Collateral ligament desmopathy of equine distal interphalangeal joint

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SHELLEY DOWN examines the clinical signs and diagnostic imaging methods available before suggesting treatment modalities for this cause of lameness

Summary

Desmopathy of the collateral ligaments of the distal interphalangeal joint is an important cause of foot lameness and poor performance in the horse. Analgesic techniques, imaging, treatment and prognosis are outlined in this article.

Key words

equine, desmopathy, collateral ligaments, distal interphalangeal joint, analgesia

THE collateral ligaments (CLs) of the distal interphalangeal joint (DIPJ) are short, strong bands that originate on the dorsomedial and dorsolateral distal aspects of the middle phalanx and insert on the dorsomedial and dorsolateral proximal aspects of the distal phalanx.

The CLs closely oppose the DIPJ capsule and cartilages of the foot[1], and function to support the DIPJ[2]. The DIPJ is greatly influenced by hoof placement[2]. Asymmetric foot placement with the quarters at differing heights induces a combination of rotating and sliding of the distal phalanx relative to the middle. The main structure that limits this motion is the CL on the opposite side to

the elevated quarter[2].

Collateral ligament desmopathy is an important cause of foot lameness[3]-[6]. In this article, abnormality of the CL is described as desmopathy, referring to any ligament pathology.

Clinical examination

History of CL desmopathy usually includes lameness or poor performance (shortened stride length, unwilling to work on the bridle or refusing certain fences – such as combinations or drops). Distal interphalangeal joint effusion is common[3],[5], although a non-specific finding[7], and may be due to a capsulitis/ synovitis secondary to CL injury[8]. Distal interphalangeal joint synovial disruption was reported with distal CL lesions in a postmortem study of 25 horses using MRI[9].

Swelling just proximal to the coronary band has been seen with severe CL injuries[10]. Localising signs are otherwise uncommon[3],[8],[11] and presence will depend on lesion severity, proximodistal site and chronicity of injury.

Shortened caudal phase of stride has been noted in severely lame horses[9], possibly reflecting avoidance of DIPJ extension when the distal CLs are under the greatest tension[12],[13]. Most cases are lamest on a circle on hard ground[3],[8],[11],[14]. Greatest strain is found in the medial ligaments when turning[15], which may explain greater incidence of medial injuries[3],[4],[9],[10],[14],[16]. Greater osseous abnormalities associated with the origins/ insertions of the CLs have also been noted medially[17].

Poor performance cases are usually worst when ridden. A significant association between extensively ossified cartilages of the foot and CL injury has been found[18]. Energy dissipation may be reduced through the ossified cartilages, resulting in greater strain placed on the CLs.

Analgesia

Perineural analgesia of the palmar digital (PD) and subsequently palmar nerves at the level of the proximal sesamoid bones is usually required to abolish lameness.

Variable responses to analgesia are cited in the literature[3],[11]. Improvement following PD analgesia may reflect DIPJ pain. Another explanation for variable analgesia pattern may be proximodistal extent of lesion, and proximal spread of local anaesthetic solution. Concurrent injury can be desensitised by the same analgesic technique as the CLs. In these cases, MRI is particularly useful in identifying the source(s) of pathology causing lameness/ poor performance.

The percentage of horses with CL desmopathy that respond to DIPJ intra-articular analgesia varies from 40 per cent[3] to 90 per cent[8]. This may reflect presence or absence of concurrent synovitis and/or osteoarthritis, volume of local anaesthetic solution used and time from analgesia to reassessment of lameness. Desensitisation of the PD nerves following DIPJ analgesia is possible

because of close proximity. Disruption of the synovial membrane axial to abnormal CLs has been described[9]; the proximodistal extent and severity of lesion may, therefore, have implications on response to DIPJ analgesia.

Diagnostic imaging

Ultrasonography of the CLs is well described[8],[16],[19]. The ligaments should be viewed in transverse and longitudinal planes. Denoix (2011)[20] states measurements in a 550kg horse to be approximately 6mm to 9mm thick, 12mm to 16mm wide and with a cross-sectional area of 0.6cm to 0.9cm^2. Contralateral CLs within and/or across the limb(s) should be measured as comparison, although lesions may be found in multiple ligaments. Ultrasonographic examination of proximal injuries has been described to assess response to exercise during rehabilitation[8].

Enthesopathy at the origin and/or insertion of the CLs may be seen radiographically[5]. Other osseous abnormalities are endosteal reaction, osseous cyst-like lesions and fragmentation[17], which are most readily visualised using MRI. The incidence of osseous abnormality associated with the CLs is greater at the insertions than the origins[17]. Distal interphalangeal joint periarticular osteophytes are sometimes seen radiographically and may be secondary to DIPJ instability.

Nuclear scintigraphy has high specificity, but low sensitivity for CL injury[11],[21]. Vascular (dorsal and lateral) and bone phase (dorsal, lateral and solar) images[22] are usually obtained. Increased radiopharmaceutical uptake (IRU) at the insertion of the CLs is best seen in solar views[3] and associated with more severe histological lesions than those without IRU[9]. IRU has not correlated with the proximodistal extent of lesion[21]. Histologically, worse lesions were more prevalent distally (44/67 limbs)[9], supporting previous suggestions the insertions are a major stress point[21]. Histopathology was worse palmarly in distal lesions, which may explain IRU commonly seen palmar to the insertion points[3],[11],[21],[23].

MRI enables proximodistal and dorsopalmar extent of injury and concurrent lesions to be identified. A low-field system produces a lower signalto-noise ratio than high-field systems, resulting in reduced image resolution[23],[24],[25]. Greater image resolution combined with thinner slices enables more subtle lesions to be identified using high-field systems, which is important in cases of poor performance.

Injury is best identified in transverse and dorsal MR planes[3]. In more commonly used low-field systems, the CLs have a heterogenous signal pattern with increased signal intensity at the periphery within the proximal to mid-portion of the ligament (which should not be confused with desmopathy). This area of increased signal intensity is due to curvature of the peripheral fibres resulting in a magic angle effect[26]. The magic angle effect is an artefactual increase in signal intensity[27],[28],[29] in tendons or ligaments orientated at or approaching 55° to the static magnetic field.

In a low-field system the lateral CL is particularly susceptible[27]. It is therefore important that signal changes are visible in several planes to ensure these changes are within the same structure and not artefactual[14], and sequences, such as fast spin echo (FSE), are used, which are significantly less susceptible because of the longer echo time than other pulse sequences[27],[30].

Enlargement of the CLs is most easily seen on T1-weighted MR images because they show anatomy well[31]. The CLs are generally symmetrical across a limb, but differing cross-sectional areas of CLs within a limb can be due to image slice obliquity[32]. It is critical the limb is positioned correctly in low-field systems, which cannot post-process 3D image data unlike high-field systems[3]. Change of size, shape, border clarity, signal intensity, presence of periligamentous tissue disruption and entheseous abnormalities may indicate pathology.

Treatment

Although there is no scientific evidence to support specific shoeing for CL injury, treatment tends to include remedial farriery to aid recovery. Farriery should be undertaken on an individual basis based upon the distal limb conformation, foot balance and shape, limb flight and foot placement.

Techniques to shorten the toe are often used to reduce the need for DIPJ flexion and ease break-over. A widewebbed shoe with increased width to the side of CL injury has been used to decrease tension in acutely injured CLs[33]. Shoeing for concurrent injury may also be required.

Extracorporeal shock wave therapy (ECSWT) or radial pressure wave therapy (RPWT) can be used on the proximal aspect of the CLs, the benefit of which is unsubstantiated. Dakin (2009)[34] has found there to be no effect of use of ECSWT or RPWT on outcome. However, there were a low number of horses within this group, giving insufficient power to the study. A controlled exercise programme is always advised. Successful treatment with cast immobilisation has also been documented[35].

Prognosis

Prognosis for return to full athletic function is poor to moderate (29 per cent[3], 33 per cent[3], 60 per cent[14] and 27 per cent[34]). Mild to moderate associated osseous lesions, such as enthesiopathy, do not appear to worsen prognosis34.

Rehabilitation times vary (18 weeks[8] ; six months walking[11]). A study in 2009 found a better prognosis than previous studies[14]. These horses had MR findings indicating probable acute injury, which may have resulted in better outcome.

Outcome

CL injury may be a primary degenerative process[9], which may explain the poor prognosis in many studies. Poor conformation and foot balance may impede recovery, regardless of underlying cause.

Conclusion

There are varying presentations of CL injury. Radiography, ultrasonography and scintigraphy may aid diagnosis although MRI is the gold standard.

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