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Caudal cervical vertebral osteoarthritis: a cause of neurologic disease, lameness or an incidental finding?

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The cervical vertebrae of horses have large dorso-lateral articular processes with obliquely-oriented, plane cartilaginous surfaces that facilitate lateral movement of the head and neck (Nickel, Schummer et al. 1992; Wissdorf, Otto et al. 2002). Degenerative osteoarthritis of the caudal cervical vertebrae articular processes is assumed to occur secondarily to early osteochondrosis caused by genetic and/or nutritional influences (Stewart, Reed et al. 1991; Mayhew 1999; Nixon 2002; Nout and Reed 2003) and is observed commonly, although there is no particular breed predilection. Determining the clinical significance of osteoarthritic lesions in these vertebrae is however difficult. Articular process degenerative joint disease (DJD) plays a significant role in the pathogenesis of equine cervical vertebral stenotic myelopathy (CVSM), known as 'wobbler syndrome', a common cause of ataxia and weakness (Powers, Stashak et al. 1986; Moore, Holbrook et al. 1992; Nout and Reed 2003). Cervical static stenosis, unlike the dynamic form of the disease (which is generally seen in younger horses), is seen at all ages but frequently in older animals (Powers, Stashak et al. 1986; Wagner, Grant et al. 1987; Nout and Reed 2003). The condition occurs mainly between C5-C7 and is caused by osteoarthritic of the articular process joints (Powers, Stashak et al. 1986; Wagner, Grant et al. 1987; Tomizawa, Nishimura et al. 1994; Nixon 2002; Nout and Reed 2003). Cartilage erosion, subchondral bone sclerosis and periarticular osteoporosis can be seen. The latter often leads to further growth and enlargement of the perimeter of the processes, which can, if directed medially, cause spinal cord compression. Although spinal cord compression is observed at sites with no articular lesions, osteochondrosis and other lesions are more severe in joints at sites of compression than in joints at non-compressed sites in horses with CVSM (Stewart, Reed et al. 1991). Furthermore, hypertrophy and distension of the joint capsule and synovial cysts can compress the spinal cord medially (Whitwell and Dyson 1987; Nixon 2002). Indeed, synovial folds of varying sizes containing adipose tissue are commonly detected in these joints post mortem (Berg, Nielsen et al. 2003) and synovial cysts are occasionally identified (Powers, Stashak et al. 1986; Trostle, Dubielzig et al. 1993). Not all horses however with DJD have clinical signs compatible with cord compression (Moore, Holbrook et al. 1992; Mayhew, Donavick et al. 1993; Moore, Reed et al. 1994; Tomizawa, Nishimura et al. 1994). Indeed, although 80% of horses with CVSM display radiographic DJD, 48% of clinically-normal, non-ataxic horses show moderate degenerative radiographic signs, (Moore, Reed et al. 1994) particularly in the more caudal cervical vertebrae (Tomizawa, Nishimura et al. 1994). Similarly, Whitwell and Dyson detected that up to 50% of adult horses have articular process degenerative changes at C6-C7 (Whitwell and Dyson 1987). The condition has however been associated with neck pain, localised cervical muscle atrophy and cutaneous hypesthesia, stiffness and forelimb lameness (Moore, Holbrook et al. 1992; Ricardi and Dyson 1993; Nout and Reed 2003; Mattoon, Drost et al. 2004). Making a definitive diagnosis in these latter cases however is difficult, and often involves the exclusion of other problems. Identification of intervertebral foramen impingement on spinal nerve roots has been documented using computed tomography however (Moore, Holbrook et al. 1992) and electromyography and/or muscle biopsy can aid in the diagnosis of neurogenic atrophy. Intra-articular medication with corticosteroids and/or hyaluronic acid, is currently used by some as a therapeutic approach to cervical articular process DJD, or occasionally as a diagnostic procedure to evaluate joint pain (Nielsen, Berg et al. 2003; Nout and Reed 2003; Mattoon, Drost et al. 2004). This procedure is performed with the rationale of reducing soft tissue swelling and joint distension and stabilising or preventing further bony proliferation (Grisel, Grant et al. 1996; Grant 1999). In humans, intra-articular injection of long-acting corticosteroids and/or local anaesthetic is routinely used for relief of symptoms (such as pain) associated with articular process joint arthropathy (Sarazin, Chevrot et al. 1999) but with questionable efficacy (Barnsley, Lord et al. 1994). In human patients with degenerative changes and synovial cysts, corticosteroid injection is however a useful alternative to their surgical removal (Sauvage, Grimault et al. 2000). Likewise, subjective clinical improvement is seen in dogs, following similar procedures (Kinzel, Hein et al. 2003). With practise, ultrasound guided joint medication (Nielsen, Berg et al. 2003) can be used to introduce medication peri- or intra-articularly (Nielsen, Berg et al. 2003; Mattoon, Drost et al. 2004). Injections are generally performed bilaterally and up to 6 joints may be medicated at any one time. Up to 40 mg of methylprednisolone acetate and 125 mg amikacin are injected at each site, although lower doses are used if more than 4 joints are medicated simultaneously due to concerns about precipitating laminitis. Collective
anecdotal evidence suggests that joint medication may be used with success in some horses with caudal cervical vertebral osteoarthritis; however, unsurprisingly, some horses may require repeat treatment. A blinded clinical trial is necessary however to evaluate the efficacy of this technique objectively.

Ventral stabilisation surgery is more commonly used to treat dynamic compression (type 1) CVSM lesions, however clinical evidence suggests that articular process remodeling occurs after long term follow up of horses that have undergone this surgery, making this a potential option in valuable animals with spinal cord compression (Moore et al. 1993) or neck pain (Nout and Reed 2003). Clinical improvement may take considerable time however or may not be complete. In conclusion, articular process osteoarthritis of the caudal cervical vertebrae may be recognized as an incidental finding, however such a finding in an ataxic or lame horse, or one exhibiting neck pain should be regarded with suspicion. Comprehensive clinical, neurological and lameness examinations in conjunction with ancillary diagnostic techniques, such as radiography, myelography, electromyography and computed tomography, will likely aid in diagnosis. Often however, response to treatment may be the best way to determine the significance of the lesions.

REFERENCES