Injuries of the Accessory Ligament of the Deep Digital Flexor Tendon in Forelimbs and Hindlimbs

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Take Home Message—Typical injuries of the accessory ligament of the deep digital flexor tendon (ALDDFT) are associated with sudden onset lameness and swelling in the metacarpal or metatarsal region and are straightforward to diagnose. Lesions of the proximal aspect of the ALDDFT in both forelimbs and hindlimbs have been overlooked historically and are more challenging to diagnose because frequently there are no localising clinical signs. Injuries can occur alone or in association with proximal suspensory desmopathy. Diagnosis is reliant on diagnostic analgesia and ultrasonography, or occasionally magnetic resonance imaging. In hindlimbs, degenerative lesions of the ALDDFT may result in a postural change with inability to load the heel or flexion of the fetlock.

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I. ANATOMY

The forelimb accessory ligament of the deep digital flexor tendon (ALDDFT) has been described as a distal continuation of the palmar carpal ligament, but a recent cadaver magnetic resonance imaging study demonstrated fibres originating from the palmar aspect of the third and fourth carpal bones which blended with the palmar carpal ligament in all horses examined (n=30). The ALDDFT in hindlimbs is smaller than in forelimbs, and based on a cadaver study of 165 limbs was found to be absent in 6% of horses and was occasionally bifid or trifid. Based on magnetic resonance images it appears to originate from the plantar aspect of the distal plantar ligament that runs between the calcaneus and the central and third tarsal bones. Normal reference ultrasonographic images of the proximal aspect of the ALDDFT in forelimbs (Fig. 1) and hindlimbs (Fig. 2) are illustrated.

The accessory ligament of the deep digital flexor tendon is a crescent-shaped structure of uniform echogenicity, thicker medially than laterally (arrows), clearly separated from both the deep digital flexor tendon and the suspensory ligament.

Fig. 1 Longitudinal ultrasonographic image of a distal carpal region. Proximal is to the left. The accessory ligament of the deep digital flexor tendon (arrows) originates from the palmar aspect of the third carpal bone and is uniform in echogenicity and has a straight palmar contour. It blends with the palmar carpal ligament (arrow heads).

Fig. 2a. Transverse ultrasonographic image of a left metatarsal region obtained from the plantaromedial aspect of the limb 4 cm distal to the tarsometatarsal joint, using a 10MHz virtual convex array transducer. Medial is to the left and plantar is to the top. Fig. 2b. Longitudinal ultrasonographic image of a left metatarsal region centred at the level of the tarsometatarsal joint, using a 10MHz linear array transducer. Proximal is to the left and plantar is to the top. The accessory ligament of the deep digital flexor tendon is a thin structure of uniform echogenicity (arrows).
II. CONVENTIONAL INJURIES OF THE ALDDFT

Typical injuries of the ALDDFT are associated with sudden onset lameness and swelling in the metacarpal region, or less commonly the metatarsal region. Injuries are more common in ponies, cob-types and warmblood breeds compared with Warmbloods and occur more frequently in horses > 10 years of age compared with younger horses, associated with ageing degenerative changes. Diagnosis is straightforward by palpation and ultrasonographic examination. Typically, there is enlargement of the ALDDFT and a diffuse decrease in echogenicity. Less commonly forelimb lesions are restricted to the lateral margin and these are more challenging to manage.

Injury of a forelimb ALDDFT can occur concurrent with superficial digital flexor tendonitis or as a sequel to previous injury of the superficial digital flexor tendon. Adhesions between these structures may predispose to the development of a permanent flexural deformity of the metacarpophalangeal joint.

III. PROXIMAL FORELIMB INJURIES

Proximal lesions of the ALDDFT in both forelimbs and hindlimbs have been described, either alone, or in association with proximal suspensory desmopathy (PSD) or as a sequel to PSD. Unlike conventional injuries there are often no localising clinical signs, especially in forelimbs.

The proximal aspect of the ALDDFT from its origin to 10 cm distal to the carpometacarpal joint was examined ultrasonographically in both forelimbs of 10 control horses with no history of carpal or metacarpal region pain, in both transverse and longitudinal planes. The most proximal aspect of the ALDDFT could only be reliably assessed in longitudinal images. There was a smooth osseous contour at the origin of the ligament on the palmar aspect of the third carpal bone. The ligament was uniformly echogenic with a regular fibre pattern, and had a straight palmar profile. The origin of the ALDDFT from the palmar aspect of the third carpal bone blended with the palmar carpal ligament. In the distal carpal and proximal metacarpal regions, the ALDDFT was uniform in echogenicity in transverse images and approximately rectangular in shape. It was separated from the dorsal border of the deep digital flexor tendon (DDFT) by variable amounts of fluid in the carpal sheath. From approximately 9 cm distal to the accessory carpal bone the palmar border became concave and at 10 cm the ligament started to become thicker laterally than medially. Its dorsal and palmar margins were clearly defined. The ALDDFT appeared bilaterally symmetrical in all horses. The dorsopalmar thickness at the origin and at 8 cm distal to the accessory carpal bone ranged from 0.78 - 0.95 cm (mean 0.86 cm, median 0.90 cm) and 0.52 - 0.77 cm (mean 0.61 cm, median 0.64 cm), respectively.

Twenty-two horses were examined between January 2006 and November 2014 with lameness associated with injury of the proximal aspect of the ALDDFT. Horses ranged in age from 5 to 12 years (median 6 years) and were used for eventing (n=6), showjumping (n=5), dressage (n=6), general purposes (n=3), driving (n=1) and racing (n=1). The duration of lameness was 1 - 4 months (median 2 months); three horses had a history of poor performance and lameness had not been recognized.

No horse had any significant palpable abnormality. Sixteen horses had unilateral lameness and six horses had bilateral lameness. Lameness ranged from grade 2 to 5 out of 8 and was consistently worst on the lunge or ridden, usually with the lame limb on the outside of a circle. Two horses showed mild improvement in lameness after palmar (at the junction of the proximal ⅔ and distal ⅔ of the metacarpal region) and palmar metacarpal nerve blocks (low 4-point nerve block) (2 ml mepivacaine at each site). Lameness was substantially improved or abolished by palmar metacarpal (subcarpal) nerve blocks (2 x 2 ml mepivacaine) in most horses (n=19); four horses then showed lameness in the contralateral forelimb that was also abolished by palmar metacarpal (subcarpal) nerve blocks. In three horses, there was mild improvement in lameness after subcarpal analgesia and substantial improvement after median and ulnar nerve blocks. Intra-articular analgesia of the middle carpal joint did not improve lameness in any horse.

No significant radiological abnormality of the carpus and proximal metacarpal region was detected in any horse. Ultrasonographic abnormalities were only identified in the lame limb in the unilaterally lame horses, but were seen bilaterally in the bilaterally lame horses. Nineteen horses had lesions restricted to the origin of the ALDDFT (three bilaterally), characterised by enlargement of the ALDDFT, a convex palmar contour and generalised reduction in echogenicity. Two horses had a proximal lesion which extended distally to 8 cm distal to the accessory carpal bone. The twenty-second horse had a lesion restricted to the proximal metacarpal region extending to 9 cm distal to the accessory carpal bone characterised by enlargement and a diffuse reduction in echogenicity. In addition, 14 horses (unilateral 5, bilateral 9) had concurrent injuries of the suspensory ligament in the same limb.

Horses were used for dressage (n=5), general purpose (n=4), eventing (n=3), showjumping (n=1) and racing (National Hunt) (n=1). The horses ranged in age from 5 to 12 years (mean 7.5 years; median 6 years). Median lameness grade in straight lines, on the lunge on a firm surface was 2/8 (range 1 – 5). On the lunge on a soft surface lameness was consistently worst with the limb on the outside (median 4/8, range 1 – 6). Of the 9 horses with bilateral forelimb lameness, 4 horses were detectably lame in both forelimbs before local analgesia; lameness appeared worse on small circles with the lame forelimb on the outside. The remaining 5 horses with bilateral forelimb lameness showed unilateral lameness initially, but when lameness in the lame limb was abolished, lameness became apparent in the contralateral limb. In unilaterally lame horses, lameness was consistently worse on a circle with the lame forelimb on the outside. Subtle thickening in the region of the ALDDFT in the proximal metacarpal region was identified in two horses and one horse had heat in the proximal metacarpal region.
Lameness was abolished by perineural analgesia of the palmar metacarpal (subcarpal) nerves in 17/23 limbs. In the remaining limbs intra-articular analgesia of the middle carpal joint (n = 2) or an ulnar nerve block (n = 4) were required to eliminate the lameness. Sixteen of 23 lame forelimbs had concurrent PSD and desmitis of the ALDDFT. Proximal suspensory desmopathy alone was present in 3 forelimbs (in the less lame limb of bilaterally lame horses). Proximal injuries of the ALDDFT alone were diagnosed in 4 forelimbs. One of these horses was humanely destroyed because of a poor prognosis due to lameness in all four limbs. There was no space between the 2 ligaments in 6 of the 16 affected forelimbs with both PSD and desmitis of the ALDDFT and in 2 of these there were strong suspicions of adhesion formation. Post mortem examination revealed extensive adhesions between the suspensory ligament and the ALDDFT.

Horses originally were treated conservatively by box rest and controlled walking exercise for a minimum of six months. The results were disappointing, with the majority (6/8, 75%) having persistent lameness. Two horses have been treated by intralesional injection of mesenchymal stem cells, and both had resolution of lameness and returned to full athletic function. Six horses treated similarly returned to full work but have had recurrent lameness after 6 to 12 months and did not respond to shockwave therapy.

The horses in this small case series were generally younger than horses with lesions of the ALDDFT in the metacarpal region and the pathogenesis of injury may be different. The sequence of events in horses with concurrent lesions of the ALDDFT and PSD is not known. However, adhesions between the suspensory ligament and the ALDDFT may be a predisposing factor.

Lesions of the ALDDFT diagnosed using magnetic resonance imaging have been described; however, the descriptions of the lesions were similar to the normal appearance described by Nagy and Dyson. Successful management by desmotomy was subsequently documented.

### IV. PROXIMAL HINDLIMB INJURIES

Between January 2006 and December 2010, there were 8 horses with hindlimb lameness associated with primary injuries of the ALDDFT. They ranged in age from 7 to 13 years (mean 10.6 years), 7 of which had lameness of 1 to 3 weeks’ duration; the eighth horse had been intermittently lame for 4 months, but had remained in work until an acute, severe exacerbation of lameness 4 weeks previously. Two horses showed grade 2 lameness at the walk; at the trot the most frequent lameness grade was 4 in straight lines, with a range of 2 to 5. Seven horses had mild localised heat and subtle to moderate oedematous soft tissue swelling in the proximomedial aspect of the metatarsal region; in one of these horses, swelling also extended laterally. The eighth horse had mild generalised soft tissue thickening in the proximal metatarsal region. In this horse plantar (at the junction of the proximal ¾ and distal ¼ of the metatarsal region) and plantar metatarsal nerve blocks did not alter the lameness, but perineural analgesia of the deep branch of the lateral plantar nerve (3 ml mepivacaine) improved lameness by approximately 80%. Intra-articular analgesia of the tarsometatarsal (TMT) joint did not alter the lameness. Diagnostic analgesia was not performed in the remaining 7 horses. No significant radiological abnormality of the tarsus and proximal metatarsal region was detected in any horse.

Ultrasonographic abnormalities were only identified in the lame limb and in the 7 horses with oedematous swelling were characterised by marked enlargement of the ALDDFT, with reduced space between the SL and the ALDDFT, and extensive hypoechogenic to anechogenic areas, extending from just distal to the TMT joint up to 9 cm distally. The eighth horse had enlargement of the ALDDFT and patchy hypoechogenic areas which extended as far proximally as the ligament could be followed in the plantar tarsal region. This horse had increased radiopharmaceutical uptake in the plantar aspect of the distal row of tarsal bones. Magnetic resonance imaging revealed increased signal intensity in the proximal aspect of the ALDDFT in T1- and T2-weighted images and fat suppressed images. There was an irregular plantar contour of the third tarsal bone and decreased signal intensity in T1- and T2-weighted images in the endosteal bone consistent with mineralisation.

Between April 2007 and April 2014, five horses with hindlimb lameness were identified with concurrent (n = 2) PSD and desmopathy of the ALDDFT or the development of desmopathy of the ALDDFT after treatment of PSD by neurectomy of the deep branch of the lateral plantar nerve and plantar fasciotomy (time since surgery 6 – 12 months) (n = 3). All 3 had become sound after surgery, resumed work progressively and had returned to their previous level of work for 3 weeks, 1 and 2 months respectively before recent recurrence of lameness (unilateral 2 horses, bilateral 1 horse). Lameness was abolished in 5 of the 8 limbs after local infiltration of local anaesthetic solution (n = 3) or perineural analgesia of the deep branch of the lateral plantar nerve (n = 2) and there was approximately 50% improvement in lameness in the remaining 3 limbs; residual lameness was abolished by a tibial nerve block. No improvement in lameness was seen after intra-articular analgesia of the tarsometatarsal joint performed on a subsequent occasion in any horse.

### V. HINDLIMB INJURIES AND POSTURAL ABNORMALITIES

Between January 1992 and July 2004, ten horses had an insidious or sudden onset of a postural abnormality of one or both hindlimbs characterised by elevation of the heel or semiflexion of one or both metatarsophalangeal joints. These were thick-skinned cob-types or British native pony breeds, making accurate palpation difficult. In seven horses, localised soft tissue swelling in the region of the ALDDFT was identified but only one horse had pain on palpation. In two horses, there was improvement in posture while the horse was sedated with detomidine and butorphanol. Mild (30–40%) improvement in lameness was seen after analgesia of the plantar metatarsal nerves at subtarsal level in 4 of 5 horses. Ultrasonographic
examination was frequently challenging because of thick skin but in all horses the ALDDFT was enlarged with diffuse decrease in echogenicity. All horses remained lame, despite surgical treatment by desmotomy or desmectomy of the ALDDFT in 6 horses. Surgery resulted in an immediate improvement in posture post operatively, but this was short-lived.

VI. CONCLUSIONS

Proximal injuries of the ALDDFT are easily overlooked without systematic ultrasonographic assessment. Currently, the success of treatment has been limited.

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Declaration of Ethics

The Author has adhered to the Principles of the Veterinary Medical Ethics of the AVMA.

Conflicts of Interest

The Author has no conflicts of interest.

REFERENCES AND FOOTNOTE


ADDITIONAL READING