Tendon physiology and repair

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Abstract

Tendon disorders are common and account for a significant proportion of musculoskeletal pain presentations. Tendon pathology can occur in all ages leading to pain and disability that can substantially impact daily function, recreational activities, occupation and quality of life. An understanding of the complex tendon macro- and microscopic structure, along with its physiology and biomechanics provides the basic rationale for its medical or surgical treatments following injury. There are numerous described techniques for surgical repair of ruptured flexor tendons, and therefore it is important to understand the core principles for a successful repair which will be summarized in this article.

Keywords biomechanics; primary repair; tendinopathy; tendon; tendon healing; tendon injury; tendon physiology; tendon structure

Introduction

Tendon is a dense connective tissue that connects muscle to bone. It has an important role in producing movement, maintaining posture and stability of joints by allowing transmission of muscle contraction forces to the skeleton. The specific composition, organization and interaction of its components are crucial to create a strong force-transmitting unit. The specific mechanosensitive properties of tendons allow it to respond and adapt to mechanical loading, which is important in tendon development, homeostasis and repair. The following sections will entail tendon macro and microanatomy, physiology, pathophysiology and also surgical repair.

Tendon anatomy and physiology

The tendon composition

Tendon is composed of tendon-specific cells embedded within a complex network of extracellular matrix. The extracellular matrix (ECM) makes up about 80% of the tendon structure and is the non-cellular component that consists mostly of water, proteoglycans, collagen and other non-collagenous proteins (e.g. elastin, fibronectin, integrins). The ground substance surrounding collagen fibres acts as a medium for nutrition diffusion; and has a role in the extracellular assembly of collagen. Collagen is the most abundant component of tendons, making up 60–85% of its dry weight (Figure 1). Type I collagen is the most predominant collage type (≈95%), with the next most abundant (3%) being type III collagen. Type V, XI, XII and XIV collagens are also reported to be found in small levels. Collagen resists tension but also has a certain degree of compliance.[1]

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The ground substance surrounding collagen consists of proteoglycans, glycoproteins and glycosaminoglycans, which bind well with water. This proteoglycan–water matrix gives the tendon its viscoelastic properties to resist compressive and to a lesser extent tensile forces. A proteoglycan consists of a protein core that is covalently bonded to one or more chains of glycosaminoglycans (Figure 2). A variety of proteoglycans are found in tendons, broadly divided into large aggregating proteoglycans, and small leucine-rich proteoglycans (SLRP). Large aggregating proteoglycans such as aggrecan and versican increase water content and are located at specific sites of the tendon to resist compression. SLRPs are the most predominant type of proteoglycans in tendons. SLRPs have overlapping functions in the extracellular assembly of collagen fibrils, and the fine-tuning of their alignment, diameter and stability.[1] Examples of SLRPs are Decorin, biglycan, fibromodulin and lumican.

Elastin is a glycoprotein responsible for the elastic flexible property of tendons allowing it to elongate and recoil. It accounts for 1–2% of the dry weight of tendon. Compared to other structures such as ligaments, the concentration level of elastin in tendon is relatively low. This is so that the tendon does not stretch excessively, and thereby can effectively transfer force into movement. Fibronectin are glycoproteins that have a role in cell-to-cell, and cell-to-matrix adhesions, and cell migration. It is also involved in the organization of collagen fibrils into bundles. There are numerous other non-collagenous proteins within the ECM (e.g. lubricin, tenomodulin, collagen oligomeric matrix component (COMP), tenascin-C) and their exact functions and interactions are beyond the scope of this summary article.[1]

The cellular component of the tendon structure consists mainly of tenocytes and tenoblasts (95%), which are specialized fibroblast-like cells that produce collagen. The remaining cells are synovial cells (in tendon sheaths), chondrocytes (at the enthesis), endothelial cells and smooth muscle cells (found in vessels). Tendon stem cells have also been demonstrated in tendon cell cultures capable of clonogenicity and self-renewal.

Tenoblasts are the immature precursors of tenocytes. Tenoblasts are spindle-shaped with a high level of metabolic activity. As they mature to become tenocytes, their shape becomes flat and elongated, their metabolism rate decreases and their energy production switches from aerobic to a predominantly anaerobic metabolic pathway. Tenocytes and tenoblasts are arranged longitudinally in rows within the endotenon. With age, the cell–matrix ratio decreases, and elongated cellular projections in tenocytes continue to allow cell-to-cell and cell-to-matrix contact. Together they are responsible for collagen synthesis and homeostasis of the extracellular matrix (production and degradation of ECM components). The low metabolic rate and well-developed anaerobic capability allow tendons to maintain tension for long periods without ischaemia. However, it is also for this reasons that the healing rate in tendons is relatively slow following injury.

Tendon homeostasis

The ECM is constantly degraded by enzymes (e.g. collagenases, proteoglycanases) and remodelled by tenocytes to adapt to mechanical loading patterns. The turnover of the ground substance is much higher than the collagenous component. Together, the ECM and cells interact to regulate the highly organized tendon

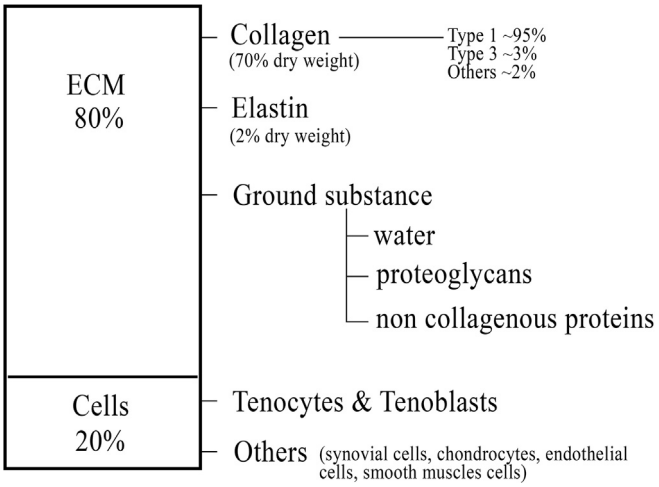

Figure 1 Extracellular and cellular content of tendon.

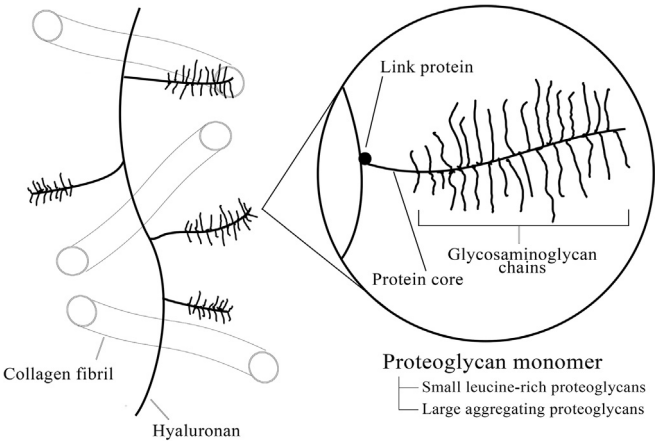

Figure 2 Simplified diagram of proteoglycan within the extracellular matrix. Proteoglycans can interact with collagen fibrils. Different glycosaminoglycan attachments provide different interactions with extracellular matrix (ECM) components, thus directs its function and ECM properties.

microstructure. These interactions occur through cell gap junctions and via a number of biological mediators such as cytokines, growth factors and transcription factors. Growth factors: transforming growth factor (TGF)-β and fibroblast growth factors (FGFs) and transcription factors: Scleraxis, Mohawk and early growth response 1 (EGR1) are known to promote collagen synthesis.[2]

Mechanical loading is important in the maintenance of ECM. Mechanical stimuli (i.e. collagen fibre tension) are converted into biochemical signals that create a cellular response. This process of mechanotransduction is mediated by integrins, which are mechanosensitive proteins that bind onto cell membranes. Loading stimulates collagen and ECM synthesis, resulting in increased cross-sectional area and tendon stiffness. Unloading has the opposite effect but may not change the cross-sectional area.[3] Hence, prolonged limb immobilization following injury can have negative effects on tendon healing and its tensile strength.

Tendon structure

The tendon unit is a hierarchical structure containing collagen organized into microfibrils, fibrils, fibres and fascicles (Figure 3). Three chains of collagen molecules are twisted to form a helical tropocollagen molecule. Five tropocollagen molecules assemble into microfibril, which then cross-links with each other in a parallel and staggered arrangement to form a collagen fibril. Multiple fibrils make up a collagen fibre. Several collagen fibres are then bundled together into a fascicle. Each fascicle would contain tenocytes alongside bundles of collagen fibres and ECM making this the basic functional unit of a tendon. The endotenon is a loose connective tissue that surrounds the fascicle and facilitates small sliding motions between each fascicle. Fascicles running in parallel form further bundles which are enveloped by the epitenon, forming a tendon unit.

Collagen fibres in tendons tend to be highly aligned (parallel to the long axis of the tendon) compared to collagen found in other parts of the body, but the structural configuration can vary depending on the attached muscle function. Tendons that transmit force over long distances tend to have more aligned collagen structures (e.g. digital flexor tendons). Whereas shorter spanning tendons, such as rotator cuff tendons can display a wider collagen structural arrangement.

The paratenon and tendon sheath

A tendon can be surrounded by a bed of loose areolar tissue called paratenon (extrasynovial tendons), or it can reside within a true tendon sheath of dense fibrous tissue (intrasynovial tendons) (Figure 4). These sleeves serve to protect the tendon and also allow sliding movements. Tendon sheaths are found in areas of the body where tendons are exposed to high friction, for example, flexor tendons in the hand. In areas where friction is less, the tendon is surrounded by paratenon only (e.g. Achilles tendon). Tendon sheaths have an outer parietal layer and an inner visceral surface which are both in continuity with each other, but separated by synovial fluid. The visceral layer is essentially the epitenon, surrounded by synovial cells which produce synovial fluid. In non-sheathed tendons, fluid may also be found between the paratenon and epitenon, enabling sliding movement of the tendon with reduced friction. Synovial fluid circulation plays an important role in nutrition delivery.

Blood supply and innervation

Tendons are relatively avascular, hence their white appearance. Blood vessels, lymphatics and nerves travel within the paratenon or synovial sheath, supplying the superficial peritendinous portion of the tendon. These then branch deeper intratendinously and penetrate the epitenon and endotenon creating an anastomotic vascular network. Within the endotenon, these small thin-walled vessels run parallel to the fascicles forming anastomoses with each other. In tendons covered by tendon sheath, supplying vessels on the parietal layer enter the tendon's epitenon (visceral layer) through a delicate connective tissue sheath called mesotenon (Figure 4). Mesotenon can take the shape of a cord-like vinculae; as observed in the digits, or sheet-like folds found at the ankle and wrist. Intrasynovial tendons are poorly vascularized and mainly rely on synovial fluid nutrition.

Major blood vessels arrive at the tendons at the musculotendinous and osteotendinous junctions. At the musculotendinous junction, vessels from the muscle and surrounding tissue continue along the paratenon and epitenon but often do not extend more than the proximal third of the tendon. Blood

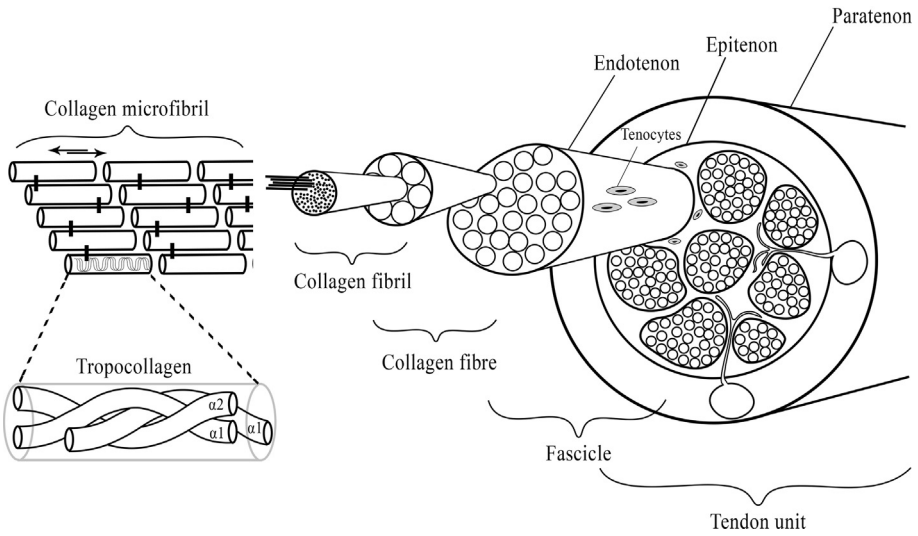

Figure 3 Schematic of the hierarchical tendon structure.

supply from the periosteum at the osteotendinous junction is sparse and is mostly limited to the tendon's insertion site. An area of hypovascularity can occur at the junctional zone between the proximal and distal vascular supply. Such watershed area has been demonstrated in the Achilles tendon 2–7 cm proximal to the tendon insertion.[4] Similarly, areas of hypovascularity can be found at areas of compression, torsion and friction. Such an example is the dorsal surface of the flexor digitorum profundus tendon adjacent to the volar plate. Another example is the supraspinatus tendon, which is prone to mechanical irritation. Some tendons could have multiple areas of hypovascularity from multiple areas of bony compression (e.g. fibularis longus tendon) or by segmented vascular supply (e.g. digital flexor tendons). Reduced blood supply can place these watershed areas at risk of degeneration, tears and rupture.[4]

Similar to vessels, nerve fibres form rich plexuses in the paratenon which branch and enter into the epitenon and endotenon septa. Tendon nerve supply is mainly afferent, to provide information to the central nervous system on joint position (proprioception), pain (nociception) and pressure. Myelinated nerve fibres terminate at the tendon by a network of terminal nerve endings that intertwine with the tendon fascicles. Specialized mechanoreceptors called Golgi tendon organs encapsulate the end of myelinated nerves, and functions to detect changes in tension and pressure. These mechanoreceptors are most abundant at musculotendinous junctions, and help regulate muscle force of contraction. Other mechanoreceptors such as Ruffini corpuscles and Pacinian corpuscles are also found in tendons and contribute to proprioception. Free unmyelinated nerve endings serve as nociceptors for pain (mostly at entheses).

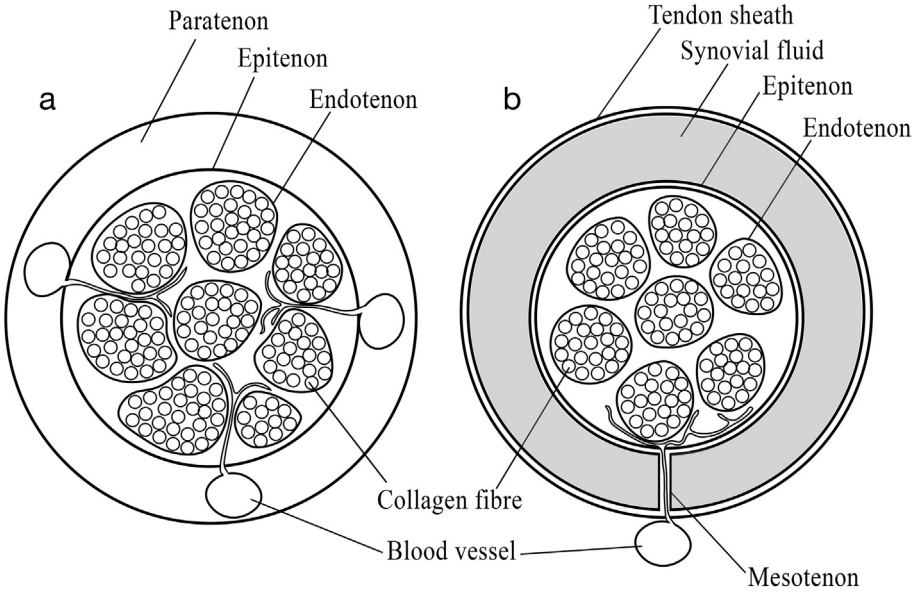

Figure 4 Schematic of the cross-section of a tendon. (**a**) paratenon-covered tendon; (**b**) tendon sheath-covered tendon.

Sympathetic and parasympathetic nerve fibres are also found in tendons and are thought to regulate blood flow.[4]

The musculotendinous junction

The musculotendinous junction is the transitional area between muscle and tendon. It is an area subject to the greatest strain during muscle contraction. Muscle tears often occur in this region due to its inferior mechanical strength compared to tendon.[5] Tendon ECM interdigitates with folds of muscle cells membrane, with collagen fibres anchored onto muscle fibres in the direction of stress. This area of the tendon contains numerous Golgi tendon bodies and nerve endings, and is most receptive to mechanical change.

The osteotendinous junction/enthesis

Tendon inserts into bone via a specialized interface called the enthesis. The enthesis can insert into bone via fibrous tissue or by a layer of fibrocartilage. Fibrous entheses connect tendon to bone or periosteum via dense fibrous tissue. These insertions commonly occur over large surface areas, typically at the metaphysis or diaphysis of long bones. Such examples are the deltoid tendons inserting into the humerus, and the adductor magnus attaching to the linea aspera of the femur. Histologically, these insertions are characterized by perforating mineralized collagen fibres known as Sharpey's fibres, which arise from tendon or periosteum and anchor directly into lamellar bone. The insertion angle only changes slightly during motion.

Fibrocartilaginous entheses are more common, and connect tendon to bone via fibrocartilage, typically at epiphyses and apophyses. Such examples are the Achilles tendon and rotator cuff tendons. Histologically, there are four distinct zones with a gradual transition from dense fibrous tendon tissue, to uncalcified fibrocartilage, mineralized fibrocartilage, and then lamellar bone. Each zone consists of varying compositions and functions (Figure 5). The tidemark is the demarcation between uncalcified and calcified fibrocartilage. This area of high-stress concentration is prone to injury because of the difference in material properties of the soft and hard tissue. Overuse injuries are more common in fibrocartilaginous entheses since their insertion angle changes greatly with motion. Similar to the tendon proper, mechanical loading also influence the composition and organisation of enthesis components and thus its mechanical properties.[2]

Tendon function and biomechanics

In the limbs, tendon functions to transmit force and resist tension during muscle contraction to allow movement. It is flexible yet relatively inelastic, and can resist tensile, and to a lesser extent compressive forces. The tensile strength of a tendon is related to its thickness and collagen content. The cross-sectional area has an impact on the average stress placed on the tendon as a given force (N/m2).[3] The tendo Achilles is one of the largest and strongest tendons in the body and can withstand loads up to 12.5 times the body weight.[4]

Tensile forces cause collagen molecules to stretch, whilst shear forces cause them to slide between one another. The ability for collagen molecules, fibrils and fascicles to slide independently against one at each level another allows the tendon to change shape and transmit forces during muscle contraction.[1]

The stress–strain curve can be used to illustrate the mechanical properties of tendons (Figure 6). At rest, the collagen fibrils and fibres display a 'crimped' configuration, which straightens under initial tension (toe region). As the fibrils are stretched and become more parallel due to molecular sliding, the tendon is deformed in a linear fashion (elastic region). This region represents the Young's modulus (i.e. stiffness, or ability to stretch). When unloaded, the collagen fibrils return to their initial configuration without damage, but only up to around 4% of strain. Beyond this yield point, microscopic failure occurs as gaps between collagen molecules increases, and at 8% of strain, breakdown of cross-links and slippage between adjacent

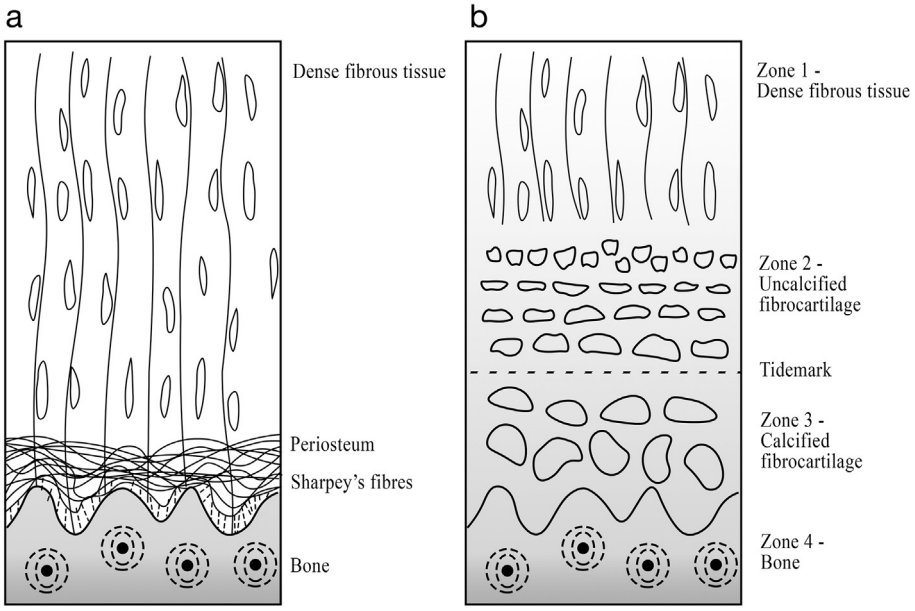

Figure 5 Schematics showing: (**a**) fibrous enthesis; (**b**) Fibrocartilaginous enthesis. Fibrous enthesis attaches onto periosteum and into bone via Sharpey's fibres. Fibrocartilaginous enthesis has a graduated transition of four zones. Zone 1 is dense fibrous tissue that contains tendon fibroblasts with linearly arranged collagen. Zones 2 and 3 contain fibrochondrocytes with increasing mineralization and irregular collagen arrangements. Zone 4 is bone containing osteocytes and mainly type 1 collagen.

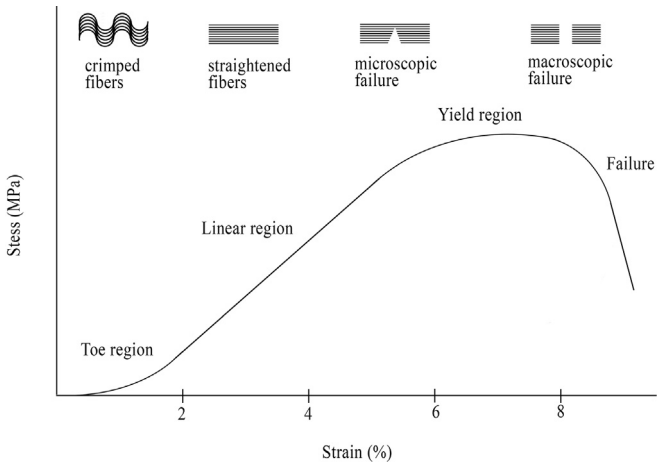

Figure 6 Stress–strain curve demonstrating the mechanical properties of tendons.

molecules leads to macroscopic failure.[4] Following complete failure, the ruptured ends of collagen fibres recoil into a tangle. Collagen alignment therefore is crucial in the physiological function of tendons.

Tendons also have viscoelastic properties largely due to proteoglycan and water content in the ECM. Therefore tendons also display time and rate dependent mechanical characteristics of creep (increasing deformation under constant load), stress relaxation (reduction of stress under constant deformation), and hysteresis (energy loss between loading and unloading).[4]

Tendons can be classified as positional tendons or energy-storing tendons. Positional tendons have higher collagen content and are relatively stiff (less length change) to enable precision in movement (e.g. digital flexor and extensors in the hand). Energy-storing tendons are less stiff, and can store and release energy by the means of elastic recoil thereby increase efficiency of movement. The amount of energy dissipation (as heat) following storage (i.e. hysteresis) negatively affects the efficiency of motion. For example, the Achilles tendon increases efficiency in movements like jumping and running, by storing energy during deceleration and then releasing it to power acceleration. Energy-storing tendons have to be highly fatigue resistant to withstand large repetitive forces.[1]

Although composed of the same basic materials, the specific functional demand and mechanical properties of tendons are determined by the various shapes, sizes and underlying structural compositions. The balance between stiffness and elasticity enables tendons to perform specific functions in different regions of the body, but can be altered as a result of injury and degeneration.

Tendon injury

There is a wide range of injury mechanisms leading to the disruption of the highly organized collagen structure in tendon ultimately causing dysfunction. Tendon injury can be acute or chronic, and secondary to extrinsic (abnormal or excessive loading, laceration) or intrinsic factors (e.g. age-related degeneration, genetic diseases affecting collagen synthesis such as Marfan's syndrome) either alone or in combination. There are several terminologies used for tendon injury. Tendinitis is characterized by inflammation, whereas tendinosis is characterized by degenerative changes without inflammation. The term tendinopathy is used clinically to describe pain and reduced tendon function, often in the chronic degenerative context.[4]

Acute ruptures in otherwise healthy tendon are predominantly driven by extrinsic factors, often secondary to overloading by excessive force beyond physiological limits (ultimate tensile strength) of the tendon, or by direct laceration on the tendon (e.g. from knife injury, fracture, implants). These injuries can be partial tears or complete ruptures. Most tensile overloading cause tears at the musculotendinous junction or the enthesis as these are areas of mechanical weakness. Mid-substance tears would require much greater force to occur due to the high tensile strength of normal tendon tissue.[5] Spontaneous tendon ruptures are often multifactorial and occurs from acute stresses in an already degenerate tendon. Tissue biopsies of Achilles rupture have shown that many have underlying degenerative changes already present.[6] Most common tendons that rupture from excessive mechanical load include the Achilles, patella, quadriceps and distal biceps tendon.

Degenerative tendinopathy is thought to be a result of repetitive loading causing cumulative microtrauma, which fails to heal. Microscopically, there are features of failed healing process with collagen fibril thinning and disorganization, increased non-collagenous content in the ECM, neovascularization and hypercellularity.[2] Over time, the ratio of type I to type III collagen decreases. Degenerated fibres can also be replaced by calcium or lipids (tendolipomatosis). Macroscopically, degenerative tendon appears grey-brown and loses its white glistening appearance. In addition, nodular, diffuse or fusiform thickening of the tendon can occur. Under the ultrasound, disorganized fibres cause multiple shadows and reflections displayed as hypoechoic areas. Infiltration of new blood vessels may also be observed on Doppler ultrasound. Clinically, degenerative tendons could remain asymptomatic. For example, asymptomatic rotator cuff tears are prevalent in the general elderly population.

The pathogenesis of degenerative tendinopathy is poorly understood. Theories suggest that hypovascularity, neutrally mediated mast cell degranulation and exercise-induced localized hyperthermia may contribute to the poor healing response of tendon damaged from microtrauma.[7] Whilst tendinopathy is previously understood to be a non-inflammatory degenerative process, recent studies have reported the presence of inflammatory cells and markers in tendinopathic tendons. It is suggested that poor resolution of inflammation may contribute to chronic tendon damage leading to degeneration. However, the heterogeneity of literature means that no conclusive remarks could be made on the inflammatory pathogenesis of tendinopathy nor explains why some patients do not respond to anti-inflammatory treatments.[8]

There are various intrinsic factors that predispose to tendinopathy and rupture. These include increasing age, gender, obesity, previous tendon injury, drugs, inflammatory arthropathies, as well as certain endocrine and genetic conditions. In diabetes, glycation of collagen negatively affects tendon biology and its mechanical function. Crystal arthroplasties such as gout and rheumatoid arthritis can deposit crystals in the tendon causing low-grade inflammation leading to degeneration. Obesity

increases the load on tendons and has a strong association in particular to lower limb tendinopathies. Tenocytes are sensitive to hormonal imbalance and as such, thyroid disorders have also been linked to tendinopathies. Some pharmacological substances can also alter tendon properties and lead to iatrogenic tendinopathy. Such known examples include corticosteroids, quinolone antibiotics, aromatase inhibitors and statins.[9]

Tendon healing

Tendons respond to injury by peritendinous inflammation and degeneration of ECM, with active repair of the ECM induced by tendon fibroblasts. Tendon healing typically follows three overlapping phases – inflammation, proliferation and remodelling.

The inflammatory phase occurs immediately following injury. Haematoma activates a cascade of vasodilators and proinflammatory mediators, which increased vascular permeability leading to an influx of inflammatory cells and platelets to the injury site. These cells release growth factors and cytokines which cause migration of macrophages. M1 macrophages engage in phagocytosis of necrotic debris and blood clot. They also release factors that stimulate angiogenesis and proliferation of resident tenocytes, to generate materials (e.g. water, glycosaminoglycan, type III collagen) for new ECM formation.[6] Tendon repaired with sutures at this phase is reliant on the suture strength.

A few days following injury, the proliferation phase is characterized by the deposition of disorganized granulation tissue. Tenocytes continues to be recruited and now predominate over other inflammatory cells, enabling abundant ECM synthesis. The newly laid ECM components such as type III collagen and proteoglycans are arranged in a random manner. This disorganized ECM bridges the tendon gap but is relatively weak. This is the period when the tendon is at its weakest, at 2–4 weeks following surgical repair and prone to re-rupture.

The remodelling phase commences approximately after 6 weeks from injury, and contains two sub-stages: consolidation and maturation. During consolidation, cellular content and ECM synthesis are reduced. The tissue becomes more fibrous as type III collagen are converted into type I. Collagen fibres begin to realign in the direction of stress, thus restoring some of its tensile strength. The maturation stage occurs after around 10 weeks, and is characterized by increased collagen fibril cross-linking and a gradual transition of fibrous to the final scar-like tendon tissue. This process may last over years, but the resulting tensile strength of a healed tendon remains inferior to that of its original uninjured state.[7]

Intrinsic versus extrinsic healing: cell recruitment for tendon healing can occur by two mechanisms – intrinsic and extrinsic. Intrinsic healing is via migration and proliferation of resident tenocytes within the endotenon and epitenon, which produces collagen and reorganises fibre alignment. Extrinsic healing on the other hand is via invasion of inflammatory cells from surrounding paratenon or synovial sheath, which results in adhesion formation between the sheath and tendon surface.[5] Which healing mechanism predominates depends on the anatomical location, type of injury, stage of healing and the amount of tendon movement during healing. Extrinsic healing occurs during early stages of healing, but also predominates following immobilization of injured/repaired tendons. Immobilized tendon shows greater inflammatory gene expression and adhesion formation which reduces tendon gliding. Tendon glide is important for synovial circulation which encourages nutrient delivery for healing. Naturally, the density of adhesion fibres and tensile strength decreases over time with cell apoptosis, allowing tendon motion and subsequent adhesion breakdown. Early active mobilization has been shown to reduce adhesion formation and encourage its breakdown process. Although excessive scar formation can limit tendon gliding, deficient scarring may lead to premature rupture. A fine balance is therefore required for optimal tendon healing and function.

Apart from adhesion formation which can limit tendon gliding and subsequent joint movement, there are several factors influencing tendon healing. The healing potential of a tendon depends on the severity of injury, its anatomical location and its local environment. Intrasynovial tendons (e.g. flexor tendons) do not heal well spontaneously compared to extrasynovial tendons (e.g. extensor tendons).[6] Patients intrinsic factors such as malnutrition, smoking, poor compliance, steroid use can negatively affect healing. In addition, surgical factors such as timing of repair, techniques and materials used for repair and surgical site infection can all influence healing potential.[5]

Surgical repair of tendon injuries

Tendon injuries can be managed with non-operative or operative treatment depending on the location, acuity and severity of injury. Acute complete tendon ruptures tend to require primary surgical repair with the aims to remove damaged tissue, restore tendon continuity and provide a strong and secure enough repair that allow early function with minimal adhesions. Primary repairs should be done as early as possible following acute tendon injury. A primary repair is done within 24 hours, whilst a delayed primary repair is done within 14 days.[5] Opposable midsubstance ruptures can be repaired with end-to-end suture techniques, whilst ruptures at the enthesis may be repaired transosseously and/or with suture anchors. An exception to early surgical repair is acute Achilles tendon ruptures, where there has been growing evidence to support non-operative management with accelerated functional rehabilitation.[10] Chronic tendon ruptures or those with large defects may require additional augmentation, tendon/tissue transfer or reconstruction with autografts, synthetic grafts or allografts. Staged reconstruction may be required in cases where primary grafting is not suitable. The following sections will focus on the core principles of primary suture repairs with reference to the digital flexor tendon. For tendon repair in other regions, please refer to the 'Further reading' section.

Primary suture repair principles

As described by Strickland, an ideal primary tendon repair should have sutures placed easily within the tendon, tied securely, with minimal interference with the tendon vascularity. The repair site should have smooth tendon ends with minimal gapping. The repair strength should be strong enough to permit early motion throughout the healing process.[11] Several factors influence the repair strength. These include the choice of suture material and size, as well as surgical techniques: the number of sutures crossing the repair site within the tendon (core sutures), the use of peripheral

epitendinous sutures, suture purchase and the use of locking suture configuration.

The ideal suture material for tendon repair should be sufficiently strong, non-extensible, be easy to handle/knot, and not cause tissue reaction. Stainless steel fulfils these characteristics but have poor handling properties. Commonly used non-absorbable sutures for tendon repair in the UK include Ethibond, Prolene, Ticron and Fiberwire. Ethibond (Ethicon) and Ticron (Tyco Healthcare) are coated braided polyesters. Prolene (Ethicon) is a monofilament polypropylene. Fiberwire (Arthrex) is an ultra-high-molecular-weight (UHMW) polyethylene core covered with a braided polyester jacket. Fiberwire has similar ultimate force to stainless steel, but with better handling properties. Bulky knots created with Fiberwire however is a disadvantage for its use, particularly in smaller tendons. Ethibond is the next strongest, and unlike nylon and polypropylene sutures, its mechanical properties does not deteriorate significantly in body temperatures. Ethibond, therefore thus far has the best desired all-round biomechanical properties as a core-suture in digital tendon repairs. Whilst monofilament sutures such as Prolene and nylon generate less friction and tendon deformity compared to braided sutures, it would be a suitable suture for the epitendinous repair.[12] Absorbable sutures such as polydioxanone (PDS) can be used but may lack the sufficient tensile strength sustained long enough for the whole healing process. In addition, they increased tissue reaction which leads to undesired adhesion formation. Larger calibre sutures are stronger but create bulky suture knots, cause more friction and reduce tendon gliding. In general, a 3—0 or 4—0 core suture, along with a 5—0 or 6—0 epitendinous suture would be suitable for flexor tendon repair in the hand.[12]

The number of core sutures crossing the repair site is proportional to the strength of repair. However, multi-strand repairs are technically difficult and have the disadvantage of added tendon bulk, restricted gliding, and potential uneven suture tension and risk of compromising tendon vascularity.[13] In general, four to six core suture strands are considered adequate.[12] There are numerous variations of suture techniques described for surgical tendon repair. These include but not exclusively Kessler, modified Kessler, Lin locking, Savage, Becker, Tajima, and epitenon suture techniques. Commonly used four-strand repairs are the Cruciate and Adelaide; a two-strand repair is the Modified Kessler (Figure 7). Readers should refer to the original articles for each specific surgical technique.

Peripheral epitendinous sutures add up to 10—50% additional strength to the core suture repair, and importantly reduce gapping between tendon ends. Gapping of greater than 3 mm at repair sites creates suboptimal healing with adhesions and reduced tendon excursion, which ultimately becomes the weakest part of the tendon prone to re-rupture.[13] The horizontal mattress and running-lock epitendinous suture are shown to be the strongest of peripheral suture configurations. Increasing the number of suture passes, taking deeper bites, and purchasing the tendon further away (at least 2 mm) from the repair site can also improve the strength of peripheral repair.[13]

Compared to non-grasping and grasping configurations, locking suture configuration improves the ultimate force and gapping resistance of the repair.[13] This effect is produced by the suture loop tightening around a cross-section of the tendon when axial tension is applied, which utilises the inherent strength of the intact tendon fibres. This also reduces the risk of suture cutting out of the tendon. In a true locking suture configuration, the transverse sutures cross superficial to the longitudinal sutures. Increasing the size of the locking loop further increases the repair strength.

Due to softening of the lacerated tendon ends during initial healing, a larger purchase further away from the repair site can improve suture grip and increase repair strength. For modified Kessler and circle-locking repair methods, the optimal distance for core suture purchase is between 7 mm and 10 mm. A purchase distance less than 4 mm significantly weakens the repair, whilst a distance greater than 10 mm has not shown to increase repair strength, and therefore deemed unnecessary.[14]

Limited skin incisions, meticulous tissue handling, sharp dissections and careful knot placement can all reduce adhesion formation. Furthermore, wide-awake local anaesthetic no tourniquet (WALANT) techniques in hand surgery have rapidly gained popularity as they allow intraoperative assessment of active tendon glide and identification of any gapping, thus enabling the surgeon to tailor the repair accordingly.

Rehabilitation

Rehabilitation following primary tendon repair must be balanced between the risks of rupture and adhesion stiffness. Early mobilization has been shown to reduce adhesion formation, improve tensile strength and range of movement. However, if the

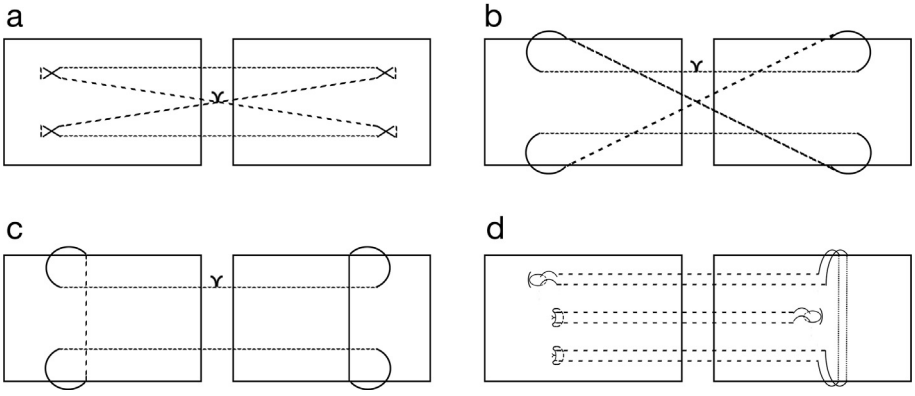

Figure 7 Examples of core suture repair techniques: (**a**) Adelaide repair; (**b**) Cruciate repair; (**c**) Modified Kessler repair; (**d**) M-Tang six-strand repair.

repair strength is inadequate, loading can lead to gapping and risk of rupture.[13] The tendon repair is weakest and prone to rupture 1–3 weeks following surgery repair, and therefore early movements must be well controlled, ideally under supervision by trained therapists. Improved surgical techniques and materials have enabled stronger repairs that can withstand early active movement. There is much debate on the optimal timings, intensity and movement range during rehabilitation and therefore numerous protocols are available. Generally, the rehabilitation process entails the steps of brief immobilization, early controlled passive and/or active mobilization. Although there is a general trend towards true early active motion regimens following flexor tendon repairs, the recent 2021 Cochrane review concludes that there is still not sufficient high-level evidence to support its superiority.[15] Nonetheless, postoperative pain, swelling and stiffness, compliance and level of instructions can limit the patient's ability to perform these movements. It is therefore important to consider each patient and their injury, and tailor their rehabilitation accordingly with the help of therapists. In some cases, even immobilization may be the appropriate option, for example in non-compliant patients and those with commitment injuries that require immobilization (e.g. fracture).

Conclusion

Tendons are highly complex connective tissues composed of specialized cells in a rich ECM predominated by type 1 collagen. The close interactions between cells and matrix are important for regulating the hierarchical tendon structure in response to applied forces as well as to injury. Structurally, the tendon unit is formed by collagen organized into microfibrils, fibrils, fibres and fascicles. Biomechanically, tendons have high tensile strength but also exhibit viscoelastic properties enabling energy-efficient movements; these material properties can be demonstrated on a stress–strain curve.

Tendons respond to injury via extrinsic and intrinsic healing mechanisms with overlapping inflammatory, proliferative and remodelling phases. Mechanical loading and tendon movement are important for tendon healing, however, its weakened state during its initial healing phases makes it prone to rupture. Adhesion formation remains one of the biggest resulting problems that limit its function after healing.

Most acute flexor tendon ruptures should be repaired early surgically. The aim is to have a repaired tendon with minimal gapping and gliding resistance, strong enough to undergo early rehabilitation. The repair strength is influenced by a number of factors, from suture material, grasp size to the actual repair configurations. To date, there are no single golden standards for surgical techniques, suture choices and rehabilitation protocols. One should therefore be mindful that management strategies should be individualized for each patient and their particular injury. Careful surgical technique, up-to-date rehabilitation regimens, and patient education remain the basis for optimal outcomes. ◆

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