedly shorter than those of the adjacent vertebrae and were white–tan in colour. The bone, cartilage and bone marrow of the epiphyses of C3 were unremarkable. Samples of the brain, cervical spinal cord, liver, lung, heart, kidney, lymph node, skin, spleen and vertebral body (C3 and fourth cervical vertebra, C4) were fixed in 10% neutral buffered formalin, processed routinely and embedded in paraffin wax. Sections (4 μm) were stained with haematoxylin and eosin (HE). Formalin-fixed vertebral bodies were decalcified with hydrochloric acid prior to embedding, sectioning and staining.

Subgross evaluation of C3 revealed a complete loss of dorsal compact bone with replacement by fibrous tissue (Fig. 2). More than 80% of the physis that runs parallel to the vertebral canal was replaced by thickened viable trabecular bone with infrequent foci of retained cartilaginous matrix. The physis and bone marrow of C4 appeared normal. Both cranial and caudal physes of C3 showed widespread bands of coagulative necrosis of the hypertrophic and calcifying zones with distinct demarcations with the viable proliferative zone (Fig. 3). The bone marrow of the metaphysis of C3 was replaced by fibrous tissue (bone marrow hypoplasia with fibrosis), while the bone, cartilage and marrow of the epiphyses were microscopically unremarkable (Fig. 3). There was mild fibrosis in the metaphyses and marked formation

Fig. 1. Metaphyseal and diaphyseal dysplasia of the third cervical vertebra (C3) in a Quarter horse foal. There is severe focal narrowing of the spinal canal due to ventral compression by the clockwise rotated C3 in sagittal sections of the vertebral column. The metaphysis and diaphysis of C3 are white–tan in colour and are markedly shorter than those of the adjacent vertebrae (square). The epiphyses appeared unaffected. The physis that runs parallel to the vertebral canal is still present in the fourth cervical vertebra (arrow). HE. Bar, 1 cm.

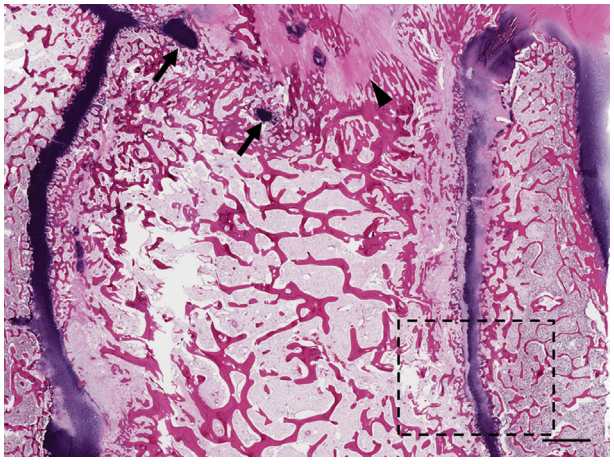

Fig. 2. Photomicrograph of a section from the area of the square in Fig. 1. There is complete loss of the dorsal compact bone. Approximately 80% of the physis that runs parallel to the vertebral canal of C3 and the overlying cortical bones are replaced by fibrous tissue (arrowhead), thickened viable trabecular bone, with infrequent foci of retained cartilaginous matrix of the previous physis that runs parallel to the vertebral canal (arrows). There are widespread bands of coagulative necrosis of the physes (square). There is no evidence of fracture or active inflammation. The bone, cartilage and bone marrow of the epiphyses are microscopically unremarkable. HE. Bar, 2 mm.