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Case Report

Conservative Management of a Cervical Vertebral Fracture in a Gelding

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ABSTRACT

The presentation, diagnostic evaluation, treatment, and 5 years follow-up of a 12-year-old Arabian-Saddlebred cross gelding with neck pain and stiffness, attributable to a fracture of the third cervical vertebrae (C3), is described. Initial cervical spinal radiographs revealed a concave defect in the ventral aspect of the cranial end plate of C3. However, both this finding and ultrasonographic imaging of the area were inconclusive for a fracture. Nuclear scintigraphy revealed that the lesion was metabolically active, prompting computed tomographic imaging that revealed a fracture of C3. Sequential radiographs documented progressive fusion of C2-C3 and no neurological deficits developed over the 5 years after the injury. Cervical vertebral injuries in horses can lead to various clinical signs including ataxia, weakness, and neck stiffness or pain. Diagnosis with cervical radiographs alone can be challenging and, in some cases as the horse in this report, multiple imaging modalities may be required to establish a definitive diagnosis. Horses without neurological signs may recover successfully with conservative medical management, which was performed in this case.

1. Introduction

Cervical injuries and vertebral fractures in horses often lead to ataxia and weakness that sometimes can be catastrophic because of damage to the spinal cord [1,2]. In a retrospective study of 450 horses with neurological disease, 60 were reported to have spinal cord injury and vertebral fractures were found in 38 cases, with the majority in the cervical region. Furthermore, case fatality rate for spinal cord injury was high with the diagnosis established by necropsy examination in 46 horses [3]. However, cervical vertebral fractures are not always accompanied by neurologic deficits and several cases, notably younger horses, have been successfully managed conservatively with rest and analgesic medications [4–6]. Thus, presenting signs for cervical vertebral fractures can vary from neck stiffness and pain to severe neurological deficits resulting in recumbency or sudden death. Although bone abnormalities may be apparent on routine cervical radiographs, detailed documentation of fracture lines and bone fragments can be challenging [1,2]. Thus, in many cases, additional imaging modalities including scintigraphy, ultrasonography, and computed tomography (CT) may need to be pursued to establish a definitive and accurate diagnosis [7].

1.1. Case History

A 12-year-old Arabian-Saddlebred cross gelding was presented for evaluation of neck pain and stiffness. Two weeks before, the horse noted to be normal in the morning, and was acting quiet and did not greet the manager as was usual later that day. No evidence of external trauma was detected, but over the next couple of days, the gelding was repeatedly observed to hold the head in a mildly extended

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position and the neck appeared stiff. Examination by the referring veterinarian revealed an alert horse with normal vital parameters, but neck movement was restricted and the gelding appeared painful on manipulation of the neck. No neurological deficits were apparent at rest or while walking. Treatment included flunixin meglumine (1.0 mg/kg IV) and dexamethasone (0.1 mg/kg IV), and the horse was started on phenylbutazone (4.4 mg/kg PO, q 12 hours) for 7 days. The horse continued to eat and drink, although it was reluctant to eat from the ground, but minimal improvement in neck stiffness was noted over the next few days. Consequently, a blood sample was collected for serum western blot testing for antibodies against *Sarcocystis neurona* and yielded negative results. Because no significant improvement had been observed over the week of treatment, the horse was referred for further evaluation. The only other previous problems were several sarcoids on the inner thighs and a few small melanomas on the tail that had responded well to surgical removal. Up until the time of suspected injury, the gelding had been used for light trail riding several times a week.

1.2. Clinical Findings and Diagnostic Evaluation

At admission, the gelding was bright, alert, and responsive and all vital parameters were within normal ranges. No asymmetry was noted in the neck musculature, although a small (5 mm diameter) nonpainful crusted lesion was found on the upper right side. The horse was reluctant and appeared painful when encouraged to bend the head and neck in either direction to follow a handful of grain toward the elbow; however, it could eat grain from the floor without signs of pain. A localized site of pain could not be ascertained. No gait deficits were apparent when walking or turning in tight circles. However, when circling, the gelding exhibited clear reluctance to flex the neck to either side, and head and neck position was maintained in extension. All other physical and neurological examination findings were normal. A complete blood count (CBC) and serum biochemistry profile, performed to assess potential adverse effects of previous treatment with nonsteroidal anti-inflammatory drugs (NSAIDs), yielded normal results.

Standing lateral radiographs of the cervical spine revealed mild arthritis in the articular facet joints of the lower neck (C5-C6 and C6-C7), but a more notable finding was a concave lucency in the ventral aspect of the cranial end plate of C3 with a thin adjacent mineral fragment (Fig. 1A). No fracture lines or larger displaced bone fragments were apparent. In addition to trauma, infectious or neoplastic destruction of bone was considered, but a lack of periosteal reaction and proliferation of adjacent bone made these differentials less likely. Avascular necrosis or osteochondrosis with bone resorption were other possible causes. Further diagnostic evaluation including ultrasonography, nuclear scintigraphy, and CT were discussed, but the owner elected ongoing conservative management. The horse was discharged with instructions for stall rest for 1 month that included daytime turnout in a small paddock. Further NSAID treatment consisted of phenylbutazone (2.2 mg/kg PO, q 12 hours for 3 days followed by 2.2 mg/kg PO, q 24 hours for 10 days).

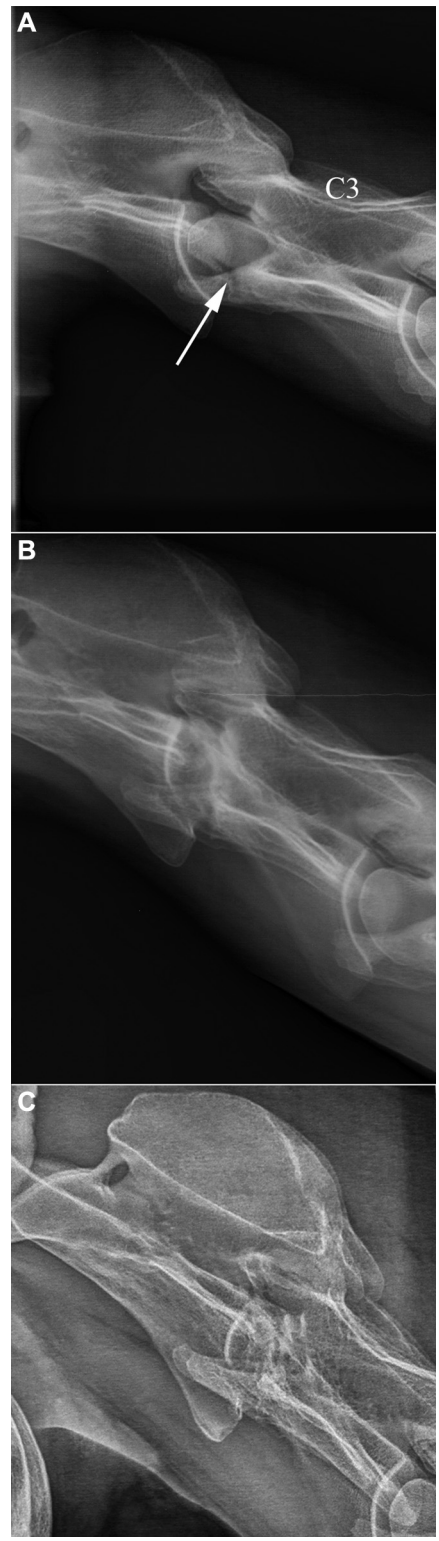

Fig. 1. Standing lateral radiographs taken 2 (A), 24 (B), and 234 (C) weeks after onset of clinical signs. (A) Concave defect in the ventral aspect of the cranial end plate of C3 with a small adjacent fragment (white arrow); (B) remodeling of the lesion with collapse of the C2-C3 disc space, surrounding sclerosis and kyphosis of the spine in this region; (C) apparently healed lesion with static kyphosis.