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Patellar ligament desmopathy in the horse – a review and comparison to human patellar tendinopathy ('Jumper's knee')

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Abstract

Patellar ligament desmopathy in horses is regarded as an uncommon condition with unclear aetiology. Of the three patellar ligaments in the horse, the intermediate is the one most often diagnosed with desmopathy in horses presenting with chronic lameness. This structure corresponds to the patellar tendon in humans. As diagnostic imaging modalities continuously improve, changes in echogenicity of the patellar ligaments are identified ultrasonographically with increasing frequency. However, disruption of the normal fibre pattern may be present also in patellar ligaments in horses that show no signs of lameness. Similarly, there is a poor correlation between pain and diagnostic imaging findings in human patellar tendinopathy. Consequently, there appears to be a knowledge gap pertaining to normal ultrasonographic variation and diagnostic criteria for disease of the patellar ligaments in horses. Furthermore, local anaesthetic techniques to verify the diagnosis are poorly described, and due to the low number of treated cases, no specific treatment modality can be recommended on a scientific basis. The aim of this paper is to review the current knowledge regarding the pathogenesis, diagnosis and management of patellar ligament desmopathy in horses, compare this condition with patellar tendinopathy in humans, and identify areas for further research to increase the diagnostic accuracy in horses. We conclude that there is a profound need for better descriptions of ultrasonographic variation and pathological changes of the equine patellar ligaments. Identification of areas of maximal ligament strain and descriptions of early histopathological changes could render more information on the possible aetiology, preventive measurements and treatment options of desmopathy. Description of regional innervation could aid in development of methods for diagnostic anaesthesia to verify pain originating from the ligaments.

Keywords: stifle, lameness, diagnostic anaesthesia, ultrasound

1. Introduction

Lameness originating from the stifle joint is estimated to comprise 8-42% of horses presenting with hind limb lameness. The anatomical location of stifle lesions varies depending on the athletic discipline and use of the horse, level of work, and age (Dyson, 2002a; Egenvall *et al.*, 2013; Murray *et al.* 2006; Singer *et al.*, 2008; Vaughan, 1965).

Historically and to the current day, the patellar ligaments have received much attention as potential causes of orthopaedic problems in the horse. The medial patellar ligament plays a crucial role when unintentional engagement of the passive stay apparatus causes a 'locking' of the stifle, leading to intermittent or permanent upward fixation of the patella, a condition that is managed conservatively or surgically depending on its severity. Other less characteristic gait abnormalities have been ascribed to milder forms of patella fixation or patellar ligament 'laxity' (Kallings, 2021; McIlwraith, 1987). In contemporary sport horse orthopaedics, despite the absence of a scientific basis for conditions such as patellar ligament 'laxity' or 'stifle instability', treatment with counterirritants or blistering agents is still practiced, despite being banned by several equestrian federations (Kallings, 2021; McIlwraith, 1987). This raises ethical concerns, since the clinical effect of

such treatments have not been validated, and it may cause pain and complications necessitating surgical intervention (Cambridge *et al.*, 1985).

Although ill-defined entities such as ligament laxity have been believed to cause lameness, true ligament pathology such as patellar ligament desmopathy has been regarded as an uncommon condition in horses. However, since the first published case series describing ultrasonographic characteristics of normal and abnormal patellar ligaments, more recent reports indicate that changes in echogenicity and fibre disruptions of the patellar ligaments are identified with increasing frequency, reported in 4-18% of horses undergoing ultrasonographic examination of the stifle (Dyson 2002b; Gottlieb *et al.*, 2016; Hoaglund *et al.*, 2019; Van der Vekens *et al.*, 2016). This apparent increase is possibly reflecting improvements in diagnostic ultrasound techniques and equipment. In addition, the increasing availability of portable ultrasonographic equipment to the equine practitioner may result in more clinicians identifying changes in the patellar ligaments and in some instances interpreting these findings as patellar ligament desmopathy.

The clinical significance of ultrasonographic findings of the patellar ligaments has, however, been challenged, as such findings have been observed in patellar ligaments of horses in full work without clinical signs of lameness (Van der Vekens *et al.*, 2016). In humans, a clinical syndrome characterised by pain from and ultrasonographic lesions in the proximal posterior patellar tendon is known as 'Jumper´s Knee' or 'patellar tendinopathy' (Santana *et al.*, 2022). Ultrasonographic findings have been recognised in asymptomatic patellar tendons also in humans, and observations indicating that these resolve without progression to clinical disease raise questions about their significance (Cook *et al.*, 2001). To date, no information exists on whether patellar ligament injuries in horses correspond to the clinical entity of human patellar tendinopathy.

The aim of this review is to describe current knowledge regarding the pathogenesis, diagnosis and management of clinical problems related to the patellar ligaments in the horse; to describe comparative aspects to human patellar tendinopathy; and to identify areas for further research in order to improve the diagnostic accuracy and provide more knowledge about appropriate treatment of patellar desmopathy in the horse.

2. Comparative anatomy of the patellar apparatus

Although some obvious anatomical differences exist between the equine stifle and the human knee (Figure 1), similarities include the bicondylar distal femur and proximal tibia; the cruciate ligaments; the meniscal structure, and asymmetrical collateral ligaments (Singh, 2016). Also, in both species, the patella acts as a sesamoid bone within the quadriceps extensor mechanism and articulates with the femoral trochleas and trochlear groove; in the horse, this portion of the joint is confined within a separate synovial cavity known as the femoropatellar joint. While all four muscle bellies of the quadriceps musculature insert onto the patella in man, the *vastus medialis* muscle inserts onto the parapatellar fibrocartilage in the horse, which is a medial extension of the patella from which the medial patellar ligament originates (Singh, 2016). At its caudodistal margin, this ligament merges with the common aponeurosis of the *gracilis* and *sartorius* muscles, until its insertion at the craniomedial aspect of the tibial tuberosity (Singh, 2016).

In humans and smaller animals, the extensor apparatus of the knee is joined into a singular patellar tendon inserting on the tibial tuberosity. The intermediate patellar ligament of the horse, originating at the patellar apex and inserting in the *sulcus tuberositas tibiae*, is the corresponding structure (Singh, 2016). In the human medical reference work Dorland´s Illustrated Medical

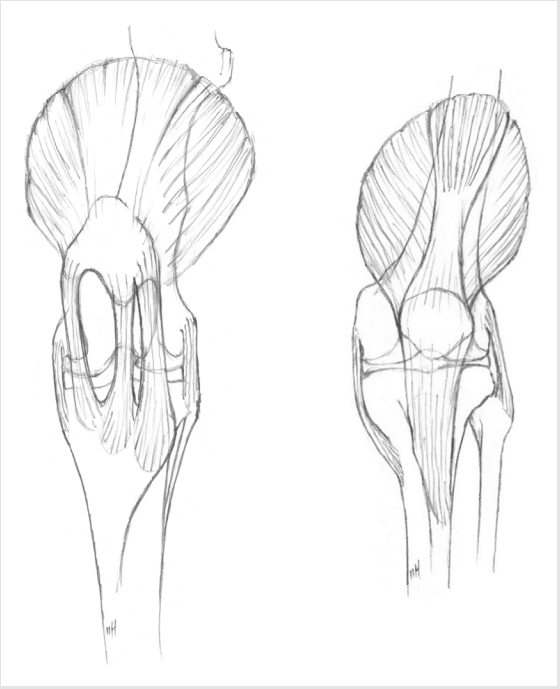

Figure 1. Cranial aspect of horse stifle (left) and anterior aspect of human knee (right). Medial is to the left. In the horse, three patellar ligaments (medial, intermediate and lateral) insert on the tuberositas tibiae. The medial patellar ligament is lifted, rotated and locked over the protruberance of the medial trochlea to engage the passive stay apparatus of the hind limb. The intermediate patellar ligament corresponds to the singular tendon of insertion of the quadriceps musculature, the patellar tendon, of the human knee. Illustration: Matthias Haab.

Dictionary, the *ligamentum patellae,* or patellar ligament, is defined as 'the continuation of the central portion of the tendon of the quadriceps femoris muscle distal to the patella; it extends from the patella to the tuberosity of the tibia' (Dorland, 1988). According to this dictionary and the *Terminologia Anatomica* (FIPAT, 2019), *ligamentum patellae* is synonymous with *tendo patellae*, patellar tendon, and infrapatellar portion of the *quadriceps* tendon (Dorland, 1988; FIPAT, 2019). As in the human medical literature, the terms 'patellar tendon' and 'patellar ligament' are used interchangeably in various species (Samuels *et al.*, 2017; Singh, 2016); however, the *Nomina anatomica veterinaria* terminology only lists the *lig. patellae*, and the specific terms *lig. patellae intermedium, mediale, laterale* 'should be used only in the horse and ox' (ICVGAN, 2017).

The equine lateral patellar ligament originates from the lateral aspect of the cranial patellar surface and is fused along its lateral aspect to the tendon from the *m. biceps femoris* and an aponeurosis from the *fascia lata*. The lateral patellar ligament inserts on the craniolateral aspect of the tibial tuberosity, where all three patellar ligaments converge and are bridged by a retinaculum (Singh, 2016; Sisson, 1975; Wissdorf, 2010).

In both horses and humans, the patellar ligaments are separated from the synovial cavity by the infrapatellar fat pad, which is covered by synovial membrane along its caudal/posterior aspect (Gallagher *et al.*, 2005).

In horses, the vascular supply to the patellar ligaments and infrapatellar fat pad derives mainly from the *saphenous* artery and descending *genicular* artery, both branching from the *femoral* artery at the level of the proximal thigh (Updike and Diesem, 1980). Branches of the *femoral* nerve innervate the lateral and intermediate patellar ligaments as well as lateral portions of the infrapatellar fat pad; the medial patellar ligament and the medial portion of the fat pad receives innervation from a caudal nerve branch arising from anastomoses of the *obturator* and *saphenous* nerve (Rankin, 1975).

3. The passive stay apparatus

The medial structures of the equine patellar apparatus, i.e. the parapatellar fibrocartilage with its muscular attachment and the medial patellar ligament, are key components of the passive stay apparatus of the equine hind limb allowing the animal to 'lock' the stifle in extension with minimal muscular activity (Schuurman *et al.*, 2003). Due to a marked asymmetry of the femoral trochleas, where the medial trochlea is elongated to form a proximal protuberance, the animal is able to hook the parapatellar fibrocartilage and medial patellar ligament over this bony prominence (Singh, 2016). When engaging this mechanism, the patella is lifted and rotated approximately 15° by action of the *quadriceps* musculature with the support of the *sartorius*, *gracilis* and *semimembranosus* muscles. Tonic activity of the *vastus medialis* muscle keeps the patella 'locked' (Schuurman *et al.,* 2003), whereas unhooking the patella occurs with action of the *quadriceps* and *biceps femoris* musculature (Singh, 2016; Wissdorf, 2010).

Inability or difficulty to release the patella from the femoral trochlea leads to upward fixation of the patella (UFP). Proposed causes include decreased tonus of *quadriceps* musculature, straight hindlimb conformation, and neuromuscular disorders (Fowlie *et al.*, 2019; Singh, 2016; Wissdorf *et al.*, 2010). A disturbance in the *vastus medialis* muscle control mechanism, is also a proposed aetiology; however, this has not been investigated further (Schuurman *et al.,* 2003). Severity of the condition varies. In permanent fixation that cannot be manually corrected, the stifle and tarsus (due to the reciprocal apparatus) are locked, and the limb fixed in extended caudo-lateral position with the phalanges flexed and only the toe touching the ground. In horses with intermittent fixation, the patella releases with or without assistance. In the milder form with delayed patellar release, the patella catches temporarily over the medial trochlea, resulting in a jerky movement of the patella when the limb is protracted, or when the horse decelerates from a faster to a slower gait (Walmsley, 2011).

Milder forms of UFP are treated conservatively with corrective trimming and/or shoeing as well as instituting a conditioning and nutrition program to promote muscle strength and development (Dumoulin *et al.,* 2007). Anecdotal reports exist of success following injection of oil-based counterirritant solution containing iodine and ethanolamine oleate into the medial and intermediate patellar ligaments; however, studies investigating effects of these solutions have only included healthy horses and mainly focused on the short-term histologic and ultrasonographic changes in the ligaments following injections. Injection of 1.9% iodine and 9% ether in almond oil, isoethanolamine oleate, and 2% iodine in almond oil in the medial and middle patellar ligaments of healthy horses induced inflammation, fibroplasia, fibrosis and increased ligament diameter (Brown *et al.*, 1984; Van Hoogmoed *et al.*, 2002). Subcutaneous infiltration or topical application of counterirritants and blistering agents have also been used, mainly in horses with proposed patellar ligament laxity, a diagnosis unsubstantiated in the scientific literature (Kallings, 2021). These practices are now prohibited by the International Federation of Horseracing Authorities (IFHA) as well as the European Trotting Union, UET, and blistering over the patellar ligaments is now also being banned by the Swedish National Equestrian Federation (Kallings, 2021).

Surgical treatment for UFP is reserved for severe cases and cases refractory to conservative management (Dumoulin *et al.,* 2007). Two surgical techniques are available. Whereas

the less invasive medial patellar ligament splitting technique carries a lower risk of post operative complications and a faster return to work compared to traditional medial patellar ligament desmotomy (Andersen and Tnibar, 2014), the efficacy of this technique has been questioned as recurrence of UFP occurred in a third of operated cases (Peitzmeier *et al.*, 2015). Conversely, medial patellar desmotomy is effective in alleviating the original problem, but continued lameness due to complications such as patellar chondromalacia, patellar fragmentation and enthesiophyte formation may occur (Gibson *et al.*, 1989; Dumoulin *et al.*, 2007). However, the risk of post-operative complications is less when horses are box-rested a minimum of 3 months post operatively (Dumoulin *et al.*, 2007).

4. Comparative biomechanics of the patellar apparatus

In the standing horse, the femorotibial joint angle is approximately 150 degrees (Madry *et al.*, 2015). With hyperextension, an angle of 155 to 161 degrees can be achieved (Halley *et al.*, 2014; Sisson, 1975). In a translational context, the gait of horses, sheep and goats most closely resembles the human; however, no large animal model animal can achieve the 180-degree angle of the human knee (Madry *et al.*, 2015).

Biomechanical studies underline the functional importance of the equine stifle in weight bearing and power production of the hind limb. The stifle contributes to the upward bounce of the horse after pushing-off from the ground. Muscle spindles in the *vastus* musculature have a role in the myotatic reflex maintaining posture against the force of gravity. Flexion of the femorotibial joint by the weight of the body results in stretching of the *vastus* muscle, thus exciting annulospiral receptors within the muscle, which, reflexively, excite the skeletomotor neurons that control the ordinary extrafusal muscle fibres of the *vastus*. The muscular action results in extension of the femorotibial joint to its original position (Hoyt *et al.*, 2002; Skerritt, 2018). Through most of the stance phase, flexors and extensors around the stifle are activated simultaneously, creating stability rather than production of power (Dutto *et al.*, 2006). Contrary, while jumping, the stifle is the primary power producer (Dutto *et al.*, 2004).

Equine femorotibial and meniscal biomechanics have been investigated *ex vivo*. Tibial contact force distribution, bone density, meniscal translocation and deformation, ligament strain, and stifle articular surface contact have been investigated in cadaver limbs; however, equine cadaver studies are limited by the absence of muscle activity, removal of relevant stabilising structures, and difficulty applying *in vivo* forces and loads. To approximate ground reaction force loads obtained in the forelimb at the gallop, a load of 8,000 N was applied in a finite element model of the equine stifle. The patellar ligaments were modelled to provide the naturally occurring cranial constraint and assigned a modulus of 300 MPa. When 8,000 N of femoral load was applied, 1000 N of proximal patellar tension, representing the *quadriceps* force, was provided to limit cranial femoral translation (Frazer *et al.*, 2019a). In a subsequent study using the finite element model, the load applied to the proximal femur of a 368 kg horse was 900 N, 1,800 N and 3,000 N to represent stall confinement, hand-walking and light exercise in a paddock, respectively. The tensile force applied to the proximal patella at the *quadriceps* attachment was 100 N (Frazer *et al.*, 2019b). To date, no studies of *in vivo* force transmission within the joint and surrounding soft tissues or descriptions of femoropatellar biomechanics exist (Bonilla *et al.*, 2015; Fowlie *et al.*, 2011a,b; Halley *et al.*, 2014; Walker *et al.*, 2016).

In the human limb, total axial forces at the walk reach at least 2-3 times bodyweight. While 65-73% of these forces are transferred through the knee joint, surrounding soft tissues transmit the remaining forces (Fregly *et al.*, 2012; Halder *et al.*, 2012). Finni *et al.* (2000) reported peak patellar tendon force of between 3,000 and 4,000 N during squat jump and counter movement jump.

Information from studies of human knee joint kinematics can partly be applied to the understanding of the equine stifle; however, species differences in anatomy and biomechanics, in particular the higher load on the equine stifle and the quadrupedal stance compared to the human bipedal stance, have to be taken into consideration. Finite element studies of the equine stifle may also provide some insight; however, as demonstrated in human subjects, *in vivo* investigation of the tensile forces acting on the patellar ligaments in all phases of the stride, at different gaits and when performing specific activities, e.g. jumping, would be of great value to further elucidate the aetiology and possible patterns of patellar ligament injury (Dillon *et al.*, 2008).

5. Pathogenesis and histopathology of chronic tendinopathies in humans and horses

Multiple models are used to describe the pathogenesis of chronic tendinopathies in humans (Fu *et al.*, 2010; Millar *et al.*, 2021). Earlier, the disease was regarded as a primary *inflammatory process* – a tendinitis. It was thought that overuse or repetitive mechanical load caused cellular damage that initiated an inflammatory process. Two decades ago this model was by large abandoned, and the disease was then regarded as a *degenerative process* – a tendinosis (Khan *et al.*, 2002), since inflammatory cell infiltrates are not present in early stages of disease and often not in spontaneously ruptured tendons (Almekinders *et al.*, 1997; Józsa and Kannus 1997; Santana *et al.*, 2022). However, this view relied on a definition of inflammation

based on the histologically visible cellular responses, and did not take into account contributions of proinflammatory mediators and tissue-dwelling cells (Scott *et al.*, 2004). Hence, the last decade's research has showed that this is an oversimplification (Rees, 2016; Rees *et al.*, 2014). Today, the pathogenesis is regarded as a complex interplay between degeneration, inflammatory mediators and cellular responses (Millar *et al.*, 2021). Cook and Purdam (2009) established a *continuum theory* where the pathogenesis is divided in three stages: The authors propose that overuse or direct trauma initially causes a reactive tendinopathy characterised by cellular metaplasia and hyperplasia and an increase in extracellular large proteoglycans (and thereby an increase in the amount of bound water). If the reactive tendon is allowed to rest, the changes will be reversed, but if the stimuli continue, the tissue will, at least focally, progress into a stage of disrepair where the cellular responses are more pronounced, the increase in proteoglycans cause disorganisation of the collagen matrix, and vessels and nerves proliferate. These changes can to some degree be reversed, they can persist for long periods, or they can progress into a degenerative stage with collagenolysis, apoptotic and/or necrotic cell death, cellular metaplasia, vessel and nerve proliferation and destruction of normal tissue architecture. A similar description of the process in three stages of injury, failed healing and clinical presentation has been proposed by Fu and co-workers (2010). Several theories have been proposed, yet to date there is little conclusive evidence on what actually initiates the pathological process and why the condition aggravates in some individuals, resolves in some and remains relatively stable in some. However, several authors have suggested a disproportionality between the mechanical load exerted on the tendon and the ability of the tissue at its current stage to cope with this load as the initiating factor (Fu *et al.*, 2010; Steinmann *et al.*, 2020).

Histopathologically, early stages of tendinopathy in humans are characterised by changes in the collagen matrix (including thinning, crimping and increased ratio of type III to type I collagen and loss of normal hierarchical structure), increased proteoglycan content and increased amounts of rounded tenocytes (Cook *et al.*, 2004; Fredberg and Stengaard-Pedersen 2007; Steinmann *et al.*, 2020). As the lesion progresses, the number of apoptotic and/or necrotic cells increases and areas of necrosis, fibrocartilaginous differentiation, mucoid degeneration, calcification of collagen fibres and interfascicular fat cells are frequently seen (Lian *et al.*, 2007). Proliferation of vessels and nerves in both the tendon and in paratenon is also frequent. The vessels are often tortuous and have thick walls and narrow lumens (Järvinen *et al.*, 1997; Józsa and Kannus, 1997).

Tendinopathy in horses, most commonly affecting the superficial digital flexor tendon, has many similarities with Achilles tendinopathy in humans (Dakin *et al.*, 2014; Oreff *et al.*, 2021, Patterson-Kane *et al.*, 2012), though there are some differences in the expression of pro-inflammatory cytokines, for example MMP13 and COX2 (Oreff *et al.*, 2021). To our knowledge, however, there are no systematic histopathological descriptions of more advanced cases or specific descriptions of patellar desmopathy in horses.

6. Patellar tendinopathy in human athletes

Patellar tendinopathy is a common clinical condition primarily affecting athletes performing jumping sports; a prevalence of 32 and 45% have been reported in elite basketball and volleyball players (Lian *et al.*, 2005), respectively. The condition is characterised by load-dependent focal pain at the inferior pole of the patella, which escalates with increasing demands on the knee extensors (Lian *et al.*, 2005; Rudavsky and Cook, 2014). In chronic cases, the pain may be accompanied by structural changes in the proximal posterior portion of the tendon identifiable by diagnostic imaging, such as local increased cross-sectional area; irregular collagen fibre structure; and neovascularisation within and dorsal to the area of structural change (Gisslén and Alfredson, 2005). However, the correlation between pain and diagnostic imaging findings is poor; in a prospective study of 60 young volleyball players, ultrasonographic findings as mentioned above were found in 14/17 painful tendons, but also in 33/43 asymptomatic tendons (Gisslén and Alfredson, 2005). Some studies indicate that pain may be related to ingrowth of sympathetic nerves along neovascularisation of the dorsal paratendinous tissue (Danielson *et al.,* 2008).

The exact etiopathogenesis of patellar tendinopathy is still uncertain, and the lesion predilection site within the proximal posterior portion of the tendon has puzzled researchers for decades. Evidence is however pointing towards regional biomechanical differences within the tendon, both concerning inherent load capacity (Haraldsson *et al.*, 2005), but also regarding tendon strain during loading (Dillon *et al.*, 2008; Lavagnino *et al.*, 2008). Tendon fascicles from the anterior portion had measurable higher peak stress, yield stress and tangent modulus versus fascicles harvested from the posterior portion, indicating that the latter is the weaker (Haraldsson *et al.*, 2005). On the other hand, the highest tendon tensile strain during simulated jump landings in a 60° knee flexion, was found at the proximal posterior aspect even causing disruption of tendon fascicles, precisely at the predilection site for the clinical condition (Lavagnino *et al.*, 2008). The authors suggested altered jump landing strategy as a preventative measure towards patellar tendinopathy, which corroborate the findings of a recent systematic review on risk factors (Sprague *et al.*, 2018). In this review, lack of strong evidence was noted for most of the previously reported risk factors for the condition; however, some evidence was found for

factors involved in jumping/landing biomechanics and greater activity volume.

Treatment for patellar tendinopathy is extensively reviewed in Millar *et al.* (2021). Although there is no 'gold standard' treatment for patellar tendinopathy, conservative treatment including physical therapy with eccentric exercise is commonly used (Schwartz *et al.*, 2015) with success rates reported between 50 and 70% (Visnes *et al.*, 2007). Combined with an exercise program, intralesional injection of platelet-rich plasma failed to improve outcomes versus the exercise programme alone (Scott *et al.*, 2019). Intralesional injection of a sclerosing agent targeting areas of neovascularisation and nerve ingrowths did however improve short-term outcome compared to placebo (Hoksrud *et al.*, 2006), although this treatment was inferior to arthroscopic debridement regarding pain, patient satisfaction and time to return to sport (Willberg *et al.*, 2011). Surgical treatment, either as open or arthroscopic debridement of affected tissue is usually advocated in refractory cases, in which an excellent to good functional outcome was reported in 97% of competitive athletes undergoing arthroscopic patellar release (Maier *et al.*, 2013). Extracorporeal shock wave therapy is another treatment modality used in chronic cases, in which long-term follow-up demonstrated comparable functional outcome to surgical debridement (Peers *et al*, 2003b).

7. Patellar ligament pathology in horses

Of the three equine patellar ligaments, desmopathy of the intermediate ligament is more common (Dyson, 2002b; Reef, 1998). In a retrospective study reviewing ultrasonographic findings of the intermediate patellar ligament (2010-2015), findings compatible with intermediate patellar ligament desmopathy were found in 41 horses, representing 18% of the study population (Hoaglund *et al.,* 2019). Of note was the high occurrence of concurrent abnormalities; in only one stifle was the intermediate patellar ligament desmopathy the sole abnormal ultrasonographic finding.

Patellar ligament injuries were initially more often recognised in horses competing over fences (show jumpers, eventers etc.) than in horses performing in other disciplines (Dyson, 2002b). However, in the retrospective study by Hoaglund *et al.* (2019), 21 horses (51%) were of Quarter or Paint horse breed; 23 horses (56%) performed in non-jumping disciplines; and only nine horses (22%) were hunters/jumpers/eventers. In this population, 9 of 10 horses in which no abnormal findings were detected on a stifle ultrasonographic examination 6 months prior, ultrasonographic signs of intermediate patellar ligament desmopathy was detected following an extended period of rest (Hoaglund *et al.*, 2019). Intermediate patellar ligament injury may in some horses be associated with upward fixation of the patella or previous ligament desmotomy for treatment of upward fixation of the patella (Dyson, 2002b). However, according to Cauvin (2014), ultrasonographic evidence of intermediate patellar ligament injury is not found in cases of upward fixation of the patella.

Lateral patellar ligament injury is often traumatic and associated with an external wound. In a retrospective study reviewing ultrasonographic findings of the lateral patellar ligament in 569 horses (1999-2011), lesions were detected in 18 horses (4%), 12 of which presented with a stifle wound, and all of which had acute onset of lameness (Gottlieb *et al.,* 2016). The mid to insertional portion of the ligament was most often affected and concurrent injuries included fracture of the tibial tuberosity, patella and lateral trochlear ridge; lesions of the middle or intermediate patellar ligaments, medial meniscus, and medial collateral ligament (Gottlieb *et al.*, 2016). Complete traumatic rupture of the ligament without a wound has also been reported (Lantzsch *et al.*, 2005).

Primary injuries of the medial patellar ligament are rare, although they have been reported in trotters (Cauvin, 2014). However, the author emphasises that altered medial patellar ligament appearance may have resulted from previous desmotomy. Enthesopathy of the origin is uncommon due to its strong fibrocartilagenous union with the patella (Barrett and Frisbie, 2016). Medial patellar fractures oriented in the sagittal plane are associated with avulsion and disruption of the medial patellar ligament (Denoix, 1998; Dyson, 1998).

8. Clinical examination in horses and clinical signs of stifle pathology

Gait changes pathognomonic for specific stifle lesions have not been identified. Abduction of the stifle; a reduced cranial phase of the stride; reduced flexion of the limb in flight; and an unwillingness to go downhill may be associated with, although are not specific for, stifle pain (Walmsley, 2011). Flexion tests of the hind limb cannot isolate the stifle joint and are thus not diagnostic for stifle disease. Stifle pathology may be present without palpable joint effusion (Barrett and Frisbie, 2016).

In horses with patellar ligament desmopathy, local clinical signs are often subtle or absent, and the diagnosis may thus be missed. The patellar ligaments should be palpated both with the limb weightbearing and with the stifle flexed (Dyson, 2002b; Whitton, 2014). Palpation may reveal periligamentous oedema, and, particularly in cases with previous medial patellar ligament fenestration or desmotomy, obvious enlargement of the medial patellar ligament. In one horse with middle patellar ligament desmitis, pain could be elicited by firm pressure to the ligament with the limb semiflexed. In one horse with a

severe tear of the intermediate patellar ligament, distension of the femoropatellar joint and periarticular thickening was recognised (Wright, 1995). Horses with patellar ligament desmopathy commonly present with concurrent stifle abnormalities (Hoaglund *et al.*, 2019), chronic lameness or poor performance, and rarely with an acute onset of lameness (Whitton, 2014). Lameness may vary from mild to moderate with an irregular rhythm and slight toe drag. All horses in one report reacted to proximal limb flexion with a slight increase in lameness (Dyson, 2002b). Of 18 horses with traumatically injured lateral patellar ligaments, all presented with acute onset of clinical signs (Gottlieb *et al* 2016). Intermittent upward fixation of the patella may traumatise the femoropatellar joint, leading to secondary effusion and lameness. A horse with upward fixation of the patella or delayed release may avoid full extension of the limb with the appearance of a more crouching gait, in some horses the gait abnormality resembles stringhalt (Walmsley, 2011).

9. Diagnostic anaesthesia of the stifle joint and patellar ligaments

The innervation of the stifle is complex (Rankin, 1975). When patellar ligament injury is suspected, periligamentous infiltration with local anaesthetic can be performed and may result in some degree of improvement of the lameness. Following intra-articular anaesthesia of the femoropatellar joint, horses with patellar ligament injuries may be unaffected or diffusion of the local anaesthetic to the area surrounding the ligament may improve lameness (Dyson, 2002b; Walmsley, 2011). However, concurrent intra-articular injuries may account for this improvement rather than true desensitisation of the patellar ligaments (Dyson, 2002b; Hoaglund *et al.*, 2019; Whitton, 2014).

Future research providing detailed information on the anatomical location and course of sensory nerves supplying the patellar ligaments, and local alterations in innervation accompanying pathological conditions, may enable diagnostic injection techniques that results in specific neural blockade of the patellar ligament(s) without confounding anaesthesia of surrounding structures. In humans, it has been postulated that the proximal region of the patellar tendon receives a rich innervation from the infrapatellar fat pad. Local anaesthetic infiltration targeted at the deep margin of the tendon and the junction between the patellar tendon and infrapatellar fat pad has in line with this yielded significant improvement in pain (Crisp *et al.*, 2008; Dillon *et al.*, 2008). In horses, a possible contribution from the fat pad to the innervation of the patellar ligaments has not been investigated. Histological studies of this region in horses with chronic patellar ligament injury may be valuable to assess suitable locations for local anaesthetic infiltration to desensitise the affected ligament.

10. Diagnostic imaging of normal and injured patellar ligaments

Ultrasonography

Patellar ligament injuries are readily diagnosed with ultrasound (Barrett and Frisbie, 2016). A limited number of studies describe the normal and abnormal appearance of the patellar ligaments in horses. In 2002 Dyson described the ultrasonographic appearance of the patellar ligaments in 10 clinically normal horses. The ligaments were described to be fairly consistent in shape and with a similar uniform echogenicity and fibre pattern and with smoothly defined ligament margins. Since this earlier study ultrasound techniques have evolved, and the quality and resolution of ultrasound images have improved. Injuries have been described ultrasonographically as *core lesions*, similar in appearance to core lesions of the superficial digital flexor tendon (SDFT) (Cauvin, 2014) or more recently as *linear lesions* (tears) (Hoaglund *et al.*, 2019). Lesions are associated with ligament enlargement and decreased echogenicity. Figure 2 depicts a longitudinal split-like lesion in the medial aspect of the intermediate patellar ligament.

In human medicine, ultrasonography with colour or power Doppler has been found useful for the assessment of tendon and ligament injuries. The Doppler technique detects and monitors increased blood flow in musculoskeletal disease (Peers *et al.*, 2003a; Terslev *et al.*, 2001). The use of colour Doppler has been described in horses with chronic flexor tendonitis and suspensory ligament desmopathies (Kristoffersen *et al.*, 2005; Rabba *et al.*, 2018) but not for the patellar ligaments.

Intermediate patellar ligament

The cross-section of the ligament is oval to triangular in the proximal aspect, triangular to circular in its midportion, and triangular distally (Hoaglund *et al.*, 2019). The bone surface, at the patellar origin, is smooth. The ligament has a uniform echogenicity in transverse and longitudinal planes except in the most distal and proximal aspects where hypoechoic striations have been described in normal horses by several authors (Dyson, 2002b; Hoaglund *et al.*, 2019; Penninck *et al.* 1990), which should be differentiated from pathology. These lines were suggested by Dyson (2002) to represent divisions between fascicle bundles but no histology was performed. Van der Vekens *et al.* (2016) described a hypoechoic or heterogeneous area in the transverse plane in the mid to distal aspect of the ligament in active jumping and dressage horses. This area corresponded to a loss of fibre alignment in the longitudinal plane.

Desmopathy of the intermediate patellar ligament has been most frequently identified in the mid to distal aspects (Hoaglund *et al.*, 2019). Lesions were most commonly

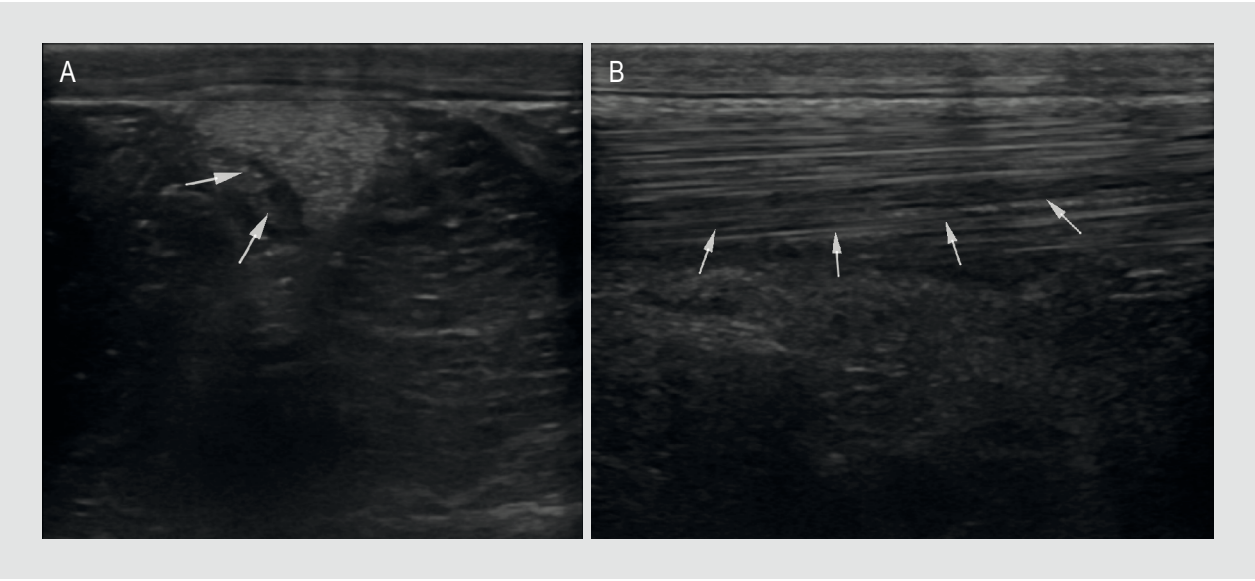

Figure 2. Transverse (A) and longitudinal (B) ultrasonographic images of the intermediate patellar ligament and the infrapatellar fat pad in a horse. Medial is to the left in the transverse image and proximal is to the left in the longitudinal image. A longitudinal split-like lesion (arrows) can be seen in the medial aspect of the ligament.

hypoechoic or anechoic, discrete and well-defined, and less commonly diffuse or ill-defined. The most frequently identified lesion was an obliquely oriented linear tear in a craniolateral to caudomedial orientation. Rounded core lesions in the caudal pole of the ligament were less frequently identified. Insertional enthesopathy was also noted. A severe tear at the ligament origin at the patella and proximal enthesopathy with ligament thickening, irregular echogenicity and bone proliferation at the cranial patella has also been described (Denoix, 1998; Wright, 1995). In addition to well-defined lesions as frequently identified by Hoaglund *et al.* (2019), intermediate patellar ligament injuries may result in ligament enlargement with diffuse hypoechogenic appearance and periligamentous oedema (Dyson, 2002b).

Medial patellar ligament

The medial patellar ligament is triangular in cross-section. The echogenicity has been described as uniform, with a more heterogeneous appearance proximally as fibres spread out into the parapatellar fibrocartilage. At the distal insertion, the ligament merges with the common aponeurosis of the *gracilis* and *sartorius* muscles (Cauvin, 2014; Dyson, 2002b; Singh, 2016).

Medial patellar ligament desmotomy results in a thickened, heterogenous, or hyperechogenic appearance (Cauvin, 2014). The initial appearance following surgery is a thickening of the ligament with hypoechoic/anechoic areas representing haemorrhage, oedema, and/or ligament relaxation, and subcutaneous hypoechoic oedema. This may affect the whole length of the ligament, not only the desmotomy site, possibly due to ligament laxity. After several months, there is extensive thickening and architectural remodelling of the ligament (Denoix, 1998).

Lateral patellar ligament

The lateral patellar ligament varies in shape and echogenicity along its length. The ligament is bilayered at the patellar origin, with ill-defined margins, mottled echogenicity, and mildly irregular fibre pattern. At the lateral trochlear ridge, the ligament is flattened and bilobed. Distal to the lateral trochlear ridge, the shape is oval to triangular, with variable echogenicity and fibre pattern. At the tibial insertion, the ligament is tapered with striations (Gottlieb *et al.*, 2016).

Lateral patellar ligament injury most often affects the mid to insertional portion and is often traumatic and associated with an external wound (Gottlieb *et al.*, 2016). Ultrasonographic findings include enlargement, anechoic or hypoechoic areas, fibrillation and fibre disruption, periligamentous oedema, bony fragments, and displacement of associated fractures (Denoix, 1998; Gottlieb *et al.*, 2016; Lantzsch *et al.,* 2005).

Infrapatellar fat pad

There are no detailed descriptions in the literature on the appearance of the infrapatellar fat pad in normal horses or in horses with lesions in the patellar ligaments. It was described briefly by Cauvin (2014) as being echogenic and having a heterogeneous appearance.

Magnetic resonance imaging

Santos *et al.* (2015) investigated the correlation between magnetic resonance imaging (MRI) findings, gross and histopathological appearance in 10 cadaver stifles of horses regarded as free from stifle disease based on clinical and standard radiographic evaluation. An area of high signal intensity on proton density sequence surrounding the patellar ligaments was identified as the infrapatellar fat pad. The medial patellar ligament had intermediate signal intensity, and similar signal alterations were also detected in the intermediate and lateral patellar ligament. Multiple areas of loose connective tissue and adipose tissue were infiltrating between the ligament fibres on histologic examination in these regions.

Computed tomography

Van der Vekens *et al.* (2011) described the appearance of the patellar ligaments on computed tomography (CT) examination. The authors described the ligaments to have a homogenous attenuation through the entire ligaments except in the distal aspect of all three ligaments which had hypoattenuating striations. In this study, no histological examination was performed.

11. Treatment and prognosis

Treatment approaches for patellar ligament desmopathy may be conservative or surgical. Reported conservative treatment includes rest followed by a rehabilitation programme; in selected cases combined with intralesional or intraarticular injections of anti-inflammatory and/or regenerative substances, or combined with extracorporeal shock wave therapy (Dyson, 2002b; Hoaglund *et al.*, 2019). In horses with patellar ligament lesions and concurrent pathology that is accessible from the intra-articular instrument position, arthroscopic surgery allows for diagnostic exploration and debridement of lesions (Dyson, 2002b; Gottlieb *et al.*, 2016; Hoaglund *et al.*, 2019; Wright, 1995). To date, as only few cases of patellar desmopathy and their outcome have been described, it is not possible to determine the success rate of the reported treatment alternatives.

Previously injured patellar ligaments will often continue to appear abnormal ultrasonographically despite clinical improvement or resolution of clinical signs (Hoaglund *et al.,* 2019). Thus, ultrasonographic findings may not indicate an active or clinically significant lesion and should be correlated to clinical signs (Barrett and Frisbie, 2016). Gottlieb *et al.* (2016) re-evaluated the traumatically injured lateral patellar ligament of 4 horses with clinical improvement at 2.5-15 months. In 2/4 cases no ultrasonographic evidence of healing was detected.

In the study by Hoaglund *et al.* (2019), detailing 41 horses with intermediate patellar ligament injury, repeat scans at 2-month intervals revealed a worsening in ultrasonographic appearance in 54% of cases, no change in 31%, and an improvement in 15%. On long-term follow-up of 25 of these horses, 90% were sound, 72% returned to work at the same level or higher, 28% returned to work at a lower level, and 8% did not return to work. However, in only one horse was intermediate patellar ligament desmopathy the sole ultrasonographic finding, and in only 11/42 horses was the intermediate patellar ligament lesion categorised as the most significant injury. Treatments varied and were mostly aimed at the concurrent intra-articular pathology. Local treatment of the intermediate patellar ligament with either intralesional injection or extracorporeal shock wave therapy was performed in 16 horses in total. The authors conclude that intermediate patellar ligament lesions may be either primary or contributory components of lameness and their absolute clinical relevance remains difficult to determine (Hoaglund *et al.*, 2019).

The outcome of horses with patellar ligament injury in the recent studies contrasts to earlier reports, in which a poor prognosis for return to work was indicated (Dyson 2002b; Wright, 1995). Of eight horses with intermediate patellar ligament desmopathy, none returned to their previous level of competition (Dyson, 2002b).

12. Conclusions

The equine stifle and human knee have the same basic anatomical structure. The patellar tendon in humans corresponds to the intermediate patellar ligament in horses and this ligament is more often diagnosed with desmopathy based on structural changes detected ultrasonographically. Changes in the medial patellar ligament may be related to interventions aimed at treating upward fixation of the patella. Lateral patellar ligament lesions are often related to external trauma.

In human athletes diagnosed with patellar tendinopathy the correlation between pain and diagnostic imaging findings is poor. The criteria for diagnosing patellar ligament desmopathy associated with clinical disease in horses have not yet been adequately defined. In lame horses, structural abnormalities have been detected at various locations within the patellar ligaments, in particular the intermediate; however, the clinical significance is not clear since comparable findings have been observed in sound working horses. To ensure horse welfare and economic interests of the horse industry, further studies are required to describe ultrasonographic normal variation of the patellar ligaments, pathological changes verified by histopathology, and their progression in healing. Further, areas of maximal ligament strain at the different phases of the gait cycle, and the regional innervation and specific placement of

diagnostic anaesthesia to verify pain originating from the ligaments should be investigated. If patellar ligament desmopathy exists as a clinical entity in the horse, optimal diagnostic criteria, treatment strategies and convalescence programmes should be established.

Conflict of interest

The authors declare no conflict of interest.

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