Tendon sheath diseases: disorders and treatment

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Soft tissue injuries in equine orthopaedics is a frequent situation encountered by clinicians; tendons and ligaments disorders still represent a great challenge for the equine veterinarian in terms of their precise diagnosis and treatments. The tendons, especially flexor tendons, and their related structures can also be affected inside the sheaths situated at the level of great motion joints where they permit a better gliding function. The most frequently affected sheaths are Digital Flexor Tendon sheath (DFTS), Carpal Sheath (CS) and Tarsal Sheath (TS) other than extensor sheaths which can also be affected but rarely responsible for lameness. The above mentioned sheaths can be affected by different disorders that we’re going to present specifically apart from those related to tenosynovitis infection, which can be responsible for serious lameness and obvious local signs and must be promptly diagnosed and treated like any synovial septic disorder.

Digital flexor tendon sheath

Synovial effusion of the DFTS is quite common and not normally associated with lameness especially in idiopathic cases when the condition is encountered bilaterally. Sometimes, the sheath distension is associated with lameness and the use of modern diagnostic techniques has permitted the identification of specific pathologic conditions. The ultrasonography and tenoscopy have become commonly used to diagnose and treat disorders of the digital flexor sheath. Recently, Magnetic Resonance Imaging has ameliorated our ability to diagnose some disorders of the tendons that are not always well addressed inside the digital sheath. The DFTS is a thin-walled structure that encompasses the deep digital flexor tendon (DDFT) and the superficial digital flexor tendon (SDFT) from the distal third of the metacarpal/tarsal area to the distal level of the pastern. The sheath wall is composed of two layers: the fibrous external one and the inner synovial layer. The palmar wall of the sheath incorporates the annular ligament, proximally palmar annular ligament (PAL) and the proximal and distal digital annular ligaments and they normally measure 2 mm or less in thickness. Dorsally, the sheath is formed by the proximal scutum, middle scutum and distal sesamoidian ligaments. Proximal and distal scutum are strong fibroucartilagineus pads that allow gliding of the digital flexor tendons. The manica flexoria (MF) is a fibrous ring that originates from the lateral and medial edge of SDFT and encircles the DDFT in the proximal part of the sheath till the apexes of the proximal sesamoid bones (PSB). From the dorsal and axial part of the PAL originates a mesotendon that attaches itself to the palmar aspect of the SDFT; another mesotendon attaches itself to the palmar aspect of the DDFT distally inside the sheath. To address the DFTS as a cause of lameness, different diagnostic modalities can be used such as local intrathecal or perineural anesthesia. The synoviocentesis is performed through proximo-lateral (medial) recesses, abaxial recesses, distally to the base of the PSBs and distal recess in the distal and axial aspect of the pastern where the presence of the mesotendon, axially located, must be considered and the needle has to be inserted laterally or mediately at 45° direction. Local anesthetic solution of 6-10 mL should be injected and 5-10 minutes waited. I personally prefer lesser amount of anesthetic solution and a wait of 5 min. to effect; some passive flexion of the fetlock just after injection is helpful. Sometimes the result is partial and perineural block must be used. In my experience, the response to the flexion test of the fetlock is very useful as it is often very positive in cases of clinically important tenosynovitis of DFTS.

Diagnostic ultrasonography is the most commonly used technique to evaluate the DFTS from proximal (3A scanning zone) to distal aspect (P1C). The Superficial and Deep Digital Flexor tendons (SDFT and DDFT) are easily visualized especially in case of tenosynovitis when the sheath is distended by fluid. A normal mesotendon joins lateral and medial border of the DDFT and should not be mistaken for an adhesion. Dorsal to DDFT, the MF can be appreciated for its thickness especially in the longitudinal scan. More distally PAL should be measured and considered normal if less than 2 mm in thickness. Dorsally to it, the mesotendon attaching the palmar aspect of SDFT can be visible especially in case of fluid distension by avoiding excessive pressure to the probe. The proximal and distal digital annular ligaments usually can’t be recognized unless they are abnormally thickened. Tenoscopy has assumed great importance as a diagnostic and therapeutic modality for DF tenosynovitis. The sheath is entered with the scope in the abaxial collateral recess, 1 cm distal to the base of the PSBs and entirely explored from proximal to distal. Many abnormalities not very visible with ultrasonography can be visualized and treated as debridement of fibrillated and torn areas of the digital flexor tendons, synovial mass and adhesion removal and PAL desmotomy. More recently magnetic resonance imaging is also used to diagnose digital sheath disorders for its superior chance to detect soft tissue abnormalities but when performed in general anesthesia, it is inferior to tenoscopy in term of its convenient application, being limited only to diagnostic purposes.

Tenosynovitis of the DFTS can be an acute and primary condition that normally responds to antiinflammatory treatment and rest. The treatment can be systemic or local with intrathecal administration of 20 mg of Hyaluronan and steroid (metylprednisolone acetate 40 mg or 10 mg of triancinolone acetonide). I usually prefer to inject the sheath and rest the horse for 2 weeks and if the swelling and lameness relapse after the training is resumed, a primary lesion involving the flexor tendons and MF can be suspected for not always being sonographically assessed. In these cases tenoscopy should be suggested for complete examination of the sheath and treatment. Longitudinal tears of the DDFT, a condition most
frequently encountered during endoscopic examination, can be treated with motorized synovial resector that can also be used for the removal of proliferating synovial mass and adhesion. The prognosis for returning to athletic function for DDFT longitudinal tears is only 40% with long rest and inferior for those with long tears (18%). In our retrospective study where 33 lame horses were subjected to tenoscopy for evaluation and treatment, 25 horses were affected by DDFT longitudinal tears that were debrided with motorized equipment; of the 22 with known follow-up, 12 (54%) returned to previous athletic activity with a rest superior to 6 months. Tendon core lesions inside the sheath are reported to have a better prognosis if treated with intralesional injection of Mesenchymal Stem cells. Better prognosis can be achieved in cases of tenosynovitis complex with masses proliferation and adhesions when tenoscopic debridement and PAL desmotomy were reported to be successful in almost 70% of cases. Even in horses with MF damage treated with its excision and removal are reported to have successful outcome in 76% of cases.

PAL desmitis and thickening is a primary or secondary condition consequent to chronic digital tenosynovitis and it can be responsible for lameness. In our experience, primary condition and lameness is quiet rare and even in case of obvious thickening and etrochogenic appearance associated to tenosynovitis, a primary chronic disorder inside the sheath is diagnosed or should be suspected. Clinically, PAL thickening is easily diagnosed when a protruding notch in the palmar aspect of the fetlock is noticed. The lameness is better eliminated by perineurial (low 4 points) than intrathecal anesthesia. Primary conditions can be successfully treated with antinflammatory medications and rest but PAL desmitis is more frequent a chronic condition and requires desmotomy that can be performed through an open, minimally invasive or endoscopic surgery. The operation resolves the lameness in cases of primary condition.

**Carpal Sheath**

Lameness originating in the carpal sheath has probably been underestimated and with the improvement in diagnostic techniques, several pathological conditions have been addressed as causes of carpal canal swelling and low grade lameness. Classic tenosynovitis, radial osteochondroma, tendonitis and myotendinitis of the flexor tendons and fracture of the accessory carpal bone have been recognized as causes of carpal canal syndrome. Recently, radial physeseal caudal spikes, exostoses developing at the level of the closed physis, are reported as a cause of damage to the deep digital flexor tendon (DDFT) and as being responsible for carpal tenosynovitis with the same pathogenesis reported for the osteochondroma, from which they differ radiographically and histologically. Both kind of exostoses mentioned can be responsible for deep digital flexor tendon damage and consequently, for the clinical signs of tenosynovitis and mild lameness. In my experience, carpal canal tenosinovitis is often related to the caudal exostosis, which can be under diagnosed especially if of small size. Carpal sheath tenosynovitis results in intermittent and eventually chronic distension of the carpal sheath, well clearly visible on the proximal lateral aspect with distension of its recess located between the ulnaris lateralis muscle caudally and lateral extensor tendon cranially. In more severe cases additional swelling can be encountered on the proximal medial aspect other than, obviously, on the distal recess in the proximal metacarpal area. Occasionally, the horse can be lame more often after the exercise when intense bleeding fluid is obtained through the centesis; this finding is frequently encountered in cases of distal radial exostosis, which impinge and damage the DDFT. Verification of carpal sheath lameness requires intrathecal anesthesia. Follow-up radiographs are necessary to demonstrate the presence of osteochondroma or physeseal spike, which aren’t always well noted especially if small in size. In these cases, the ultrasonography is of extreme value for the definitive diagnosis other than essential to evaluate the damage to the DDFT. It is predictable that the synovial effusion of the sheath, in the presence of an osteochondroma or physeseal spike, is caused by the exostosis damaging the soft tissue structures and in particular the DDFT, due to its location next to the site where the osteochondromas are typically encountered. The characteristic site where the osteochondroma develops is axial to the medial edge of the caudal cortex of the distal metaphysis of the radius although the lateral location is reported, and this explains the difficulty in detecting, radiographically, the small exostoses on the latero-medial and dorsomedial-palmarolateral oblique (Dm-PiO) radiographic views for the superimposition of both edges (lateral and medial) of the caudal cortex of the radius (LM views) due to its concave shape. On the other hand, the small size of the exostosis is clinically very important for their frequently sharp shape, as shown in our cases, where the osteochondromas were responsible for the damage to DDFT. In these cases, ultrasonography can be quite accurate as a diagnostic procedure even more than radiography. Ultrasonography can also highlight primary tendinitis lesions encountered in the proximal metacarpal area especially of the superficial digital flexor tendon with its enlargement and core lesion, which is a good indication for carpal retinaculum desmotomy. In the proximal part of the sheath, the muscolotendinous junction is not to be mistaken for damaged tendons.

Most cases of carpal sheath distension without any obvious signs of causes are treated with intrathecal corticosteroid and hyaluronan, but often, carpal tenosynovitis has a cause which requires a surgical treatment through an arthroscopic approach. Additionally, horses with tendinitis of the superficial digital flexor tendon are frequently treated with superior check ligament debridement which can be performed arthroscopically.

**Radial Osteochondroma and Radial physeseal spike removal.** The access to the sheath is performed as described for the sheath exploration; the osteochondroma and the physeseal spike are clearly localised on the palmar radial metaphysis; a second portal is made at the level of the exostose with the help of a needle. Through a stab incision, an osteotome of 4 mm
is introduced to separate the osteochondroma from the radius and it is removed with a large ronguer. The site of the bone can be smoothed with a curette, or a rasp or a motorized burr. The damage to DDFT is eventually debrided and the sheath finally flushed before the skin closure.

**Proximal check ligament desmotomy.** This procedure is preferably performed in dorsal recumbency and is recommended bilaterally and for a better control of bleeding. The arthroscope is inserted for the sheath exploration. It is important to recognize the point of blending of radial head of the DDFT with the main body of the tendon as landmark for the distal edge of the ligament. Instrument portal is made 4-5 cm distal to the arthroscope portal; the distal landmark is palpated with the blunt obturator and the ligament is severed with a curved, serrated banana blade or with curved fixed scalpel, as the author prefers. Other cutting modalities can be used as radiofrequency. The ligament is severed from distal to proximal direction. More proximally, the ligament becomes ticker and the body is located beyond the proximal reflection of the carpal sheath. This area is more quickly severed using a biopsy punch ronguer or arthroscopic scissors which allows a better visualization of the proximal extent and any contained vessel especially if the arthroscope and instrument are inverted with the scope in the distal portal viewing in proximal direction. Bleeding from damage to the artery contained in the proximal part of the ligament can be controlled with pressure or with bipolar laparoscopy or ligaclips. Penetration of the sheath of the flexor carpi radialis is routine and represents the medial landmark of the dissection.

**Carpal retinaculum desmotomy.** This procedure can also be done arthroscopically and it is preferred when a carpal tunnel syndrome is diagnosed. In the author’s experience, this procedure is more frequently indicated when the horse presents a tendinitis of the SDFT next to the accessory carpal bone and can be advisable performed with other specific treatments for the tendon damage (regenerative treatments). The desmotomy is performed with the same arthroscopic approach and the instrument portal at the level of the remnant physis. The ligament is severed on its medial aspect, 5-10 mm caudal from its aponeurosis with the palmar carpal ligament from the proximal part to 10-16 mm proximal to the accessory carpal bone. The entry of the sheath of the flexor carpi radialis is the medial landmark and the palmar retinaculum runs deep alongside, although there are some portions that are superficial to this tendon but in the author’s experience, the desmotomy of this superficial portion wasn’t required to release the carpal tunnel. Desmotomy is performed with the same blade used for the superior check ligament desmotomy: serrated banana blade or with curved fixed scalpel.

The prognosis is good to excellent in cases of osteochondroma and physeal spike while it is obviously guarded in all other cases where desmotomy of the superior check ligament or the carpal retinaculum is performed for cases of superficial digital flexor tendonitis.

**Tarsal sheath**

Tarsal sheath represents the synovial sheath of the lateral digital flexor tendon at the level of the hock. The most frequent disorder is distension without lameness well known as idiopathic thoroughpin, very rarely associated with obvious lameness related to primary or secondary damage of the lateral digital flexor tendon. The DDFT in the hind leg is formed by a large lateral digital flexor tendon and a thin medial digital flexor tendon that pass in different sheaths and they fuse in the proximal metatarsal area. The lateral digital flexor muscle is located on the caudal aspect of the tibia and the tendon originates just proximal to the tarso-crural joint, passes medial to the calcaneus over the sustentaculum tali and more distally medial to SDFT where it joins the medial digital flexor tendon just distal to the tarso-metatarsal joint. The sheath accompanies the entire tendon from its musculo-tendinous junction at the distal aspect of the crus where it forms a large pouch dorsal to the calcaneus tendon and becomes more enlarged in the lateral aspect when distended. The sheath ends in the recess dorso-medial to the DDFT in the proximal metatarsal region. At the level of sustentaculum tali, the sheath with its content is compressed by a transverse thick ligament, the plantar retinaculum, which extends distally with a superficial fascia. The vessels and plantar nerves run inside the retinaculum and in the caudo-medial aspect of the sheath, a mesotendon joins the tendon for its entire length.

Tarsal tenosynovitis occurs in light form with mild distension and without lameness in young warm blood horses especially with straight hock conformation and most of the time, it tends to heal spontaneously. The inflammatory condition can be consequent to direct trauma for a kick or during jumping hitting a hard obstacle. A serious trauma to medial aspect of the hock can be responsible of fragmentation of the sustentaculum tali well visible from a dorso45medial-plantarolateral oblique radiographic view of the hock and better with a skyline view of the calcaneus. The inflammation can lead to chronic condition with synovial thickening and parietal fibrosis between the synovial wall and the tendon and more frequently associated with lameness. Primary sprain of the tendon occurs for overstretching over the sustentaculum tali and can be ultrasonographically diagnosed for the presence of irregular hypoechic lesion. Distention of the sheath must not be confused with tarsocrural artrosynovitis when other than the enlargement of the caudal recess, even the dorsomedial is distended. The lameness is exacerbated by the hock flexion that can be reduced to chronic cases with extensive fibrosis. The sheath should be confirmed as a cause of lameness with intrathecal injection using 10 mL of local anesthetic solution. Ultrasonography, with 7.5 MHz linear transducer, is performed on the medial aspect of the hock after an accurate preparation including partial removal of the chestnut that can be softened with warm water. In chronic cases, a thickening of...
the parietal wall can be detected and adhesions with the tendon can also be damaged with the presence of hypoecogenic lesions. The tendon is frequently affected by longitudinal tears, which can be missed.

The acute and primary conditions respond well to systemic and local antinflammatory treatment as reported in the previous condition combined with reduced exercise for 2-3 weeks. The chronic and symptomatic disorders can be treated tenoscopically and the prognosis is fair for horses with fragmentation of sustentaculum tali and guarded for those with tendon damage treated with motorized debridement.

References
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