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Insertional desmopathies of the cranial attachment of the nuchal ligament in horses – A review

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Abstract: In recent years, neck pain and stiffness of the upper cervical region have become a commonly recognised cause of reduced performance in jumping and dressage in horses. One of the postulated causes of these clinical signs is the insertional desmopathy of the nuchal ligament. Its aetiology still remains unclear, chiefly due to the limitations associated with providing correct diagnoses in this region and the correct identification of the abnormalities. These lesions may be conductive to limb lameness, dental diseases and neurological changes. Considering that the resulting irreversible pathological changes present as pain and stiffness, patients are usually treated symptomatically. Insertional desmopathies of the nuchal ligament may underlie a lack of progress in training as well as stiffness and limb lameness in their broader sense. Further research is necessary to determine which lesions observed in the radiological examinations are of clinical significance.

Keywords: insertional desmopathies; nuchal ligament; equine; occipital area; neck; enthesophytes; radiology

Contents

1. Introduction

Insertional desmopathies are lesions on the ligament attachments. They cause subsequent structural degeneration and tissue remodelling. Exostoses are defined as benign growths of bone extending outwards from the bone surface. They can occur in any bone and may be triggered by a number of factors and are very often the final form of remodelling in the affected area (Dietz 2008) (Figures 1 and 2). Bone mineralisation, which is the process in which an organic bone matrix

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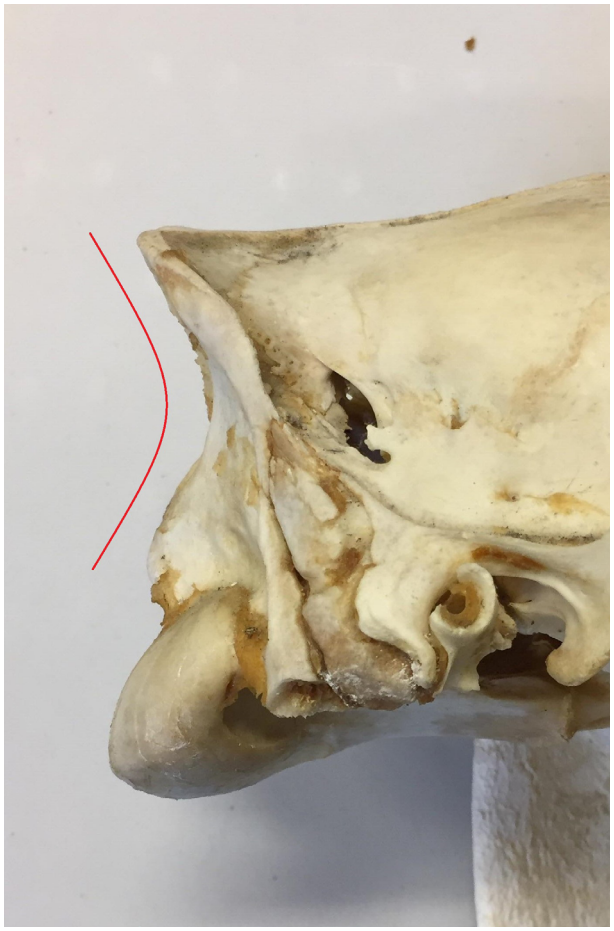

Figure 1. Physiological occipital bone- latero-medial view (sectional image of the skull, red line)

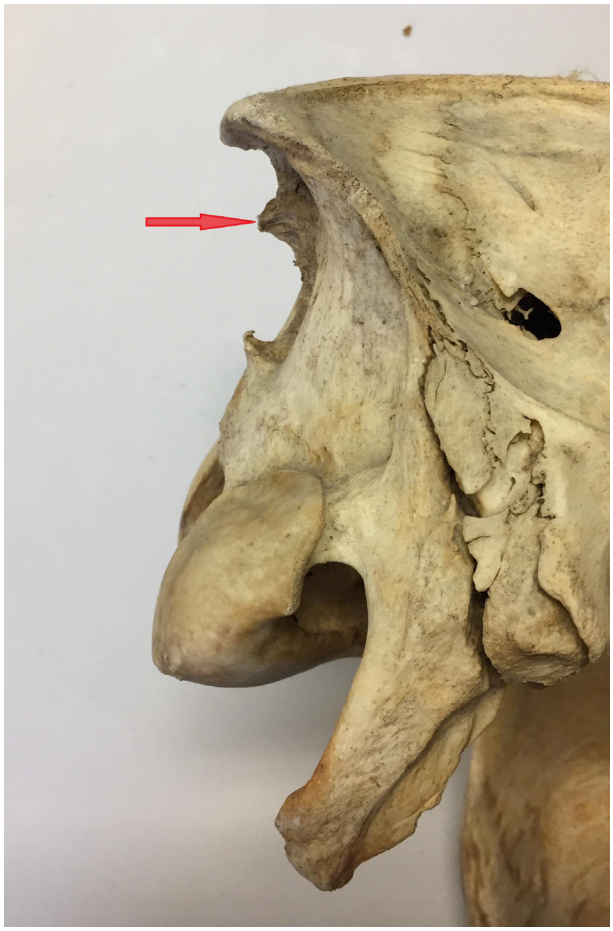

Figure 2. Exostoses on the occipital bone- latero-medial view (sectional image of the skull, red arrow)

becomes filled with calcium phosphate nanocrystals, occurs in a specific, highly ordered process. The process is mediated by osteoblasts and confined to the organic osteoid matrix produced by the osteoblasts (Schenkman et al. 2009; O'Brien et al. 2012; Magne and Bougault 2015).

Insertional desmopathies of the nuchal ligament have complex aetiologies. They may be caused by mechanical injuries, especially if the horse pulls back on the halter while tied (Dietz 2008; Dyson 2011) when it falls over an obstacle or somersaults. Jumping and dressage horses are predisposed to these pathologies due to the hyperflexion of the upper cervical region (Hinchcliff et al. 2013). Insertional desmopathies, which occur not only in the occipital region, for example, in the proximal and distal attachment of the suspensory ligament, are very common pathologies in sport horses (Dietz 2008; Baxter 2011). Although injuries of the proximal and distal attachments of the suspensory ligament are frequently observed in veterinary practices, there seems to be a need to characterise insertional desmopathies of the nuchal ligament and how significant these lesions are based on their radiological appearance. This review presents recent knowledge concerning insertional desmopathies of the nuchal ligament, taking their anatomical and physiological basics, new diagnostic methods as well as prophylactic and therapeutic strategies into account.

2. Search strategy

This literature review is based on a PubMed (https://www.ncbi.nlm.nih.gov/pubmed/) and Scopus (https://www.scopus.com/home.uri) search using the terms "horse, nuchal ligament, insertional desmopathy". Our own photos and X-ray archives were used to illustrate the characteristics of the insertional desmopathy of the nuchal ligament in horses.

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3. Anatomy of the nuchal ligament

The elastic nuchal ligament is one of the ligaments of the spine. It consists of two paired parts. Dorsally, there is a cord-like funicular part (*funiculus nuchae*) and ventrally to this lies a flat laminar part (*lamina nuchae*). The funicular extends from the external occipital protuberance to the summit to the withers, where it continues as the less elastic supraspinous ligament, which ends at the sacrum (Budras et al. 2005). The cranial segment of the funicular is an oval cord that passes dorsally to the atlas and axis without attaching to these bones. In the mid-neck region, the paired funicular gradually flattens and takes on a para-median position and it dorso-laterally ensheaths the thoracic spine so that its right and left parts almost make contact with the scapular cartilages. Caudal to the withers, the transition into the supraspinous ligament gradually narrows again to form a single median structure (Bejamin et al. 2002; Benjamin and McGonagle 2009).

The supraspinous bursa is localised to the cranial thoracic vertebrae (T3–T4). The cranial and caudal nuchal bursae have a similar function and lie dorsally to the atlas and axis. The fenestrated lamina nuchae attaches predominantly from the cervical vertebrae C2–C5 (Schmidt et al. 1982; Gellman and Bertram 2002; van Weeren 2004; Budras et al. 2005; Krysiak et al. 2008; May-Davis and Kleine 2014).

4. Histological features of the nuchal ligament

The nuchal ligament consists of a fibrous connective tissue that includes collagen and elastin. The characteristic feature of these fibres is their susceptibility to distention. Despite the high resistance to damage, with time, they are subject to degenerations or fragmentations, thus losing their structure (Dowling et al. 2000; Kuryszko and Zarzycki 2000; Sawicki and Malejczyk 2008). The elastic fibres branch out forming a net. They run singularly, assuming a wavy course, parallel to the long axis of the ligament. They weave bundles of type I collagen fibres. Therefore, the bundle of collagen fibres is straightened during the tension of the fibrous structure, whereas the elastic fibres are strained. Fibrocytes, glycosaminoglycans and a small amount of ground substance are found between the bundles of collagen and elastic fibres (Dowling et al. 2000; Kuryszko and Zarzycki 2000; Sawicki and Malejczyk 2008). Proteoglycans, and particularly decorin, modulate the formation and final dimensions of the fibrils in the extracellular matrix (Yoon and Halper 2005). Small proteoglycans, such as the aforementioned decorin, as well as fibromodulin, aggrecan and biglycan, regulate the function of tenocytes, the fibrillogenesis of collagen and the spatial organisation of fibres during normal development, as well as during the healing of tendons and ligaments following trauma (Yoon and Halper 2005; Kim et al. 2010). The properties of proteoglycans have a direct impact on the tendon strength (Hedbom and Heinegard 1993; Svensson et al. 1995; Gu and Wada 1996; Dowling et al. 2000). The physiological histological features of ligaments in horses include the presence of regular bundles of collagen fibres (Figure 3). Pathologically affected structures exhibit considerable disturbances of tissue integrity, which are associated with the occurrence of fibrous-cartilaginous metaplasia with chondrocyte clusters (Figure 4) (Schenkman et al. 2009; Ehrle et al. 2019). This metaplasia is defined as a phenotypic transformation of fibroblasts into the chondrocytes, with the concomitant change in the production of the cell matrix, which constitutes the organism's response to the chronic soft tissue irritation (distention, crushing, shearing) (Wren et al. 2000; Voigt et al. 2009). Under physiological circumstances, the area of attachment of the ligament to the bone

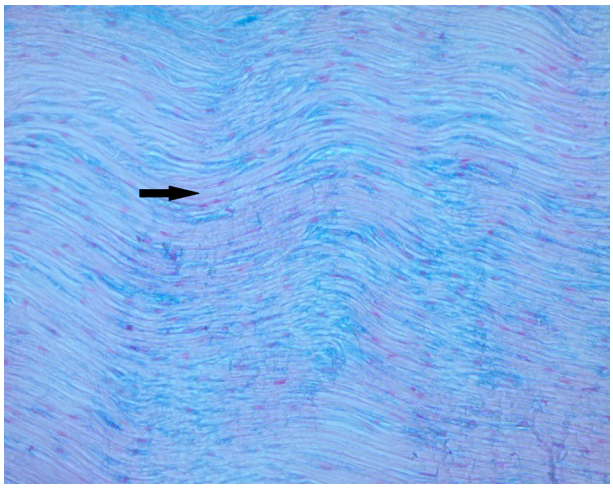

Figure 3. The physiological histological picture of the nuchal ligaments in horses is characterised by the presence of regular bundles of collagen fibres (black arrow), Alcian blue staining

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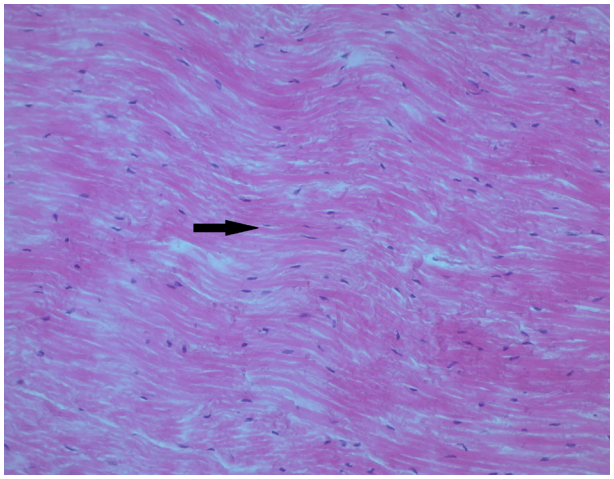

Figure 4. The physiological histological picture of the nuchal ligaments in horses is characterised by the presence of regular bundles of collagen fibres (black arrow), haematoxylin and eosin staining

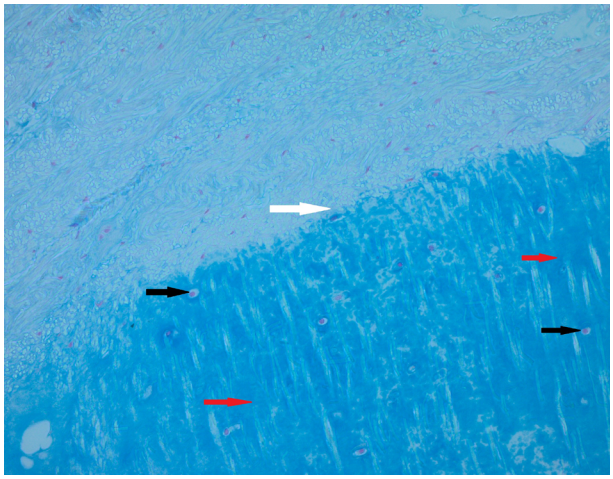

Figure 5. The structures covered by the pathology exhibit considerable disturbances of tissue integrity, which is associated with the occurrence of a fibrous-cartilaginous metaplasia and chondrocyte clusters (black arrow), diffusively distributed proteoglycans (red arrow), distinct boundary between the physiological and pathological tissue (white arrow), Alcian blue staining

may contain singular cartilaginous cells (Ehrle et al. 2019). A pathological change within the tissues of this area consists in the supersaturation of the tissue with proteoglycans and glycosaminoglycans, particularly between the collagen fibres, within the spaces between ligament bundles and around the blood vessels (Voigt et al. 2009; Ehrle et al. 2019). It should be noted that the changes described above are not accompanied by an inflammatory cell infiltrate (Halper et al. 2006).

Efficient connective tissue staining is performed using haematoxylin, eosin, Alcian blue, orcein, resorcin or fuchsin. In the case of a pathologically changed ligament tissue, it assumes a strong blue colour when stained with Alcian blue due to the presence of acid proteoglycans (positive staining, Figure 5).

In the case a physiological view of the ligament tissue (negative staining), the stained tissue becomes pink (Figure 3) (Ehrle et al. 2019) or light blue (Voigt et al. 2009). The above described difference in the connective tissue staining may be associated with the diffusively distributed proteoglycans, also providing blue colouration to the collagen fibres. In preparations stained with haematoxylin and eosin, proteoglycans assume a light pink colour (Figure 4).

In a pathologically changed ligament, mineralisation foci including calcification and endochondral ossification, can be observed (Halper et al. 2006; Halper et al. 2011; O'Brien et al. 2012). Mineralisation within the ligament may be caused by calcification or ossification. Calcification occurs with the deposition of calcium salts in the undamaged tissue (metastatic calcification) or in the damaged tissue (dystrophic calcification). In turn, ossification refers to the formation of osseous tissue within the collagen matrix (Chan et al. 2002).

These two processes are often indistinguishable radiographically, as pseudo trabeculation may be present alongside the calcification, mimicking the bone tissue (Strumia et al. 1997). Hence, the term mineralisation refers to both of these processes. A significantly lower content of adipose tissue is observed in the affected preparation compared to the physiological preparation.

Density of the small nerve fibres (sensory) are significantly bigger in the persistently irritated tissue than in the healthy/normal tissue (Ehrle et al. 2019).

5. Physiology and function of the nuchal ligament

Due to the flexibility of the nuchal ligament, it plays an important role in lifting the horse's head and neck. Muscles actively elevate the head and neck, while the nuchal ligament does this passively. Due to its elasticity, it passively relieves the dorsal extensors of the neck and participates in all spinal movements that take place in the median

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plane of the body (Budras et al. 2005; Krysiak et al. 2008; Baxter 2011; van Weeren 2016). In addition, the hypaxial cervical muscles, the brachiocephalic and omotransversarius muscles, rely on the traction provided by the funicular cord and lamellae as a counterbalance on the cervical vertebrae to bring the forelimb forward (Schmidt et al. 1982). This reduces the muscular work that would otherwise be required to hold the head above the ground and it is likely that there are dynamic links between the pendular mechanisms of the limbs and that of the head and neck segment (May-Davis and Kleine 2014; van Weeren 2016). It was further noted that by oscillating the cantilevered head-neck mass during locomotion it is likely to assist in the forward propulsion via the stretching and recoil of the nuchal ligament, particularly in the cervicothoracic region (Weaver and Barakzai 2010).

6. Etiopathophysiology

Microtraumas cause tears, inflammation and irritation, particularly in the cartilage matrix of the occipital bone (which covers the occipital bone) and the avulsion fragments. In turn, this causes the formation of scar tissue (after remodelling of the damaged tissue) which then undergoes calcification (Dietz 2008). Also, lesions caused by the invasion of Onchocerca species have been reported (Schmidt et al. 1982). Training methods such as neck hyperflexion, rollkur or the excessive use of side reins that are too short may lead to the pathology in this area (Baxter 2011).

7. Clinical signs

The clinical symptoms of insertional desmopathy are not homogeneous. They are usually manifest at work while riding, especially during bit-hand contact. Furthermore, during circular movements, the horse's head is directed outside, while its neck remains straight. When the rider maintains bit contact, the horse affected by a desmopathy is stiff, reluctant to ride in collection or in the right position and incites asymmetric rein tension. Some positions of the horse's head may cause pain and result in the animal's reluctance to carry out certain exercises and progress in training (Benjamin et al. 2002; Dietz 2008; Benjamin and McGonagle 2009; Baxter 2011; Dyson 2011; Lavoie and Hinchcliff 2011). Due to the fact that the nuchal ligament runs in the wither region and merges into the supraspinatus ligament running along the thoracic and lumbar spine regions, pain may occur in the back region as well as during neck-bending movements, e.g., to eat grass or hay (Dietz 2008).

8. Diagnosis of insertional desmopathies of the nuchal ligament

Differential diagnoses of insertional desmopathies of the nuchal ligament should take pain within the cervical region and other diseases into account, which are presented in Table 1. Septic/aseptic bursitis manifests as an oedema or areas of enlargement above the atlas or axis, which may be unilaterally or bilaterally divided by the nuchal ligament (Brems and Weiss 2002; Dietz 2008). The difficulty in a differential diagnosis with insertional desmopathies is caused by the close location of the cranial attachment of the nuchal ligament and nuchal bursae. During palpation, the whole cranial area of the neck can be painful. An ultrasonographic examination is the most sensitive diagnostic procedure in this case.

A cervical vertebral fracture and/or dislocation most commonly affects foals and young horses. The symptoms associated with such a trauma largely depend on the location and type of the frac-

Table 1. Differential diagnosis of the insertional desmopathies of the nuchal ligament in horses

Diseases of the cervical region	Other diseases
Septic/aseptic synovial bursitis	Dental diseases
Cervical vertebra fracture and dislocation	Temporomandibular joint arthritis
Nuchal ligament inflammation	Idiopathic headshaking
Cervical arthritis	Back pathologies
	Thoracic and pelvis limb lameness
	Behavioural disorders

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ture. For example, in the case of the vertebral body or an arch fracture, paralysing symptoms causing spinal cord compression or rupture may occur (Brems and Weiss 2002; Dyson 2011).

Nuchal ligament inflammation manifests as an oedema, soreness and increased heat in this area (Brems and Weiss 2002).

Cervical arthritis is associated with an increased amount of fluid in the joint and some bone lesions related with the neck stiffens (Brems and Weiss 2002; Baxter 2011).

Dental diseases, such as wolf teeth, sharp edges, broken tooth roots, funnel caries and temporomandibular joint arthritis can cause intolerance to the bit, pushing on one rein, stiffness or even lameness (Brems and Weiss 2002; Dyson 2011; Lavoie and Hinchcliff 2011).

Idiopathic head shaking may be of neurological origin, but may also be associated with many musculoskeletal pathologies (Brems and Weiss 2002).

Back pathologies connected with muscle pain and the kissing spine syndrome may also resemble symptoms associated with insertional desmopathies of the nuchal ligament (Brems and Weiss 2002; Baxter 2011).

The difficult to diagnose thoracic and pelvic lameness may be one of the reasons of the difficulty to differentiate pathologies causing poor performance. Many different traumas of the upper and lower part of the legs cause pain leading to stiffness.

Behavioural problems may be caused by pain and stiffness, which also reduce training achievements (Brems and Weiss 2002).

All of the abovementioned pathologies may resemble the main clinical symptoms of insertional desmopathies of the nuchal ligament.

The clinical examination of the attachment of the nuchal ligament includes its palpation, with special attention paid to pain reactions on palpation, its asymmetry and thickening due to a fibrosis. In order to assess the elasticity, provocative tests with a treat are carried out to make the horse bend its head maximally towards its shoulder blades and between its thoracic limbs (Baxter 2011). It is also necessary to observe the horse moving on the lunge and under the rider. The horse should work "on the bit", meaning that it should calmly and softly seek contact with its rider's hands through the bit. This affects the carriage of its whole body. The necks of correctly ridden horses show evenly developed muscle tone that appears to flow smoothly from the withers to the poll and the underneck is soft and concave, the left and right musculature side should be symmetrical.

Pathological signs occur when the horse is ridden "above the bit", whereby the head is raised and the neck stiffened, resulting in a dropped back, increased weight on the forehand and shortened steps in the hind legs. If the horse is ridden "behind the bit", where the vertical plane of the neck vertebrae is compressed, the strides become uneven, causing the horse to take shorter steps with the hind legs and larger ones with his front legs. Any other behavioural changes, such as the tilting of the head or a tongue prolapse are clinically significant and interpreted according to the personal experience. The differential diagnostics should exclude any possible lameness (Dietz 2008; Baxter 2011; Dyson 2011).

Diagnostic anaesthesia to identify the source of pain of the attachment of the nuchal ligament to the occiput, performed blindly or under ultrasound guidance is usually carried out with 2% mepivacaine (5–20 ml) (Dietz 2008; Baxter 2011; Lavoie and Hinchcliff 2011). The efficacy of the infiltration block should be checked after 20 minutes.

Sometimes there are false negative results due to a larger number of pathological structures and due to the chronic nature of the lesions.

In everyday practice, X-ray images of the occipital region in the lateral projection are used, where the occipital surface resembles a sort of smooth ski jump (Weaver and Barakzai 2010; Dyson 2011). However, finding enthesophytes on the occipital bone or in the ligament structure does not validate the source of the problem (Dietz 2008) (Figures 2, 6, 8). The ultrasound examination is an additional tool that enables the diagnosis of the lesions in the structure, the size and shape of the ligament fibres, especially in the area of mineralisation with shadowing artefacts.

Computed tomography and magnetic resonance imaging are the most precise diagnostic methods imaging the structure of both hard and soft tissues, allowing the visualisation of the changes within the bone structure and areas difficult to palpate and image. However, the use of these tests is limited due to their high cost and the need for general anaesthesia (Landman et al. 2004; Brunsting et al. 2017).

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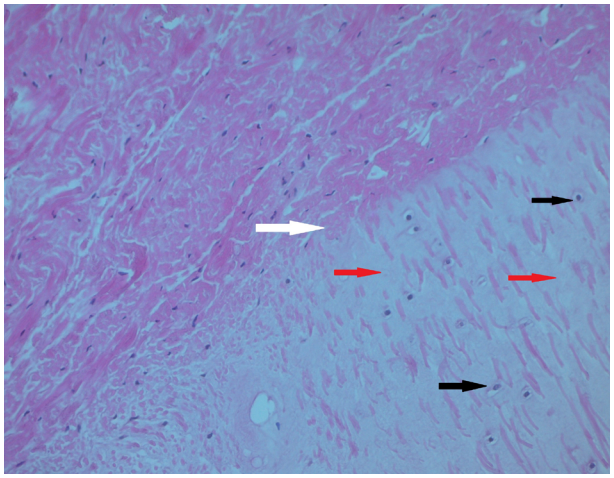

Figure 6. The histopathological picture of the nuchal ligaments in horses with the occurrence of a fibrous-cartilaginous metaplasia and chondrocyte clusters (black arrow), diffusively distributed proteoglycans (red arrow), a distinct boundary between the physiological and pathological tissue (white arrow) haematoxylin and eosin straining

9. Therapy

The therapy for insertional desmopathies of the cranial attachment of the nuchal ligament can be treated conservatively and invasively. Most importantly, treatment consists of rest for at least 8 weeks. The horse returns to training gradually (intensity, duration, frequency, type – straight lines, encouraged to flex the poll region gently from side to side and up and down), but work "on the bit" should begin only after the inflamed tissue has loosened completely (Dietz 2008; Dyson 2011; Brunsting et al. 2017).

Treatment consists of multiple injections, around 3–4 times, of steroidal anti-inflammatory drugs such as methylprednisolone (40 mg) or triamcinolone (6–9 mg) diluted in mepivacaine/lignocaine/ropivacaine (10 ml) or a *Sarracenia purpurea* (10 ml) suspension within 1–6 weeks. During this procedure, penetration of the affected areas should be avoided by circular infiltration. This therapy can be complemented by the intravenous administration of hyaluronic acid (60 mg, 3–7 times, interval between doses 3–9 days). A local treatment may be supplemented with a general treatment in the form of a three-week oral therapy with phenylbutazone (2.2–2.4 mg/kg every 12 h p.o.).

The use as local injections of stem cells and platelet-rich plasma is still at an experimental phase.

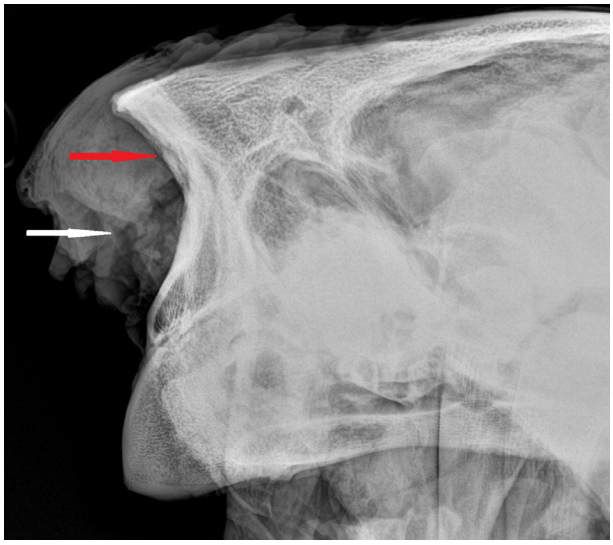

Figure 7. Physiological occipital bone- latero-medial view X-ray image (red arrow – occipital bone, white arrow v nuchal ligament)

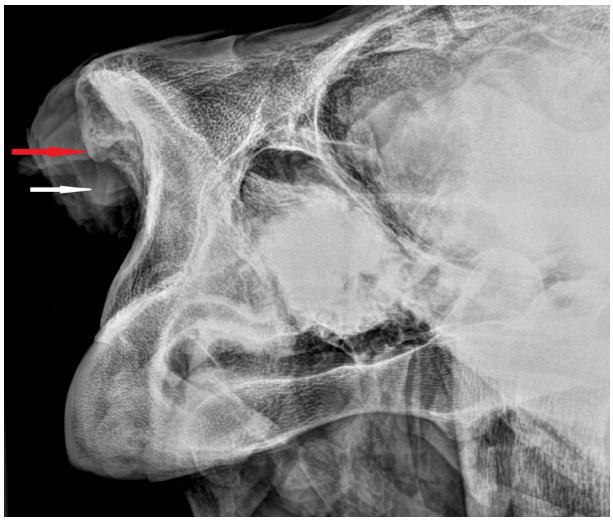

Figure 8. Insertional desmopathy of the nuchal ligament latero-medial view X-ray image (red arrow – occipital bone changes, white arrow – nuchal ligament)

Shockwave therapy is applied more and more effectively. Treatments should be repeated thrice, at 4–8 day intervals (Dietz 2008; Brunsting et al. 2017). Prior to the treatment, the hair in the occipital area should be clipped and the animal should be sedated, e.g., with detomidine (10–20 mg/kg i.v.) and butorphanol (0.01–0.02 mg/kg i.v.). The treatment should include 1 200 strokes at a level 6 intensity (0.5 nJ/mm^2) (Brusting et al. 2017). Follow-up clinical examinations (reducing tension and pain) and ultrasound examinations by a linear transducer with a frequency bandwidth of 7.5–8.5 megahertz

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(MHz) and gain at 180 (changes in the structure, shape and size on the ligament) should take place 4, 10 and 20 weeks following the completion of the treatment.

Follow-up examinations (clinical – reducing tension and pain and ultrasound examinations – changes in the structure, shape and size on the ligament) should take place 4, 10 and 20 weeks following the completion of the treatment. The patient's condition is expected to improve within 10 weeks (Dietz 2008; Baxter 2011; Dyson 2011).

If both types of lesions, i.e., free mineralisation in the ligament body and exostosis (bone spurs), occur simultaneously, the prognosis is worse than in the case of isolated lesions. In the case of fractures of the attachment of the occipital bone, surgical removal of the detached fragments is recommended.

If the treatment is ineffective (pain and stiffness remain), it is possible to conduct a desmotomy of the nuchal ligament at a distance of about 7 cm caudally from the attachment to the occipital bone. In some cases, the procedure is carried out with a desmotomy of the *m. semispinalis capitis* tendon (Baxter 2011).

If the recovery time is long enough, the prognosis is usually good and the horse can fully return to the previous form of use (Waever and Barakzai 2010; Dyson 2011). Due to insufficient research on this subject, it is still unclear which treatment method is the most effective.

10. Conclusions

The equine neck contains complex structures that occupy a central position in the entire musculoskeletal system; hence they can be decisive for performance. Therefore, it is essential to understand the neck mechanics and physiology. The close interrelationship between the limbs and the back and neck is often underestimated. In the current study, the occurrence of lameness in horses with a diagnosed back and neck problem was much higher than in animals solely with limb lameness (Brunsting et al. 2017).

A lack of progress in training, lameness and stiffness may be caused by insertional desmopathies of the nuchal ligament. Subsequent research is essential to determine which radiological lesions have a clinical significance.

Conflict of interest

The authors declare no conflict of interest.

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