

Introduction to Equine Tendon Injury

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Tendon injury initiates inflammation characterized by vascular leakage, fibroplasia, and scarring. Healing rarely results in normal microscopic or functional structure, leaving it prone to reinjury. Authors' address: Marion duPont Scott Equine Medical Center, PO Box 1938, Leesburg, VA 20177; e-mail: jgbarret@vt.edu. © 2008 AAEP.

1. Introduction

Equine tendon injuries have been recognized and treated for centuries. In early American texts, poor conformation was considered a primary risk factor. "Tied-in" at the knees and long, weak, and slender pasterns were thought to predispose the horse to injury because of mechanical disadvantage. Any trait that favored fetlock extension such as low heels and long toes was thought to increase a horse's risk of injury, particularly injury to the superficial digital flexor tendon (SDFT). "Race horses and hunters and horses required to trot at sharp pace for long periods are the most general sufferers."¹ Injury of the deep digital flexor tendon (DDFT) was recognized less commonly, and causes and risks of injury to the check (accessory) and suspensory ligaments were similar to those currently recognized.² Injuries were reported as a strain caused by either tension and repetitive strain or bruising from external trauma.

Until use of ultrasonography, diagnosis and localization of tendon injuries relied solely on recognition of lameness and external palpation. The term "bowed tendon" was derived from the shape exhibited by the SDFT or DDFT that is caused by inflammation. Swelling is the hallmark of tendon or ligament injury, but ultrasonography can reveal in-

juries before pain or swelling are observed or palpated as well as abnormalities of a chronic nature. Differentiating the pain and inflammation from individual tendons and ligaments is possible in the metacarpus and metatarsus unless marked swelling and multiple injuries make separation impossible.

2. Risk of Injury

There is little information about specific risk factors for tendon injury; however, training regimen, footing, and conformation are thought to play a role in injury. Most tendon injuries are reported in racehorses, but they are diagnosed in all types of performance horses. In recent studies of racing over fences, horses with a valgus conformation, or increased fetlock joint angle, have increased risk for SDFT injury.³ Singer et al.⁴ found an increased risk of SDFT injury in Cours Complete Internationale compared with one-day events.⁴ Elite eventing and show jumping results in increased risk for SDFT injury; show jumping increased the risk of DDFT injury. The risk for hindlimb suspensory desmitis was increased with dressage.⁵ In racehorses, age correlated with increased risk of SDFT injury, which tended to be higher in male horses. Also, injury was more likely to occur in horses train-

NOTES

ing that had not yet raced.^{6,7} SDFT injury in racehorses occurred more often as distance and body weight at race time increased. Horses that had competed in steeplechase races were more likely to injure the SDFT than any other tendon or ligament.⁸ The ability to detect subtle injury is needed, because horses with suspected risk for SDFT or suspensory ligament injury during pre-race examinations were more likely to be injured during racing.⁹ The risk of tendon injury in performance or racehorses during training is unknown.

3. Anatomy and Composition

Tendon consists of water, collagen, and proteoglycan, which make up the ground substance. Additionally, there is a sparse population of cells termed tenocytes. Flexor tendons from growing animals have a high concentration of cartilage oligomeric matrix protein with the highest levels residing in tendons undergoing tension.¹⁰ Tropocollagen is the main end product of collagen type I production and is organized into triple helical overlapping molecules that create a collagen fibril. Covalent crosslinks stabilize these fibrils, which in turn make up tendon fibers. Collagen fibril populations are affected by age with increasing crosslinks with maturation and aging.¹¹ Exercise seems to be necessary for tendon development and remodeling.^{12,13} Excessive and long-term training may adversely affect fibril size.¹⁴

Other components of tendons include proteoglycans, which seem to help retain water, promote collagen fiber size, and therefore, potentially affect tendon fiber strength. Small amounts of type III and type VI collagen are also found in tendon as are fibronectin, hyaluronic acid, thrombospondin 4, and elastin.¹⁵

Tendon structure is altered with different function. Tendon changes occur with aging, and tendon maturation occurs at 2 yr of age.¹⁶ Aging decreases the tendon crimp, decreases the number of fascicles, and increases the thickness of the endotenon. The flexor tendon cross-sectional area increases with age, exercise, and injury.^{12,17-21}

Tenocytes are found in linear alignment along tendon fibers, which are grouped into visible tendon subunits called fascicles.¹⁵ Fascicles are surrounded with connective tissue containing blood vessels, nerves, lymphatics, and elastic fibers (Fig. 1). The connective tissue, termed tenon, surrounds each fiber (endotenon) and fascicle (epitenon) as well as the entire tendon (peritenon). The link of the connective tissue throughout the tendon allows the movement of tendon fibers and fascicles during tendon stretching during loading. Blood vessels course in both longitudinal and transverse planes. Transverse vessels branch from the vasculature in the paratenon and communicate through vessels in the endotenon with the longitudinal vessels. This supplies a fine capillary network in the endotenon around the tendon fascicles.²²

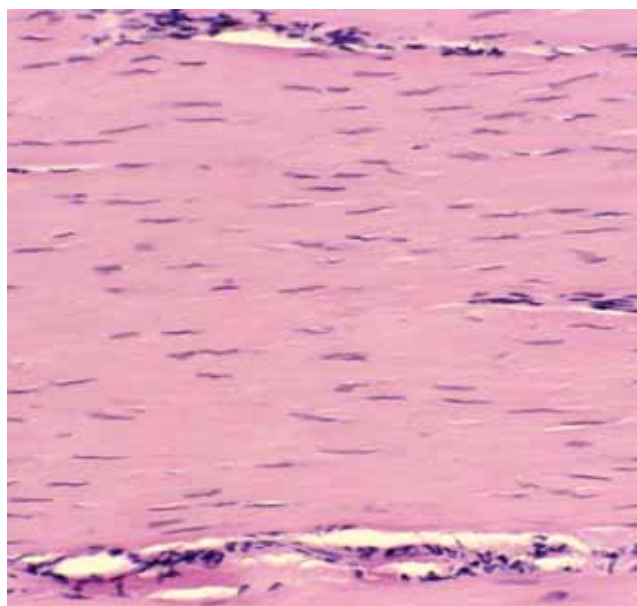


Fig. 1. Photomicrograph of tendon with H&E stain. This micrograph shows normal linear alignment of the collagen fibers with tenocytes. Note the abundance of extracellular matrix and the limited vascularity of this tissue.

Tendon fascicles also have crimp created by altered angles of the fibers within the fascicle. Crimp decreases with age and is reduced in the center of the SDFT. Tendon crimp seems to allow for a small amount of elongation during the initial phase of loading, and it may contribute to tissue elasticity.

4. Injury and Pathology

Flexor tendon injury can be caused by an intratendinous strain with collagen fiber disruption or from extratendinous trauma from compression or laceration. SDFT strain occurs predominately in the metacarpal region, whereas DDFT strain is more often found in the distal metacarpus and tendon sheath. Rear limb tendon strain affects the DDFT more frequently. Original histological description defined three grades, which are similar in severity to the ultrasonographic grades used to describe fiber echogenicity.²²⁻²⁴ Although not yet reported extensively in horses, initial tendon fiber damage creates an inflammatory reaction, which results in edema, closure of capillaries, and induction of catabolic cytokine release.²⁵ Breakdown of fibers is suggested by the increased enzymatic reaction in tendons exposed to cytokines.^{26,27} The increased tumor necrosis factor alpha (TNF α) found in inflamed SDFT suggests that the inflammatory process stimulates proteases, specifically stromelysin.²⁸ Lymph flow is decreased in the injured area of experimentally induced tendonitis, which suggests that vascular compromise is caused by a compartment syndrome with increased intratendinous pressure from the inflammatory response. This is supported by restric-

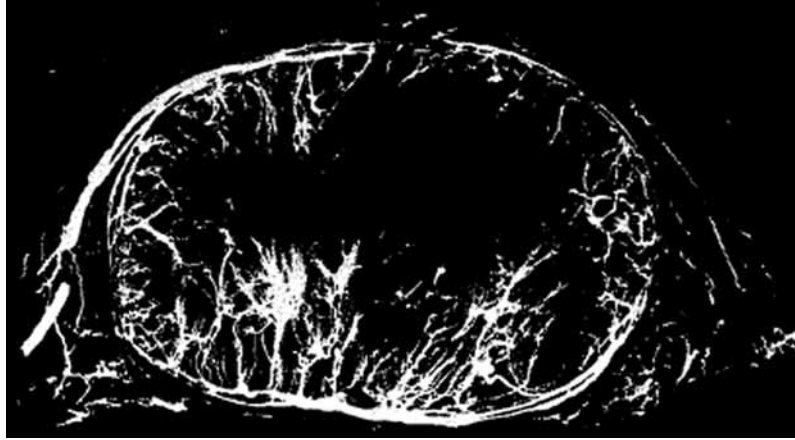


Fig. 2. Cross-sectional microangiogram from an SDFT from a 5-yr-old stallion with a 3-wk-old injury. The central region of the tendon is devoid of open blood vessels, and the vessels around the injury are dilated. Image from Stromberg B and Tufvesson G. Lesions of the superficial flexor tendon in racehorses. A microangiographic and histopathologic study. *Clin Orthop* 1969;62:113–123.

tion shown by microangiography of tendons at different stages of natural injury.²² Leakage of the contrast material into the tendon fascicles suggests vascular damage in the area of injury with resultant decreased blood flow (Fig. 2).²⁹ The region is inflamed with progression of the lesion size during the inflammatory phase of injury, even if exercise has stopped.

Strain of the SDFT creates a lesion in the central core with swelling and reddening of the central fascicles (Fig. 3). This seems related to the increase in core temperature and the initiation of tendon stretch during loading in the central fascicles before loading in the peripheral fascicles.³⁰ There is evidence that cells isolated from the center of the tendon are more resistant to thermal stress than cells from the periphery. Core lesions may also occur because of reduced crimp within the central core of the tendon. In the grade II lesion, the hemorrhage extends to the epitenon and peritenon.²² The affected core lesion is enlarged. In the grade III tendon, the paratenon is thickened, and the overall diameter is markedly enlarged (Fig. 4). There is no filling of the vasculature in this lesion.^{22,29} The regions of injury are devoid of tenocytes and remain so during the maturation phase of healing.

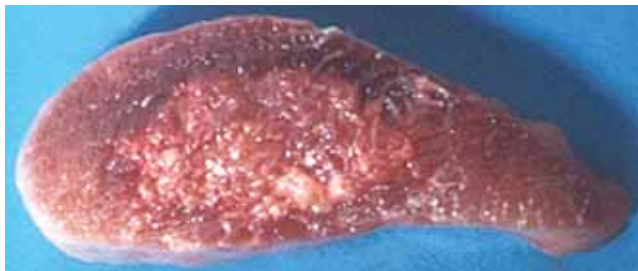


Fig. 3. Cross section of an injured SDFT with disorganized granulation tissue in the central region of the tendon.

In subacute and chronic cases, the number of vessels increases in the area of injury along with mesenchymal cells and fibroblasts that result in granulation tissue (Fig. 5). As scar tissue is formed, vessel number decreases in the central core, and vessels around the region of scar do not course in the normal fascicular pattern.²²

There are few reports on the sequence of healing of equine flexor tendons. Most of the reports deal with clinical cases, which have no histology or necropsies from tendons that are past the acute phase of healing. Both natural and experimental SDFT

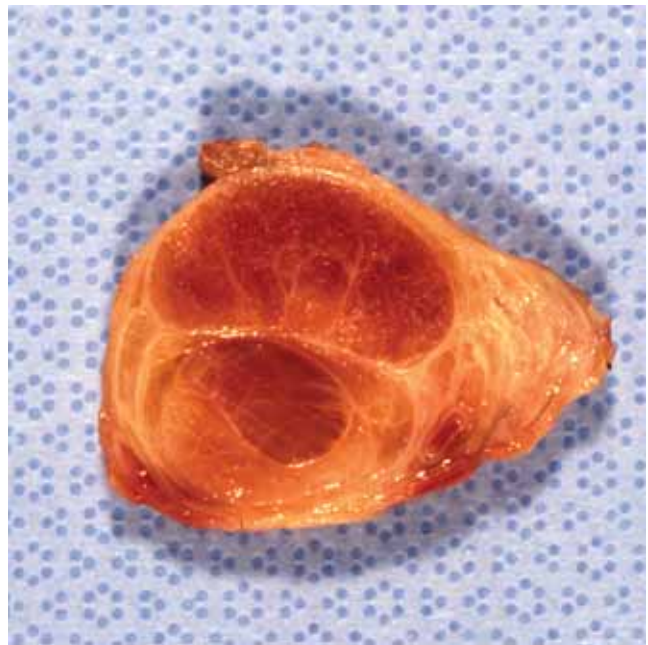


Fig. 4. Cross section of the SDFT, DDFT, and check ligament. There is marked enlargement of the SDFT caused by an acute grade III lesion with loss of the fascicle pattern.

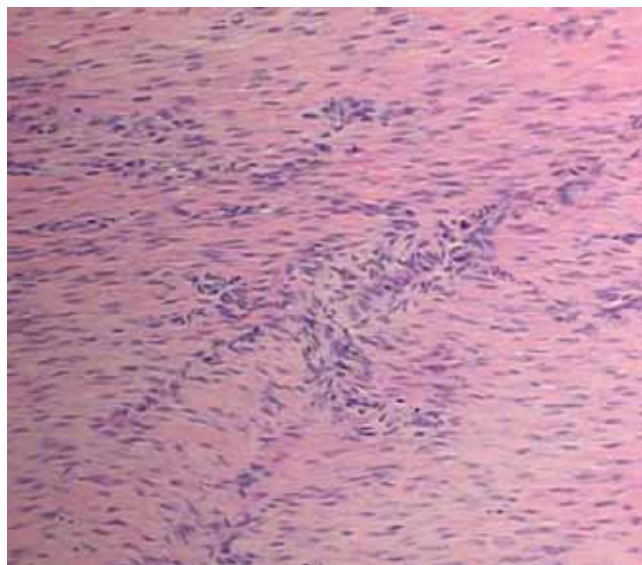


Fig. 5. Photomicrograph of SDFT with tendonitis from a strain (H&E stain). This tendon has focal areas of hypercellularity and increased vascularity in the early stage of healing.

injuries heal by fibroplasia. Tendon cross-sectional area is usually increased, and there is an increased cell-to-matrix ratio. Additionally, increased amounts of type III collagen compared with normal tendon are present.^{31,32} Even after complete healing, biomechanical properties are not thought to return to normal. Although new tendon fibers are formed, scar is formed between fibers and fascicles that limit fiber and fascicle movement. The lack of normal elasticity in the scarred portion of the tendon hypothetically creates a site for future injury during tendon loading.

5. Tenosynovitis

Tenosynovitis can be caused by injury to tendons within the sheath. The DDFT is more often injured within the digital tendon sheath just at and just proximal to the fetlock or in the tarsal sheath over the calcaneus. Core lesions can be eccentric and are most prevalent just palmar/plantar to the fetlock joint. Lesions may consist of internal core lesions but can develop as tears or splits on the side of the tendon (Fig. 6).³³⁻³⁶ If the DDFT is enlarged, impingement within the digital sheath can cause erosion of the tendon surface. SDFT injuries can affect the manica flexoria or the tendon branches distal to the fetlock. Excess sheath effusion and lameness are the most typical signs. Ultrasonography can identify tendon injuries, but tears of the DDFT or the manica flexoria may not be detectable during weight bearing.^{36,37} A definitive diagnosis is made with tenoscopy.³⁵ The prognosis for tears in the tendons that are exposed within the sheath is poor without surgery.

Increased size of the DDFT within the digital sheath can create a constriction by the annular ligament, and this can result in tenosynovitis and lame-

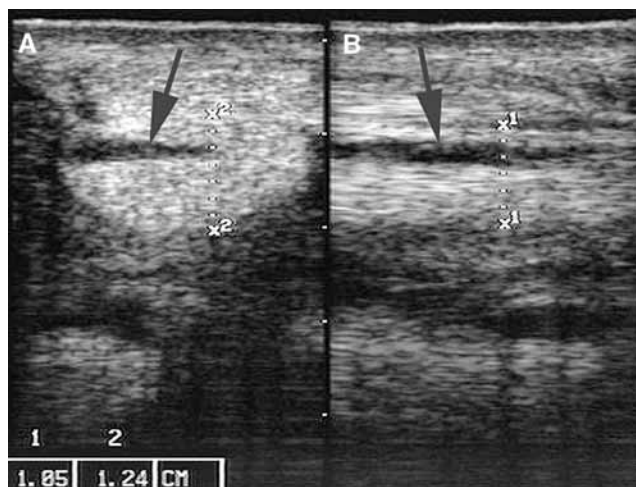


Fig. 6. (A) Ultrasonograph of a linear tear in the DDFT that was open and communicating with the synovial fluid within the digital sheath. (B) Photograph of the DDFT 8 mo after the ultrasonograph in A. After surgery to remove the frayed tendon fibers, healing was incomplete, and tenosynovitis persisted because of the incomplete healing of the DDFT.

ness.^{34,38} Primary annular ligament desmitis can appear similar to tenosynovitis, because they both show annular ligament constriction.³⁹ Constriction of the annular ligament caused by DDFT injury often requires severing of the annular ligament, whereas annular ligament desmitis can heal with rest, local injection of corticosteroids, or splitting of the annular ligament.³⁹ Response to resection of the annular ligament is dependent on the severity of the injury to the DDFT. If the tendon has been lacerated and adhesions occur during healing, the response to surgery is guarded.

Septic tenosynovitis of the digital sheath is most often caused by punctures or laceration of the tendon sheath and frequently involves one or both the



Fig. 7. Ultrasonograph of a septic digital tendon sheath. There are excess fluid and fibrin strands around the DDFT.

SDFT and DDFT. Early recognition and treatment of the infection (within 24 h) is associated with a better prognosis (Fig. 7).⁴⁰ Numerous treatments have been used to help resolve sepsis, including systemic antibiotics, lavage, local and constant infusion of antibiotics, regional limb perfusion with antibiotics, tenoscopic removal of fibrin, and open drainage. Tenoscopic evaluation with lavage of the tendon sheath, although helpful to identify tendon injuries and remove fibrin, did not improve the outcome compared with lavage or open drainage.^{40,41} The presence of pannus within the sheath significantly lowers the chance for return to previous use.⁴⁰ Resection of the proximal annular ligament may be of help in resolving the pain after resolution of the infection.

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