

Nuchal crest avulsion fracture in 2 horses: a cause of headshaking

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ABSTRACT

The medical records of 2 Thoroughbred horses that developed headshaking after blunt trauma to the occipital region are reviewed. The history, signalment, clinical signs, diagnostic methods, diagnosis and treatment were recorded in each case. Both horses displayed headshaking, while one horse repeatedly lifted its upper lip and pawed excessively at the ground. In both horses, diagnostic imaging of the occipital region revealed avulsion fragments of the nuchal crest and a nuchal desmitis in association with hyperfibrinogenaemia. The presence of an avulsion fragment of the nuchal crest with associated nuchal desmitis should be considered in horses presenting with headshaking and may respond favourably to conservative therapy.

Keywords: equine, headshaking, nuchal crest desmitis, trauma.

Voigt A, Saulez M N, Donnellan C M **Nuchal crest avulsion fracture in 2 horses: a cause of headshaking.** *Journal of the South African Veterinary Association* (2009) 80(2): 111–113 (En.). Department of Companion Animal Clinical Studies, Equine Internal Medicine, Faculty of Veterinary Science, Private Bag X04, Onderstepoort, 0110 South Africa.

INTRODUCTION

Although headshaking is a widely recognised disorder, a precise aetiology is seldom found and treatment is often unrewarding. The purpose of this case report is to review the clinical records of 2 horses that developed headshaking after blunt trauma to the occipital region with resultant nuchal crest avulsion fracture and associated nuchal desmitis. The significance of the abnormalities was confirmed by the infiltration of a local anaesthetic agent and an anti-inflammatory substance. This report further explores the heterogeneity of this syndrome by discussing the causes, clinical signs, diagnostic procedures and treatments that may be considered in horses with headshaking.

MATERIALS AND METHODS

The clinical records of 2 horses admitted between October 2007 and June 2008 to the Equine Clinic of the Onderstepoort Veterinary Academic Hospital (OVAH) that had a primary complaint of headshaking are reviewed. The history, signalment, clinical signs, diagnostic methods, diagnosis and treatment were recorded in each case.

CASE HISTORY

Case 1

A 12-year-old, retired Thoroughbred

racehorse gelding developed acute onset of spontaneous and repetitive vertical, horizontal and rotary movement of his head and neck of 3-week duration. The headshaking varied in intensity throughout the day and occurred both at rest and during exercise. A lateral radiograph (Fig. 1) of the occipital region revealed a 22 × 15 mm mushroom-shaped trabeculated mineral opacity visible 30 mm caudal to the nuchal crest with an additional 6 mm opacity located 10 mm crani-

ally to the 1st larger opacity. The nuchal crest appeared irregular and was suspected to be the corresponding fracture bed.

Ultrasonographic examination was performed of the nuchal ligament with a 7.5 MHz T-Piece linear array multi-frequency transducer. The right nuchal ligament had a 23 × 8.4 mm, diagonally up to 18 mm, hyperechoic line which cast a clean acoustic shadow 28 mm caudally to the nuchal crest. An additional 2.7 mm smaller structure could be seen 8 mm cranially to the large one. The right nuchal ligament measured 1.07 cm² vs 0.69 cm² on the left. The nuchal ligament on the right lost its homogenous appearance and the ligament fibres appeared to be hypoechoic.

Following interpretation of diagnostic imaging results, avulsion fragments of the nuchal crest, with an associated nuchal desmitis caused by suspected blunt trauma to the occipital region constituted the most significant finding. Serum biochemical analysis revealed hyperfibrinogenaemia (7 g/l, reference range <4 g/l) which may have been due to inflammation of the nuchal ligament.

The gelding was managed with



Fig. 1: Lateral view of the occipital region of a 12-year-old Thoroughbred racehorse, showing a 22 × 15 mm mushroom-shaped trabeculated mineral opacity located caudal to the nuchal crest (arrow) and a 6 mm opacity located cranial to the 1st larger opacity (arrowhead) (Horse 1).

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Received: March 2009. Accepted: May 2009.

phenylbutazone (Phenylbutazone BP, Kyron Laboratories Pty Ltd) (2.2 mg/kg, q12h, PO), dexamethazone (Kortico, Bayer Ltd.) (0.1 mg/kg, q24h, PO) and trimethoprim-sulfamethoxazole (Purbac[®], Aspen Pharmacare) (25 mg/kg, q12h, PO) to reduce swelling caused by the suspected trauma to the poll. Local infiltration consisting of 45 mg methylprednisolone acetate (DepoMedrol[™], Pfizer), 100 mg lignocaine hydrochloride (Lignocaine Injection 2 %, Bayer Ltd.) and Sarapin[®] (High Chemical Company) was administered in the nuchal ligament in the region of the nuchal fragment. No further headshaking episodes occurred following treatment and the gelding was discharged from the clinic. Follow-up consultation 12 weeks after discharge indicated that no headshaking behaviour had been noticed.

Case 2

A 5-year-old, retired Thoroughbred racehorse gelding developed signs of headshaking at rest and during exercise of 6-month duration. Clinical symptoms included lifting of the upper lip, pawing excessively and resting his head on the ground. Physical examination revealed hypertrophy of the ventral neck muscles. A radiograph (Fig. 2) of the occipital region showed mineral opacities ranging from 5 to 10 mm adjacent to the nuchal crest external occipital protuberance which appeared irregular and was suspected to be the corresponding fracture bed. These mineral opacities had a rounded appearance and were considered to be due to trauma to the poll region which occurred several months previously.

Ultrasonographic examination of the nuchal ligament was performed as described above. An 11 mm hyperechoic line could be seen casting an acoustic shadow 10 mm to the right of the midline and 11 mm caudoventral to the nuchal crest. Additional smaller concave hyperechoic lines up to 5 mm on the right and 3 mm on the left of the midline were observed. The nuchal ligament on the right lost its homogenous appearance and the ligament fibres appeared to be hypoechoic with associated loss of fibre alignment. The right nuchal ligament measured 1.9 cm² vs 0.85 cm² on the left.

Following interpretation of diagnostic imaging results, an avulsion fragment of the nuchal crest, with an associated nuchal desmitis was the most significant finding. Serum biochemistry revealed hyperfibrinogenaemia (5 g/l).

Local infiltration consisting of 18 mg triamcinolone acetonide (Vetalog[®], Fort Dodge) and 150 mg ropivacaine hydrochloride (Naropin[®], AstraZeneca Pharmaceuticals) was administered into the



Fig. 2: Lateral oblique radiographic view of the occipital region of a 5-year-old Thoroughbred racehorse, showing mineral opacities ranging from 5 to 10 mm (arrow) adjacent to the nuchal crest external occipital protuberance (Horse 2).

nuchal ligament in the region of the bony fragment. After performing the infiltration, the gelding was lunged and no headshaking behaviour was noticed. Follow-up consultation 1 year after discharge indicated that the gelding's headshaking behaviour had decreased by 90 %.

DISCUSSION

In a retrospective study of 100 horses with headshaking, a specific aetiology could not be identified in 90 % of the cases and in only 2 horses could it be shown that correction of the disorder led to elimination of clinical signs⁶. Several underlying diseases can present with headshaking signs, but the majority of cases are idiopathic^{7,8,17,18}. Irritation by insects, dental disease, guttural pouch mycosis and empyema, otitis media and interna, cranial nerve disorders, ocular disease, subepiglottal cyst, premaxillary bone cyst, ethmoid haematoma, fracture of the frontal bone, maxillary osteoma, protozoal myeloencephalitis and *Trombicula autumnalis* larval infestation are all considered possible causes^{5,6,9,14,16,20}.

Nuchal crest avulsion fractures with corresponding nuchal ligament desmitis was the most significant finding in the 2 cases in this report. Furthermore, the headshaking behaviour decreased in both after the nuchal ligament was infiltrated with local anaesthetic and corticosteroid. Various published reports on equines describe cervical injuries and bony changes to the skull and spine which may cause clinical signs of headshaking⁶. New bone formation may occur in the region of insertion of the nuchal ligament as well as dorsal and ventral to the site of the inser-

tion on the occiput². This may be seen as an incidental finding, but affected horses tend to resist the reins, find difficulty in flexion at the poll and may rear or shake the head²⁴.

Horses with insertional desmitis of the nuchal ligament or injury to the tendon of insertion of semispinalis may have a tendency to shake their heads⁴. These injuries usually occur due to trauma to the region or an excessive amount of lunging exercise while restricted with side or draw reins⁴. In a report of insertional desmopathy of the nuchal ligament, 12 horses presented with various clinical signs such as reluctance to bend the head, stiffness of the neck, excessive head turning and headshaking when exercised¹. Diagnostic procedures performed on the 12 horses indicated exostosis of the protuberantia occipitalis, calcification of the ligamentum nuchae or insertion desmopathy¹.

As found in this report, horses with headshaking commonly show uncontrollable shaking of the head in a vertical, horizontal or rotary direction without any apparent physical stimulus^{7,18}. Other signs include nose rubbing, striking at the nose with the forelegs, head pressing and active avoidance of light, warmth or wind on the face. Worsening of clinical signs with exposure to light are common features of photic headshaking^{7,8,14} and seasonality is demonstrated in half to three quarters of horses which developed clinical signs of headshaking in the spring and early summer that ceased in late summer or autumn^{7,18}. In some cases horses only showed headshaking when exercised⁷ while others only showed it at rest^{14,18}.

Horses with headshaking at rest can be regarded as having a particularly severe form of the disorder as noticed in both cases reviewed in this report.

A thorough appraisal of the case history and the observation of the type of headshaking may give an indication of the underlying aetiology^{11,14}. Intolerance of the bit or problems with the tack are frequently blamed as a cause of headshaking and should be eliminated before further diagnostic procedures are performed. Typically performed diagnostics may include radiography of the head and neck including specific views of the paranasal sinuses and pharyngeal region. As changes to the protuberantia ossis occipitalis may be an incidental finding and not exclusively the cause of headshaking behaviour, ultrasonography of the nuchal ligament or insertion of the semispinalis tendon is indicated to determine the extent of desmopathy. Other diagnostics include nasopharyngeal endoscopy including guttural pouches and the trachea up to the level of the carina, percussion of the paranasal sinuses, tracheal lavage cytology, nasal lavage, skin testing for atopy, pinch mucosal biopsies for histopathological examination for evidence of allergic or inflammatory disease, aural endoscopy, otoscopy, dental examination, neurological examination, local infiltration of local anaesthetic solution of the trigeminal nerve, complete blood count, serum chemistry profile and virus isolation for equine herpes virus 1 and 4^{3,8,11,13,17,18}.

Depending on the cause of the headshaking, therapy differs substantially and is often unrewarding especially when a specific aetiology could not be identified. Idiopathic headshaking may be treated using carbamazepine and cyproheptadine with a reported success rate of >80%¹⁸, cyproheptadine alone with a reported success rate of >70%^{7,8} tracheostomy, perineural anaesthesia/neurectomy of the posterior ethmoid branches of the trigeminal nerve, nasal cavity flushing, contact lenses, and occlusive nasal masks^{15,18}. Bilateral infraorbital analgesia has demonstrated variable success in decreasing the signs of headshaking¹⁸ and is associated with nasal irritation and neuroma formation¹⁰. Caudal compression¹⁹ of the infraorbital nerve has also been described in the treatment of idiopathic headshaking and limits input from the trigeminal nerve at a more caudal location than infraorbital neurectomy¹⁰. The treatment of photic headshaking with antihistamines, corticosteroids, non-steroidal anti-inflammatory agents and hyposensitivity therapy has been unrewarding⁸. Antimicrobials, external parasite

control, chiropractic manipulation, melatonin administration and acupuncture also had limited success⁷. Rhinitis can be managed medically with variable success, using topical and systemic treatment including corticosteroids, antihistamines, sodium cromoglycate, anticholinergics or sympathomimetics¹⁵.

Desmopathy of the nuchal ligament causing headshaking may be treated with repeated infiltration of corticosteroids and local anaesthetic solution and modification of the training programme. The combination of drugs used to infiltrate the nuchal ligament may differ due to availability, cost and previous experience. In this report, an anaesthetic agent was used to provide local anaesthesia and for acute pain management in both horses. Sarapin (a pitcher plant extract) is thought to affect nerves involved in pain sensation while corticosteroids may be indicated in inflammatory conditions.

The use of acupuncture or magnetic field therapy, ultrasound or shock wave therapy may help some horses^{4,12}. A study exploring the success of extracorporeal shock wave therapy included 12 horses presenting with various clinical signs such as headshaking and stiffness of the neck¹. These signs were attributable to changes of the protuberantia ossis occipitalis or the ligamentum nuchae as indicated by radiographic imaging. The shock wave treatment showed positive results as demonstrated by the fact that 84 % of the horses showed improvement after 3 treatments with a treatment interval of 4–18 days. The extracorporeal shock wave therapy is furthermore a non-invasive method, causes no additional trauma to the area treated and has favourable cosmetic results¹.

In conclusion, a specific aetiology in horses with headshaking is seldom confirmed and even if an abnormality is detected, its significance may be uncertain. In this report, avulsion fractures of the nuchal crest with associated nuchal desmitis was suspected to be the primary cause of the headshaking in both Thoroughbred racehorses and responded favourably to conservative therapy.

ACKNOWLEDGEMENTS

The authors would like to thank Drs Edward Evans and David Evezard for the referral of these interesting cases to the Onderstepoort Veterinary Animal Hospital, University of Pretoria, South Africa.

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