CASE REPORT

Cervical Intervertebral Disc Protrusion in Two Horses

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Summary

Two horses with ataxia of all four limbs were found to have cervical intervertebral disc protrusion. Severe pelvic limb ataxia, proprioceptive deficits and spasticity were present in both horses with similar but less severe signs in the thoracic limbs. Cerebrospinal fluid analysis was within normal limits. Metrizamide myelography allowed definitive diagnosis in one case when a compression of the spinal cord was demonstrated at the level of the second intervertebral space. In the second case, an intervertebral disc protrusion between cervical vertebrae 6 and 7 was found at necropsy. Fiber degeneration with poor myelin staining characterized the spinal cords histologically.

Résumé

Protrusion de disques intervertébraux cervicaux, chez deux chevaux

Les auteurs ont décelé une protrusion de quelques disques intervertébraux cervicaux, chez deux chevaux qui manifestaient une ataxie locomotrice des quatre membres. Les membres pelviens de ces deux chevaux affichaient de l'ataxie locomotrice, des déficits proprioceptifs et de la spasticité; leurs membres thoraciques manifestaient des signes similaires, mais moins prononcés. Une analyse du liquide céphalo-rachidien ne révéla rien d'anormal. Une myélographie, avec de la métrizamide, permit de poser un diagnostic définitif, dans un cas, parce qu'elle démontra la présence d'une compression de la moelle épinière, au niveau du deuxième espace intervertébral. Dans l'autre cas, la nécropsie révéla une protrusion du disque intervertébral entre les sixième et septième vertèbres cervicales. Les lésions microscopiques de la moelle épinière se caractérisaient par de la dégénérescence des fibres nerveuses et une mauvaise coloration de la myéline.

Introduction

Intervertebral disc protrusion has been described in several species but is apparently rare in the horse, as there is only one report (1). The following is a report of two horses with cervical intervertebral disc protrusion.

Case 1

A six year old Paint stallion was admitted to the Iowa State University College of Veterinary Medicine with the complaint of pelvic limb ataxia and spasticity. The horse had been found down in his stall approximately one week earlier. A tentative diagnosis of myositis had been made and treatment initiated with flunixin meglumine,[1] B-vitamins, furosemide,[2] procaine penicillin G and sodium bicarbonate. The horse stood up within a day, but remained very stiff and ataxic. Over the next several days no improvement was noted and the horse was referred for further diagnostics.

At admission, a spastic, dysmetric, ataxic gait was noted in both the pelvic and thoracic limbs, with the pelvic limbs being more severely affected. No paresis was evident. Cutaneous sensation and cranial nerve function were within normal limits. Although the horse was alert, he was reluctant to move. There was a proprioceptive deficit in the rear limbs as evidenced by delayed foot replacement from the cross-legged stance and a positive sway response. All hooves scuffed during limb protraction, and when circled the rearlimbs exhibited excessive circumduction. The horse resented having the head turned to the left. A lesion of the cranial portion of the cervical spinal cord was suspected.

Creatine phosphokinase, serum glutamic-oxalocetic transaminase, blood ureau nitrogen and venous pH values were slightly elevated, otherwise routine hematology and serum chemistries were within normal limits.

Approximately three weeks after onset of signs the horse was anesthetized with glyceryl guaicolate and maintained with halothane for cervical radiography and myelography. Cerebrospinal fluid (CSF) was withdrawn from the atlanto-occipital site for analysis and cytology. All CSF parameters were within normal limits (2).

Plain radiographs of the cervical vertebrae showed an active collapse of the C-2/3 intervertebral disc space with accompanying sclerosis of the opposing endplates. There were no obvious signs of cervical vertebral malformation. The appearance at C-2/3 was interpreted as a possible disc protrusion (Figure 1).

A myelogram using metrizamide[3] demonstrated a ventral extradural

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[1]Banamine®, Schering Corporation, Kenilworth, New Jersey.

[2]Lasix®, Taylor Pharmaceutical, Decatur, Illinois.

[3]Nyegaard & Co., Oslo, Norway.

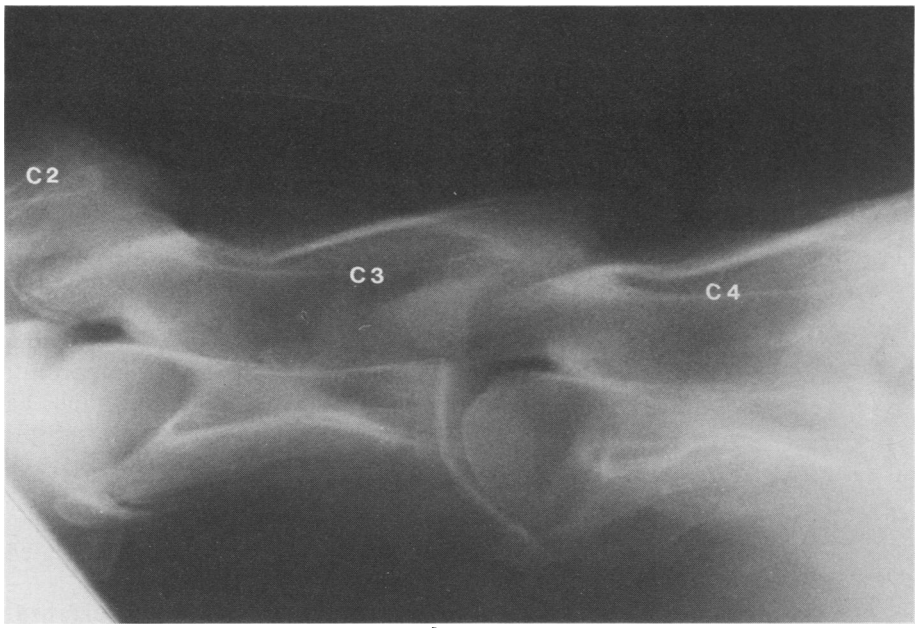

FIGURE 1. Survey cervical radiograph of horse 1, showing collapse of the intervertebral disc space between C-2/3. The opposing endplates are markedly sclerotic when compared to C-3/4.

lesion at the level of C-2/3. With the neck in a neutral lateral position the degree of compression on the spinal cord was minimal. With the head and neck in ventral flexion the degree of spinal cord compression was slightly increased (Figures 2a and 2b). No other lesions were identified on the myelogram. Measurement of the minimum flexion diameter (MFD), minimum sagittal diameter (MSD) were within the normal ranges reported in the literature for a horse of greater than 320 kg body weight (3,4). These myelographic findings supported the suspicion of an intervertebral disc protrusion.

Due to an unfavorable prognosis the owners elected euthanasia one month after the initial episode. At necropsy the intervertebral disc material and cartilage was absent on the opposing surfaces of cervical vertebral bodies 2 and 3, protruding dorsally about 5 mm into the vertebral canal. Approximately 2 mm of underlying bone was eburnated on both sides of the joint space (Figure 3).

Histologically the white matter of the cervical cord was characterized by fiber degeneration with poor myelin staining and scattered throughout all funiculi, but concentrated in the ventral and lateral funiculi in the first cervical segment and at the point of disc protrusion. Necrosis of individual neurons within the gray matter was noted at the level of the second and third cervical vertebrae.

Case 2

A three year old Arabian-saddlebred cross gelding was presented with a history of pelvic limb ataxia of sudden onset and two weeks duration. Treatment had consisted of furosemide, phenylbutazone, and dexamethasone with no response.

At admission physical examination revealed severe ataxia and proprioceptive deficits in the pelvic limbs with similar but slightly less severe signs in the thoracic limbs. Movement of all limbs was spastic and dysmetric. No paresis was evident. A positive sway response and delayed foot replacement from the cross-legged stance was exhibited most notably in the pelvic limbs. Pain was not apparent during cervical palpation and manipulation.

Anesthesia was induced with glyceryl guaicolate and thiamylal and maintained with halothane for cervical radiography and collection of CSF from the atlanto-occipital space. The CSF was within normal limits (2). No abnormalities were noted on plain radiographs, with or without cervical flexion, although insufficient exposure of the caudal cervical vertebrae hampered evaluation. The owner elected not to have myelography performed.

Routine hematology and serum enzyme levels were within normal lim-

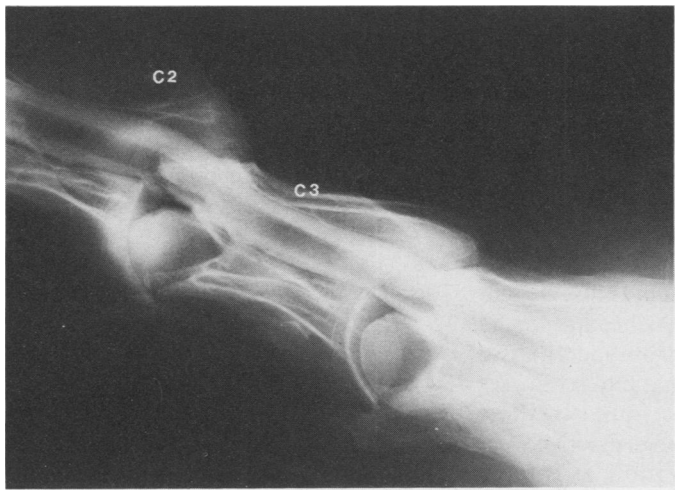

FIGURE 2a. Myelographic study of horse 1, demonstrating some narrowing of the ventral subarachnoid contrast column at the C-2/3 disc space.

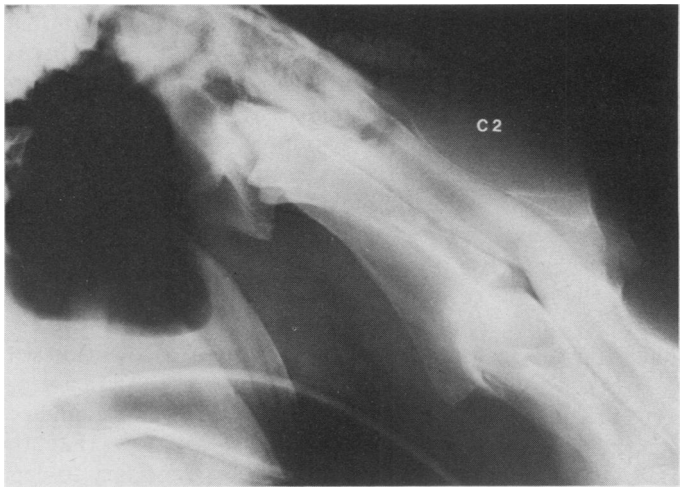

FIGURE 2b. With ventral flexion of the head the narrowing of the ventral subarachnoid space is accentuated.

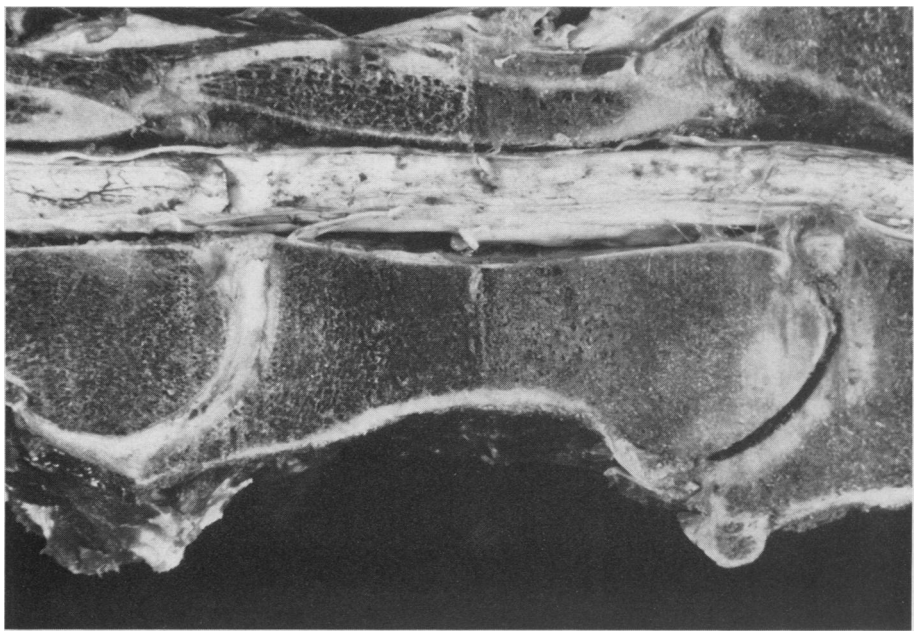

FIGURE 3. Sagittal section through the spinal column of horse 1. C-3/4 (left) and C-2/3 (right) showing collapse of the C-2/3 intervertebral disc space with subchondral eburnation as well as protrusion of the disc.

its. Clinical improvement was not noted over the next two weeks and the owner requested that the horse be euthanized.

Intervertebral disc protrusion between cervical vertebrae 6 and 7 compressed the spinal cord in that area. Histologically, there was fiber degeneration of the dorsal, lateral, and ventral columns of the spinal cord in the area of protrusion. Microglial cells were scattered, sometimes occurring in clumps of three to five. Myelin balls, swollen axons and empty spaces were numerous.

Discussion

With the advent of surgical treatment of selected spinal cord disease in the horse (5,6), accurate diagnosis is becoming increasingly more important to provide appropriate therapy and prognosis. The clinical signs shown by these horses with cervical intervertebral disc protrusion mimicked those of cervical spinal cord compression from other causes, including cervical stenotic myelopathy (3,7-11), vertebral fractures (12-14), vertebral luxations (15), occipitoatlantoaxial malformation (16) and neoplasia (17). Noncompressive entities such as thromboembolic ischemic myelopathy (18), rhinopneumonitis myeloencephalopathy (19-23), protozoal myelitis (3,19), parasitic myelitis (3,19,24,25), equine infectious anemia (26) congenital abnormalities of venous drainage (27) and degenerative myeloencephalopathy (3,28) should also be considered in the differential diagnosis.

Diagnosis in these cases, after the lesion was localized to the cervical spinal cord, was based on history, cerebrospinal fluid analysis and cytology, apparent neck pain, radiography, and postmortem evaluation. History of a relatively rapid onset when combined with equine herpesvirus 1 serology and CSF analysis helped rule out infectious, inflammatory and neoplastic spinal cord disease. The apparent neck pain in horse 1 could be attributed to a fracture, vertebral osteoarthritis, or a compressive lesion causing meningeal or nerve root pain (29). The definitive diagnosis was reached with myelography in the first horse and necropsy in the second.

Although intervertebral disc degeneration in the horse has been reported in association with thromboembolism (18) and in asymptomatic older horses (30) no evidence was found in these animals that the protrusions were secondary to degeneration, as is often the case in the chondrodystrophoid dog (29). Instead, these protrusions may be of traumatic origin.

The histological spinal cord lesions exhibited were similar to those noted in cervical disc protrusions in the canine. In the dog, the clinical signs seen are dependent on the location, force, extent, speed, and duration of the protrusion (29). After the initial edema subsides, the spinal cord lesion can be produced in several ways. An extruded nucleus causes an epidural inflammatory reaction. The physical presence of the disc with resulting compression of the cord is important. Vascular derangements such as reduced blood flow in the ventral spinal artery and intramedullary branches with resultant ischemia, as well as venous obstruction causing edema, have also been incriminated (30-33).

Although cervical intervertebral disc protrusion is apparently a rare occurrence, it warrants addition to the list of causes of ataxia in the horse.

References

1. McKELVEY WAC, OWEN RapR. Acquired torticollis in eleven horses. J Am Vet Med Assoc 1979; 175: 295-297.
2. MAYHEW IG, WHITLOCK RH, TASKER JB. Equine cerebrospinal fluid: reference values of normal horses. Am J Vet Res 1977; 38: 1271-1274.
3. MAYHEW IG, DELAHUNTA A, WHITLOCK RH, KROOKER L, TASKER JB. Spinal cord disease in the horse. Cornell Vet 1978; 68 suppl. 6.
4. NYLAND TG, BLYTHE LL, POOL RR, HELPHREY MG, OBRIEN TR. Metrizamide myelography in the horse: Clinical radiographic and pathologic changes. Am J Vet Res 1977; 41: 204-211.
5. WAGNER PC, GRANT BD, BAGBY GW, GALLINA AM, SANDE RD. Evaluation of cervical spinal fusion as a treatment in the equine "wobbler" syndrome. Vet Surg 1979; 8: 84-88.
6. WAGNER PC, BAGBY GW, GRANT BD, GALLINA A. Surgical stabilization of the equine cervical spine. Vet Surg 1979; 8: 7-12.
7. BEECH J. Cervical cord compression and wobbles in horses. Proc Am Assoc Eq Pract 1976; 22: 78-79.
8. FRASER J, PALMER AC. Equine incoordination and wobbler disease in young horses. Vet Rec 1967; 80: 338-355.
9. YAMIGIWA J, YOSHIKAWA R, OYAMADA T. Pathological studies on equine ataxia in Japan. Jpn J Vet Sci 1980; 42: 681-694.
10. SAKURA Y. Histopathological changes in the nervous system in "equine incoordination" — observations on eight affected foals including cases of the earliest neonatal onset. Jpn J Vet Res 1967; 25: 30-35.
11. MORDRAKOWOKI A, KSZUBIEWICZ C. Correla-

tion between clinical symptoms and localization of anatomical lesions in spinal ataxia (wobbler disease in horses). Med Weter 1977; 33: 712-716. (Vet Bull 1978; 48: 601).
12. SCHEBITZ H, DAHME E. Spinal ataxia in the horse. Proc Am Assoc Eq Pract 1967; 13: 133-148.
13. FRASER H, PALMER AC. Equine incoordination and wobbler disease of young horses. Vet Rec 1967; 80: 338-353.
14. DEBOWES RM, WAGNER PC, GAVIN PR, RODER PH. Vertebral compression fracture in a foal following electric shock. J Vet Orth 1981; 2: 14-19.
15. GUFFY M, COFFMAN J, STRAFUSS, A. Atlantoaxial luxation in a foal. J Am Vet Med Assoc 1969; 155: 754-757.
16. MAYHEW IG, WATSON AG, HEISSAN JA. Congenital occipito-atlantoaxial malformations in the horse. Eq Vet J 1978; 10: 103-113.
17. TRAVER D, MOORE J, THORNBURG L, JOHNSON J, COFFMAN J. Epidural melanoma causing posterior paresis in a horse. J Am Vet Med Assoc 1977; 170: 1400-1403.
18. TAYLOR H, VANDEVELDE M, FIRTH E. Ischemic Myelopathy caused by fibrocartilaginous emboli in a horse. Vet Path 1977; 74: 479-481.
19. DELAHUNTA A. Neurological problems of the horse. Proc Am Assoc Eq Pract 1977; 19: 25-32.
20. BITSCH V, DAM A. Nervous disturbances in horses in relation to injection with equine rhinopneumonitis virus. Acta Vet Scand 1971; 12: 134-138.
21. JACKSON T, KENDRICK JW. Paralysis of horses associated with equine herpes virus I infection. J Am Vet Med Assoc 1971; 158: 1351.
22. JACKSON TA, OSBURN BI, CORDY DR, KENDRICK JW. Equine herpes virus I infection of horses: Studies on the experimentally induced neurologic disease. Am J Vet Res 1977; 38: 709-719.
23. PURSELL AR, SANGSTER LT, BYARS TD, DIVERS TJ, COLE JR. Neurologic disease induced by equine herpes virus I. J Am Vet Med Assoc 1979; 175: 473-474.
24. FRAUNFELDER HC, KAZACOS DR, LICHTENFELS JR. Cerebrospinal nematodiasis caused by a filariid in a horse. J Am Vet Med Assoc 1980; 177: 359-362.
25. LITTLE PB. Cerebrospinal nematodiasis of equidae. J Am Vet Med Assoc 1972; 160: 1407-1413.
26. McCLURE J, LINDSAY W, TAYLOR W, OCHOA R, ISSEL C, COULTER J. Ataxia in four horses with equine infectious anemia. J Am Vet Med Assoc 1982; 180: 279-283.
27. GILMOUR JS, FRASER JA. Ataxia in a welsh cob filly due to a venous malformation in the thoracic spinal cord. Eq Vet J 1977: 9: 40-42.
28. MAYHEW IG, DELAHUNTA A, WHITLOCK RH, GEARY JC. Equine degenerative myeloencephalopathy. J Am Vet Med Assoc 1977; 170: 195-201.
29. HOERLEIN BF. Canine neurology. Philadelphia: W.B. Saunders, 1978: 470-560.
30. ROONEY JR. The horse's back: biomechanics of lameness. Eq Pract 1982; 4: 10-12.
31. LINDBALD G, LJUNGGREN G, OLSSON SE. On spinal cord compression in the dog: an angiographic study. Adv Small Anim Pract 1962; 3: 121-124.
32. TARLOV IM. Spinal cord compression mechanisms of paralysis and treatment. Springfield, Illinois: Charles C. Thomas, 1957.
33. TARLOV IM, KLINGER H, VITALE S. Spinal cord compression studies. I. Experimental techniques to produce acute and gradual compression. Arch Neurol Psych 1950; 70: 813-818.

BOOK REVIEWS

Problems in Small Animal Neurology. C.L. Chrisman. Published by Lea & Febiger, Philadelphia. 1982. 461 pages. Price US$37.50 and C$45.00.

The author uses a rational stepwise approach in the diagnoses and treatment of the neurological problems found in veterinary practice. The first part of the book introduces the reader to the nervous system and to the many ways and means which can be used to investigate the complaints. The chapter dealing with the medical management of the patient will certainly be most useful to many practitioners, as it deals with the practical steps to be taken at the time of the neurological emergency and thereafter.

Much of the second part of the book deals with specialized examination and investigation practices which belong to the realm of the reference neurological clinic. However, it gives an intense understanding of the neurological problems which may be encountered. Diagnosis, prognosis and patient care and treatment are all considered, thus giving an overview of each individual problem. This is specially well demonstrated in the chapter on Behavior and Personality Disorders. All relevant changes, signs or symptoms are well studied, and analyzed.

Relevant references follow at the end of each chapter for those who wish more intensive information.

Although large parts of the book are addressed to the animal neurologists, the book itself allows a better understanding, and will often clarify some cases which are puzzling to the practitioner. *C. Gardell.*

Recent Developments in Ruminant Nutrition. W. Haresign and D.J.A Cole. Published by Butterworths Inc., Massachusetts. 1981. 367 pages. Price $21.50.

This excellent book consists of a series of review articles drawn from the Proceedings of the University of Nottingham Nutrition Conferences for Feed Manufacturers. Many of the articles are by world renowned nutritionists. Half of the book is concerned with protein utilization by the dairy cow and the modifying effects of rumen fermentation on dietary proteins. Veterinarians may prefer to read these chapters last because of the uncertainties in applying this information in the field. There are excellent chapters on milk fever, processing of feeds, silage, complete diets, body condition and production, and concentrate: forage rations. These chapters will offer many veterinarians new insights into feeding dairy cattle. There is helpful information on the causes and prevention of butter fat depression and on the benefits of complete rations versus self-feed grain systems for high producing cows. The benefits of feeding sodium bicarbonate to dairy cows are described. Although the book is not aimed at the beef producer the chapter on processing of grains contains data on how to minimize rumenal upsets on high grain diets, there is also a chapter on growth promotants in cattle. Buy the book but read it selectively. *J.M. Naylor.*