# INTRODUCTION

Hand injuries account for up to 20% of all treated injuries in an Emergency Department. Furthermore, acute hand injuries are the most common occupational injury evaluated in emergency departments worldwide. The spectrum of traumatic hand injuries includes minor soft tissue injuries and fractures to complex injuries requiring nerve, tendon, or artery repair. Within this spectrum, tendon injuries are quite common and were reported in 54.8% of patients with a small laceration and 92.5% of patients with a deep injury through a small laceration. Thus, it is important for the treating physician to recognize that even a small laceration to the hand may involve the flexor or extensor mechanism ***(Johanna et al., 2014)***.

Historically the results of primary tendon repair were so poor that the area beyond the distal palmar crease was considered to be 'no man's land' and delayed tendon grafting of FDP was the standard treatment. Kleinert and Verdan established the superiority of primary repair for flexor tendon injuries. Successful outcomes depend upon meticulous surgical technique and early post-operative mobilisation. A non-absorbable 3/0 or 4/0 braided or monofilament stitch is usually used for the core suture, combined with a 6/0 epitendinous suture. There have been significant improvements in suture material and suture techniques for flexor tendon repair ***(Langley and Hobby, 2009)***.

Hyaluronic acid (HA) is an important component of articular cartilage; it is present as a coat around chondrocytes, where it bounds to aggrecan monomers, which imbibe water and are responsible for the resilience of cartilage (i.e., resistance to compression). Moreover, HA is a major component of the synovial fluid, and, along with lubricin, it is one of the fluid's main lubricating components. The biological activities of HA are very complex: (a) it inhibits matrix metalloproteinases (MMPs) and the phagocytic activity of macrophages and leukocytes; (b) it promotes the release of prostaglandins and the production of tissue inhibitor of MMP-1 and favours the normalization of native hyaluronan synthesis; (c) it acts as free radicals scavenger and stimulates proteoglycans synthesis by chondrocytes; (d) fnally, it is provided of protective effects on chondrocytes or cartilage explants from degradation by enzymes ***(Abate et al., 2014)***.

Recently, the viscoelastic properties of hyaluronic acid (HA) on liquid connective tissue have been proposed for the treatment of tendinopathies. Some fundamental studies show encouraging results on hyaluronic acid's ability to promote tendon gliding and reduce adhesion as well as to improve tendon architectural organisation. Some observations also support its use in a clinical setting to improve pain and function ***(Kaux et al., 2015)***.

# AIM OF THE WORK

The purpose of this study is to assess the effect of hyaluronic acid application in flexor tendon repairs to minimize adhesions and improve tendon gliding.

*Chapter (1)*

# ANATOMY OF FLEXOR TENDONS

## Flexor Tendon System

The flexor tendon system of the hand consists of the flexor muscles of the forearm, their tendinous extensions, and the specialized digital flexor sheaths. These components work in concert to produce smooth and efficient flexion of the individual digits of the hand. Injury to the flexor tendon system can lead to significant morbidity for patients.

In addition to having technical expertise, experienced hand surgeons must have precise knowledge of flexor tendon anatomy to guide appropriate treatment of injuries to the flexor tendon system ***(Bojsen-Moller and Schmidt, 1974).***

## Flexor Muscles of the Digits

The forearm can be divided anatomically into anterior and posterior compartments. The anterior compartment contains the flexor-pronator group of muscles, most of which arise from a common flexor attachment on the medial epicondyle of the humerus. The 8 muscles of the anterior compartment may be divided further into 3 distinct functional groups, as follows: (1) muscles that rotate the radius on the ulna, (2) muscles that flex the wrist, and (3) muscles that flex the digits.

The muscles that flex the digits include the flexor digitorum profundus (FDP), flexor digitorum superficialis (FDS), and the flexor pollicis longus (FPL).

## Flexor Tendons of the Digits

The anatomic relationships of the flexor tendons are usually discussed in terms of zones, shown in the figure below (Fig. 1).

The 5 zones discussed below apply only to the index through small fingers—separate zone boundaries exist for the thumb flexor tendon.

**Zone I** consists of the profundus tendon only and is bounded proximally by the insertion of the superficialis tendons and distally by the insertion of the flexor digitorum profundus (FDP) tendon into the distal phalanx.

**Zone II** is often referred to as "Bunnell's no man's land" indicating the frequent occurrence of restrictive adhesion bands around lacerations in this area. Proximal to zone II, the flexor digitorum superficialis (FDS) tendons lie superficial to the FDP tendons. Within zone II and at the level of the proximal third of the proximal phalanx, the FDS tendons split into 2 slips, collectively known as Camper chiasma. These slips then divide around the FDP tendon and reunite on the dorsal aspect of the FDP, inserting into the distal end of the middle phalanx.

**Zone III** extends from the distal edge of the carpal ligament to the proximal edge of the A1 pulley, which is the entrance of the tendon sheath (see the A1 pulley in the image above). Within zone III, the lumbrical muscles originate from the FDP tendons. The distal palmar crease superficially marks the termination of zone III and the beginning of zone II.

**Zone IV** includes the carpal tunnel and its contents (ie, the 9 digital flexors and the median nerve). Zone V extends from the origin of the flexor tendons at their respective muscle bellies to the proximal edge of the carpal tunnel.

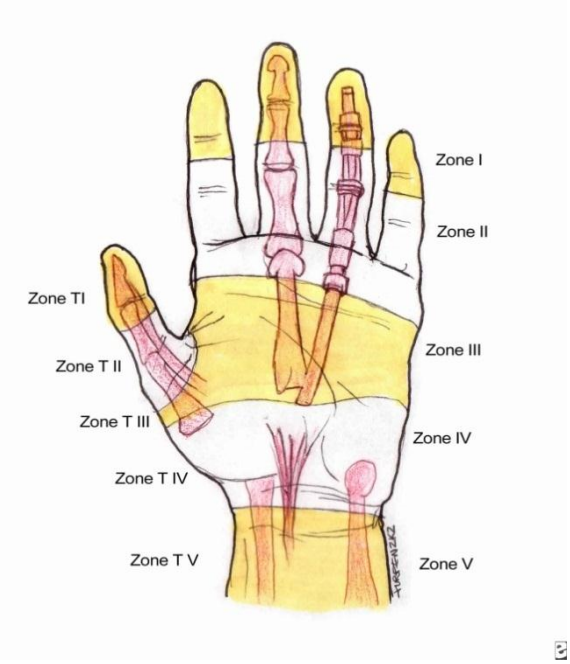


**Fig. (1):** Zones of the hand.

## Digital Flexor Sheath

The digital flexor sheath is a closed synovial system consisting of membranous and retinacular portions. The membranous portion is made up of visceral and parietal layers that invest the flexor digitorum profundus (FDP) and flexor digitorum superficialis (FDS) tendons in the distal aspect of the hand. The retinacular component consists of tissue condensations arranged in cruciform, annular, and transverse patterns that overlie the membranous, or synovial, lining. (Fig. 2) ***(Cohen and Kaplan, 1987).***

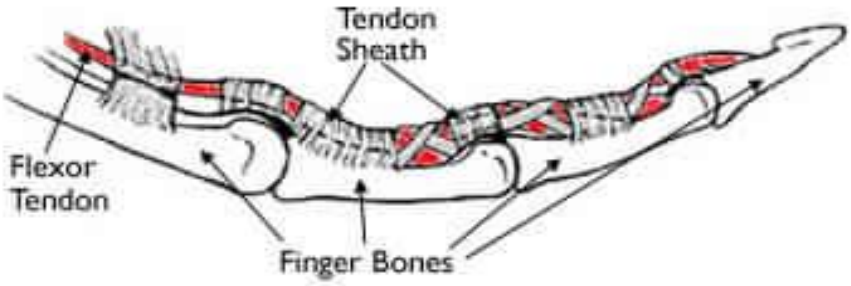


**Fig. (2):** The Flexor tendon sheath.

## Annular pulleys

*The 5 annular pulleys are as follows:* (Fig.3)

- **A1 pulley** - The first annular pulley arises from the palmar plate and proximal portion of the proximal phalanx, overlies the membranous sheath at the level of the MCP joint, and is approximately 8 mm in width; this pulley is released during surgical treatment of trigger finger (stenosing tenosynovitis).

- **A2 pulley** - The second annular pulley consists of oblique fibers that overlie annular fibers, originates from the proximal and lateral areas of the proximal phalanx, and is approximately 17 mm in width; this pulley should always be preserved when dealing with injuries to the retinacular system.

- **A3 pulley** - The third annular pulley is located at the level of the PIP joint; it attaches to the palmar plate and is approximately 3 mm in width.

- **A4 pulley** - Like the A2 pulley, the fourth annular pulley, located in the middle phalanx, also consists of oblique fibers overlying annular fibers and is always preserved during surgery of the retinacular system; the A4 pulley is approximately 6.7 mm in width and has been shown to be the most important biomechanical pulley for maintaining independent interphalangeal joint function.

- **A5 pulley** - The fifth annular pulley is located proximal to the DIP joint, just proximal to the termination of the membranous sheath; the A5 pulley is the thinnest of the 5 annular pulleys and has a width of 4 mm.

## Cruciform pulleys

***The 3 cruciform pulleys are as follows:***

- **C1 pulley** - The first cruciform pulley lies just distal to the A2 pulley.

- **C2 pulley** - The second cruciform pulley is located in the space between the A3 and A4 pulleys.
- **C3 pulley** - The third cruciform pulley is located distal to the A4 pulley; a number of anatomic variations have been described for the retinacular system.

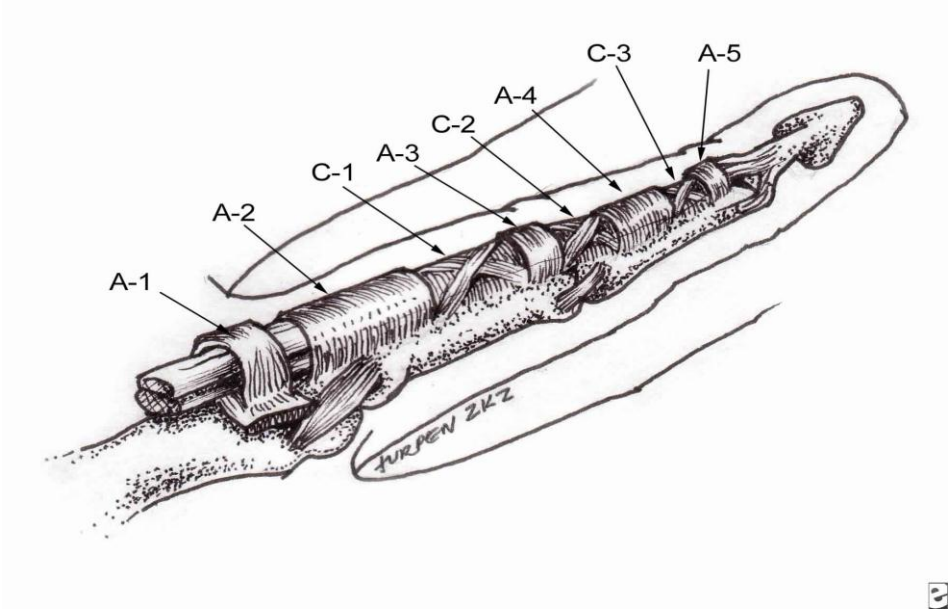


**Fig. (3):** The pulley system.

## Flexor Tendon Blood Supply

From their musculotendinous origin to the level of the A1 pulley, the flexor tendons receive their blood supply from the surrounding paratenon—a filmy layer of connective tissue that invests the tendon. The vascular supply is positioned primarily within the dorsal surface of the tendon; segmental blood vessels arise within the connective tissue, enter the

tendon, and run longitudinally between tendon fascicles to supply nutrients. (Fig.4)

Blood vessels are rarely, if ever, seen surrounding the tendons within the sheath. Within the sheath, the only connection between the tendons and the periphery is at the level of the vincula, which are folds of mesotenon that carry a blood supply to the tendons within the sheath. A number of variations exist, but most commonly, each tendon within the sheath is supplied by 2 vincula, 1 long (vinculum longum) and 1 short (vinculum breve) ***(Azar et al., 1983).***

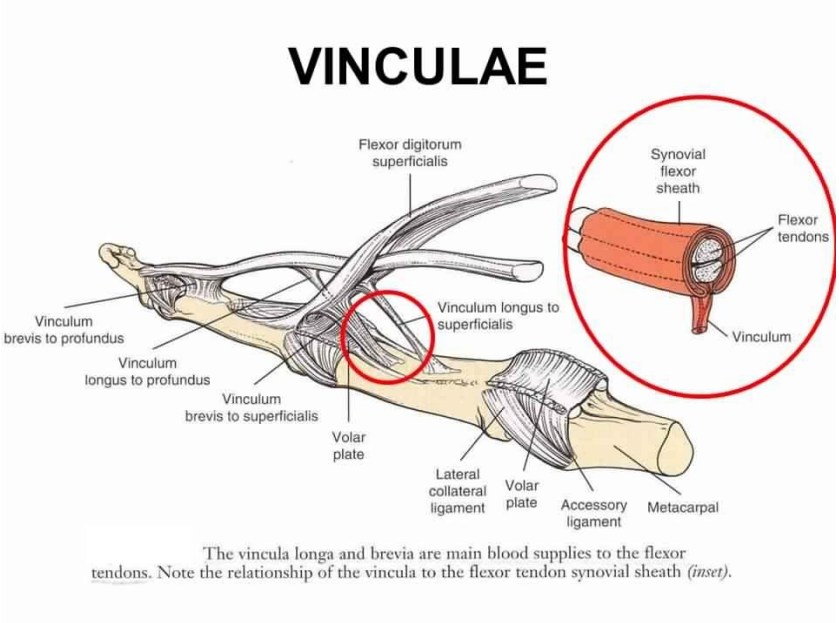


**Fig. (4):** Flexor tendons with attached vincula.

*Chapter (2)*

# PATHOPHYSIOLOGY OF TENDON INJURY AND TENDON HEALING

## Mechanism of Injury

Tendon injury can be classified by types of injury and are generally considered to be acute or chronic and either direct or indirect. Direct acute tendon injuries occur due to contusion, non-penetrating blunt injury from accidents and sports injuries, or laceration by a sharp object.

Indirect, tendon injuries are often the result of acute tensile overload and repetitive micro-trauma as seen in overuse injuries ***(Hyman and Rodeo, 2000).***

## Phases of Tendon Healing

When tendons are injured, the body initiates a process of healing and scar formation that can be divided into phases which are distinguishable by specific peaks in a cascade of cellular and biochemical events **(Fig. 5)**. These phases overlap and their duration can vary greatly due to location of injury or disease. Although the tensile strength of the healing tendon improves over time, it does not reach the levels of uninjured, normal tissue ***(Gomez, 1995).***

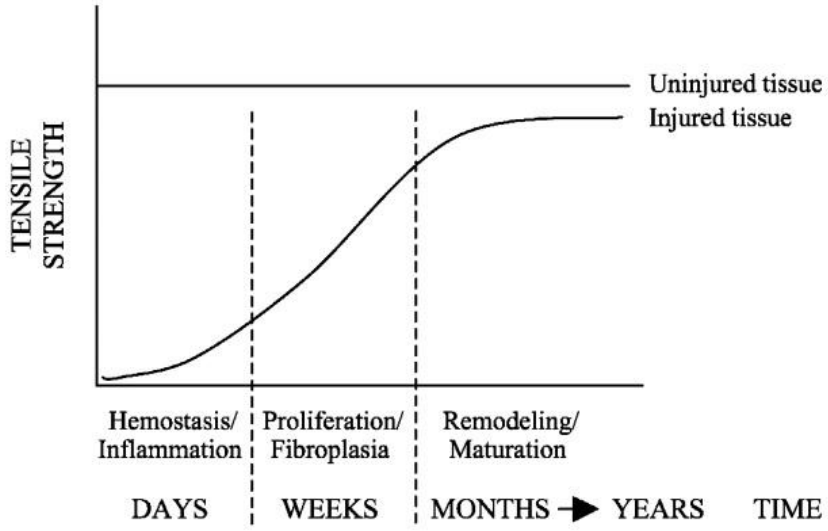


**Fig. (5):** Phases of tendon healing.

## ***Phase 1: Hemostasis***

This phase of tendon healing occurs almost immediately after tendon injury. First, the injury to the surrounding vascular vessels causes the formation of a hematoma. Next, the resultant clot and hemostasis activates a cascade of vasodilators and platelets, as well as the release of pro-inflammatory chemicals from mast cells. Inflammatory cells are attracted to the injury site and aggressively engage in the phagocytosis of necrotic tissue and debris and break down the blood clot. Macrophages aid in the recruitment of new fibroblasts and release of angiogenesis promoting factors to initiate growth of capillary networks within the wound ***(Fenwick et al., 2002).***

## ***Phase 2: Proliferation***

During this phase, a disorganized matrix of granulation tissue is present at the injury site. Histologically, the predominant cell types are fibroblasts along with a smaller number of macrophages and mast cells, type III collagen and DNA concentrations reach their peak amounts during the entire reparative process. These changes are believed to help with the optimization of collagen synthesis and the gradual conversion of type III to type I collagen ***(Gomez, 1995).***

## ***Phase 3: Remodeling***

Changes in the healing tissue can be seen with the naked eye as the previous red scar now becomes pinkish and translucent, forming a connection between the two ends. Histologically, fibroblasts have decreased in size and slowed their matrix synthesis, and collagen fibers have begun to orient themselves longitudinally along the long axis of the tendon. As the scar enters maturation there is a notable return of type III to type I collagen ratio, collagen crosslinks, and glycosaminoglycan, water, and DNA concentrations. However, healed tendon has been shown in many studies to take upwards to a year to more closely approach the functional strength of uninjured tissue ***(Buckwalter and Hunziker, 1996).***

## ***Cellular and extra-cellular matrix response to injury***

The process of tendon repair is a complex, orchestrated series of physiological events involving significant synthesis, migration, and degradation of extracellular matrix (ECM) components ***(Liu et al., 1995).***

After trauma, Tendon healing occurs intrinsically, via proliferation of epitenon and endotenon tenocytes, also the ECM degradation products are essential for tendon healing because they provide chemotactic signals for fibroblasts, leukocytes, and endothelial cells, and serve as a reservoir for cytokines ***(Jaibaji, 2000).***

Local factors such as infection, disease, tissue hypoxia due to vascular deficiencies, and malnutrition, as well as external factors such as rigid fixation and prolonged immobilization dramatically affect the healing process ***(Woo et al., 2000).***

## ***Effect of Immobilization on Tendon Healing***

Prolonged immobilization following tendon injury often results in harmful effects. Immobilization reduces the water and proteoglycan content of tendons, and increases the number of reducible collagen cross-links. Immobilization also results in tendon atrophy, but, due to low metabolic rate and vascularity, these changes occur slowly ***(Maganaris et al., 2006).***

*Chapter (3)*

# BIOMECHANICS OF FLEXOR TENDONS

Tendons are soft connective tissues consisting of parallel collagen fibers embedded within an extracellular matrix. This organized structure allows tendons to withstand and transmit large forces between muscle and bone. However, as tendons are subjected to repeated motion and degeneration over time, they are prone to both acute and chronic injuries. After injury, the healing process in tendons results in the formation of a fibrotic scar. The structural, organizational, and mechanical properties of this healed tissue are inferior to normal tendon ***(Frank et al., 1992).***

The functional outcome of the repair of tendon injuries depends upon many factors, including activity level, motion after injury, various injury modalities, and different injury locations. Many researchers have applied tissue engineering concepts to achieve better outcomes such as creating bioscaffolds, using exogenous compounds and applying cytokines to injured tissue, and recently using gene and cell therapy ***(Frank et al., 1997).***

## Mechanical properties of tendon

Tendons connect muscle to bone and form a musculotendinous unit whose primary function is to transmit tensile loads generated by muscles to move and stabilize joints. Under normal loads, it has been shown that tendons maintain