INTRODUCTION

Tendons are specialized tissues that connect muscle to bone and transmit the forces generated by muscle to bone, resulting in joint movement. Tendon injuries are common and affect a substantial portion of recreational and professional athletes and those in many occupations involving repetitive work[42].

Tendinopathy (often called tendinitis or tendinosis) is the most common tendon disorder. It is characterized by activity related pain, focal tendon tenderness, and decreased strength and movement in the affected area. Tendinopathy can occur in almost any tendon, common examples include planter fasciitis, Achilles tendinitis, patellar tendinitis and tennis elbow and supraspinatus tendinitis[58].

Tendinopathy or tendinosis is not characterized by an inflammatory response, but rather infiltration of fibroblasts and vessels, with an ensuing chronic cycle of tendon degeneration and repair resulting in a weakened tendon. These changes have been shown to appear as hypoechoic areas on sonography. Several techniques have been described to treat tendinosis[35].

Calcific tendinitis results from the deposition of calcium hydroxyapatite crystals in or around tendons, mostly in peri-articular locations. The shoulder is the most frequent location. However, calcific tendinitis may be encountered in a large variety of anatomical locations such as the hip, wrist, foot, and

cervical spine[12]. Calcific tendinosis is usually a self-limiting condition in which the calcification resorbs after period of worsening pain. however ,in some patients, the condition can lead to chronic pain and functional impairment the resolution of calcification correlates well with clinical improvement of symptoms and, therefore, various treatments have been devised to promote their removal[35].

Conventional non-surgical treatment options typically recommended include relative rest, cryotherapy, non-steroidal anti-inflammatory medications, physical therapy, and biomechanical devices. Surgical intervention has been reported as additional treatment option in those cases in which conservative treatment has failed [54] .Reported surgical success rates have been variable, with undesirable complication rates and prolonged recovery. In an effort to shorten recovery and reduce morbidity, less invasive approaches to the treatment of chronic tendon injuries have been studied and shown to be effective [22].

Despite the wide variety of medical and surgical therapies that have been used to treat chronic tendinosis, no one therapy has gained universal acceptance [2] .Various studies and meta-analysis have been failed to show support for definitive treatment options, with many studies producing inconsistent results [40].

With its associated technological improvements and associated lack of ionizing radiation, ultrasound (US) imaging

is ideal for guiding most musculoskeletal interventional procedures [32].

Unlike other imaging modalities, US has a unique advantage in that it can visualize soft tissues, bony landmarks, and the needle using real-time scanning, allowing dynamic visualization. In addition, there are no known contraindications to US [26].

Peritendinous injection of anesthetic and short acting corticosteroid is an effective means to treat tenosynovitis. Ultrasound guided injections have been shown to be an effective means to ensure correct localization of therapeutic agents [13].

Percutaneous needle aspiration and lavage is quite effective in calcific tendinitis in the short term and especially in the long term treatment. Lavage of calcification is performed using a needle introduced under sonographic guidance, which connected to a syringe filled with Lidocaine [14].

AIM OF THE WORK

The aim of the current study is:

To evaluate the effectiveness of the percutaneous ultrasound guided intervention in improving or treating different tendinopathy problems.

SONOGRAPHIC ANATOMY OF THE TENDONS

Anatomical point of view:

Tendons are the critical link in the musculoskeletal system, they connect muscle to bone .Their function, is to transmit the muscular tension to a mobile skeletal segment [16].

They are characterized by high tensile strength, similar to that of the bone. It is known that a tendon with 10 mm^2 transverse section can bear up to 600-1000 kg. On the other hand, tendons are not very elastic, and can only tolerate a maximum elongation of about 6% before being damaged [37].

Macroscopically, they appear as ribbon-like structures, with extremely variable shape and dimensions, they can be round, (e.g., biceps tendon), oval (e.g., Achilles tendon), or flattened (e.g., patellar tendon) in cross section [25].

Microscopically, they are made of type I collagen (about 70 % of the dry weight). This collagen has a complex arrangement made up of highly ordered bundles of fibers grouped into fascicles; most of them have a course longitudinal to the tendon axis; while some of them assume transverse and spiral arrangements. This configuration leads to their higher tensile strength [18].

These fibers run parallel together to form the primary bundles (sub fascicle), among them, fibrocytes are endowed with large laminar protrusions, named tenocytes or alar cells. Also elastic fibers (about 4%) can also be found; their role is not different from that of a “shock absorber "when muscular contraction begins. The collagen and elastic fibers both have the same direction as the main lines of force and are lying in a gel consisting of proteoglycans and water [53].

The primary bundles are assembled to form secondary bundles (fascicle), which represents the tendon’s functional unit; these fascicles are clustered in tertiary bundles. These bundles are separated and surrounded by a thin connective strip called the **endotenon,** within which vessels and nerves run. The **epitenon** is a stronger connective tissue covering and surrounding the whole tendon [37]. (Figure 1)

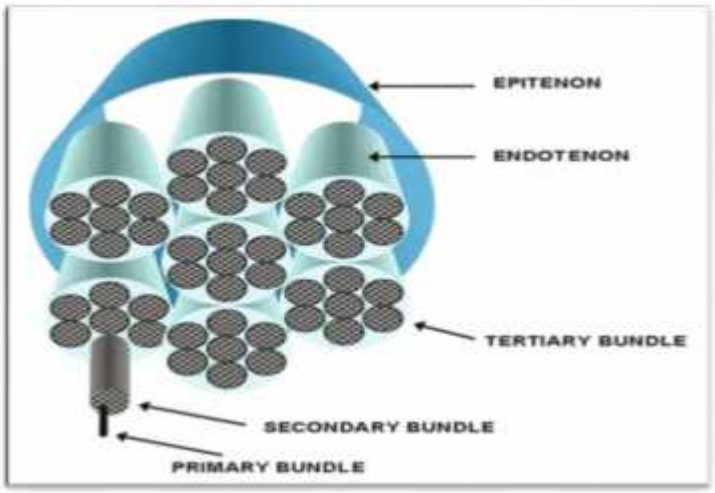

Figure (1): Anatomical drawing of the tendon ***(Quoted from Martino, 2007)***

- **Types of the tendons:**

From a functional and anatomical point of view, tendons can be divided into two types: supporting tendons (or anchor tendons) and sliding tendons [37].

1) **Anchor tendons** (such as the Achilles and the patellar tendon): are typically bigger and stronger than sliding tendons, they are not provided with a synovial sheath, but they are surrounded by a connective lamina external to the epitenon, called peritenon; the two connective sheaths (epitenon and peritenon) form the paratenon together with highly vascularized adipose and areolar tissue [53.]

2) **Sliding tendons** (such as the biceps tendon) are wrapped in a covering sheath called tenosynovial sheath, whose function is to guarantee better sliding and protection to the tendons, when they run adjacent to irregular osseous surfaces, sites of potential friction. This sheath consists of two layers: a visceral layer, strictly contiguous to the epitenon, and a parietal, more external, layer. The two layers come together to form a synovial "fold" named mesotenon, a closed cavity, nearly virtual, containing a very small amount of synovial fluid, is. This sheath corresponds anatomically and functionally to the peritenon of anchor tendons and, similarly, the tenosynovial sheath and the epitenon together constitute the paratenon of the sliding tendon [37]. (Figure 2**)**

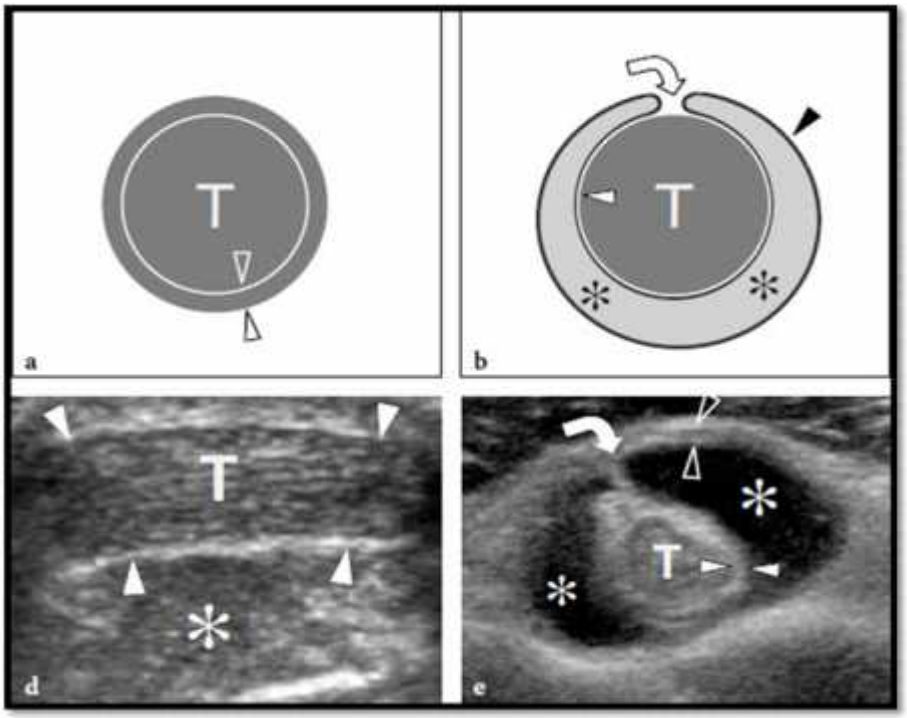

Figure (2): Tendon envelope: (A) schematic drawing and (D) correspond Sonographic short axis view of type I tendon, where peritenon (arrowheads) is demonstrated, separating the tendon form the surrounding fat (asterisks).(B) schematic drawing and (E) correspond Sonographic short axis view of type II tendon covered by synovial sheath , where the presence of synovial fluid (asterisks) allow detection of both visceral (white arrowheads) and parietal (open arrowheads) layers with mesotenon (curved arrow) connecting them ***(Quoted from Zomorani, 2007)***

- **Vascularization:**

The vascularization varies according to the type of tendon. In sliding tendons, the vessels run within the mesotenon, these vessels pass therefore along the tendon's surface, where some arterioles arising from the vessels penetrate into the tendon following the course of connective laminae. On the other hand, the vessels of the anchor tendons

constitute a thick and irregular anastomotic net within the paratenon, from which arteriolar vessels arise and penetrate inside the tendon to different levels, following the course of the connective laminae [34]. (Figure 3)

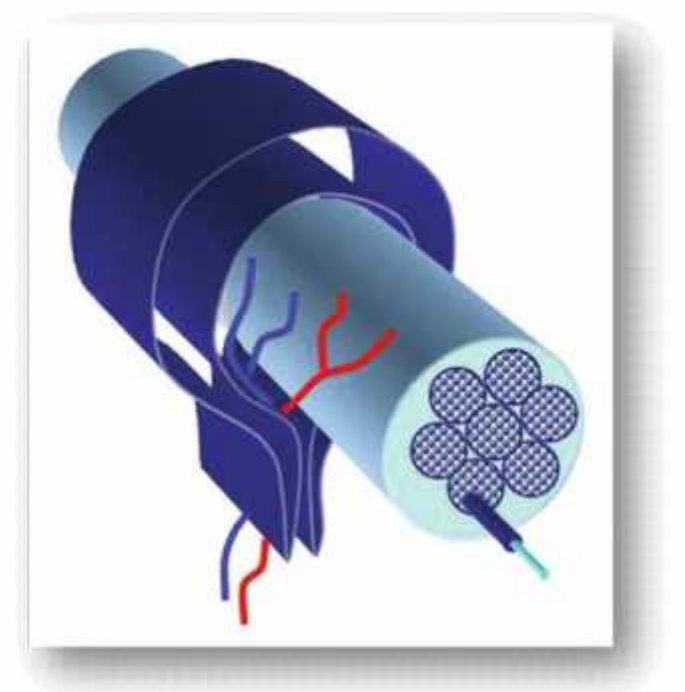

Figure (3): Vascularization of the sliding tendon ***(Quoted from Martino, 2007)***

Tendons may present with less vascularized zones, which is named critical areas, which are extremely important in the pathogenesis of several tendon diseases. Examples include: the pre-insertional area of the supraspinatus tendon of the shoulder joint, or the central part of the Achilles tendon, which typically constitute highly susceptible sites of the degenerative disease and tendon rupture [37].

Enthesis:

It is the point of union between the tendons and the muscle or the bone, known as myotendinous junction and osteotendinous junction respectively. The myotendinous junction is usually well-defined and represent the level where the tendon fibers intertwining with the endomysium fibers. The osteotendinous junction has a more complicated structure: its nature may be either fibrous or fibro cartilaginous according to the tendon mobility [34].

Sonographic anatomy:

Nowadays US represents the gold standard technique for the assessment of tendons. With the advantage of high resolution transducers and specific image processing software, it became possible to make detailed analysis of the shape and structure of tendons. In addition, US is the only technique that allows the radiologist to perform a dynamic study of tendons, which is extremely important for the diagnosis of tendon pathology [19].

In longitudinal ultrasound views (long axis): the tendons appear as echoic ribbon-like bands, defined by a marginal hyperechoic line corresponding to the paratenon and characterized by a fibrillar internal structure, represented by a succession of thin hyperechoic parallel bands, slightly wavy, which tend to grow apart from one another when the tendon is released and to move closer when the tendon is tense. This

fibrillar echo structure is caused by the specular reflections within the tendon determined by the existing acoustic interface between the endotenon septa. The number and thickness of such structures change depending on the frequency of the transducer[38]. (Figure 4, 5)

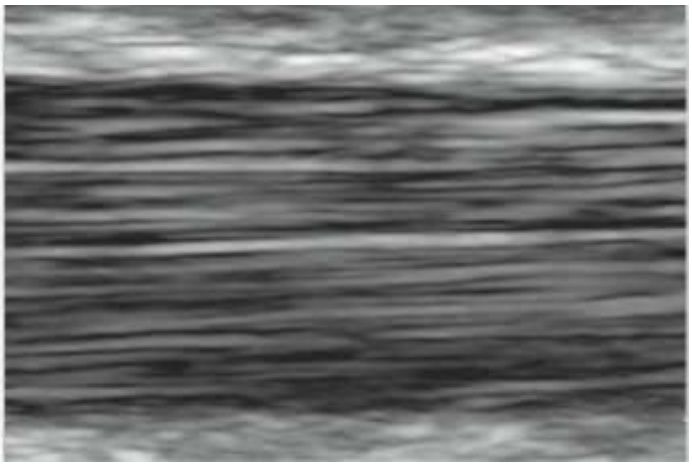

Figure (4): The fibrillar echotexture of a normal tendon in longitudinal axis view ***(Quoted from Martino, 2007)***

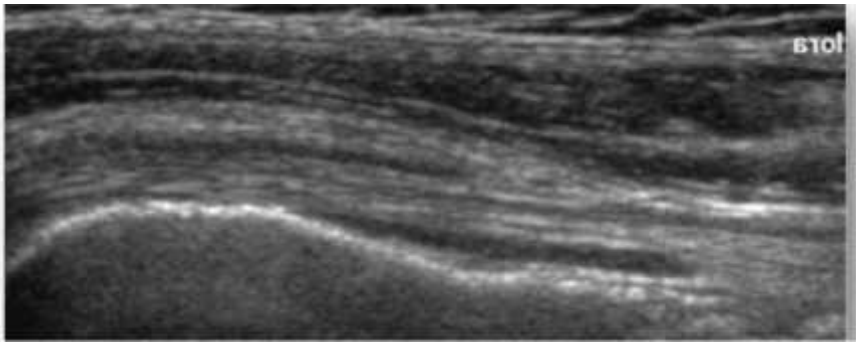

Figure (5): Normal longitudinal view of biceps tendon ***(Quoted from Zomorani, 2007)***

In transversal views (short axis), tendons appear as round or oval-shaped structures characterized by several homogeneously scattered spotty echoes[38]. (Figure 6, 7).

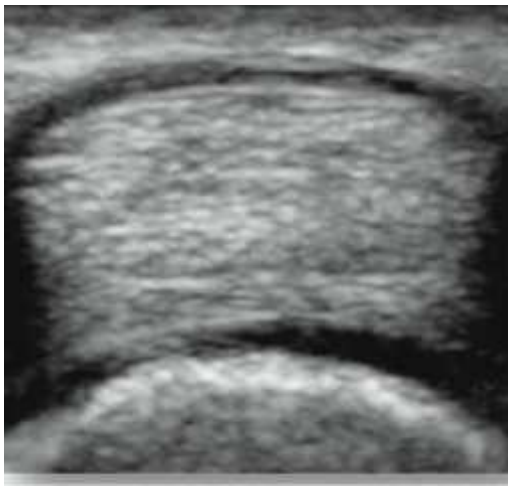

Figure (6): Short axis view of tendon, showing the characteristic hyper echoic pattern, with scattered spotty echoes ***(Quoted from Martino, 2007)***

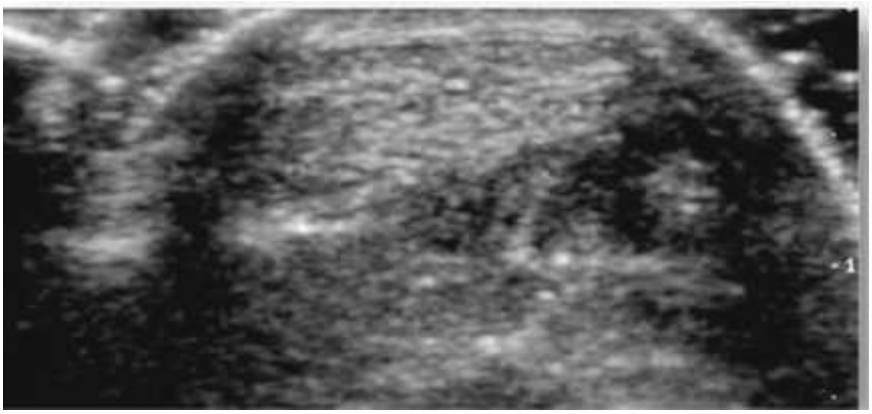

Figure (7): Short axis view of Achilles tendon, showing the oval shaped tendon with characteristic hyperechoic spotty echoes ***(Quoted from Zomorani, 2007)***.

PATHOLOGY OF TENDINOPATHY

Introduction:

Tendon injuries are common and affect a substantial portion of recreational and professional athletes, and those in many occupations involving repetitive work [42].

It is a common clinical problem, which can occur in any tendon, mostly near its insertion (enthesis), where there is an area of stress concentration; also it is directly related to the volume of repetitive load, to which, the tendon is exposed [58].

In inflammatory tendinopathies all the layers of the tendons are involved (*tendinitis*), while the tendon's parenchyma (collagen fibers, proteoglycans) is usually only affected in degenerative conditions (*tendinosis*), where the two pathologic conditions often coexist [37].

Moreover, tendinitis can be distinguished in *tenosynovitis* and *peritendinitis*, according to the specific involvement of sliding tendons or supporting tendons, while inflammatory and degenerative involvement of the osteo-tendinous junction is called *enthesopathy* [37].

Tendinopathy (often called tendinosis) is the most common tendon disorder; it is characterized by activity –related pain, focal tendon tenderness, and decreased the strength and the movement of affected area [58].

It is poorly understood, and has many described remedies with a very little evidence to support their efficacy, one of the

reasons there are few, if any good treatment for it, is the lack of knowledge regarding its pathogenesis[58].

It is believed that the incidence of tendinopathy increase with age, male gender and obesity, also there is an association between Tendinopathy and hormone replacement therapy and oral contraceptives in women [27].

Histopathological changes [45]. (Table 1):

- **Macroscopically*:*** the normal tendon is brilliant white in color with firm fibroelastic texture, while the tendinopathic tendon is gray or brown in color with soft, thin and fragile texture.
- **Microscopically:** the normal tendon show well organized parallel collagen fibers with spindle shaped nuclei in parallel manner, while in tendinopathic tendon, the collagen bundles are disorganized with increased ground substance and the nuclei are darker stained, rounded shaped **.** (Figure 1)
- **By electron microscope:** the tendinopathic tendon shows angulated collagen fibers that vary in diameter and orientation, with changes consistent with hypoxia as lipid vacuoles, enlarged lysosomes, degranulated endoplasmic reticulum, often shows vascular and small vessels infiltration with rarely inflammatory cells, these changes are consistent with (degeneration) and attempt (regeneration) .

Table (1): Comparison between the normal and tendinopathic tendon by macroscopy, both the light and electron microscope and ultrasound finding ***(Quoted from Yinghua, 2008)***

	Macroscopic	Itrasound	Light microscopic	Electron microscopic
Normal tendon	Brilliant white Fibroelastic firm texture	Parallel hyperechoic or bright white lines Regular uniform fiber structure	Organized parallel collagen bundles. Spindle shape tenocyte nuclei. Nuclei parallel alignment	Densely packed collagen fibers Uniform in diameter and orientation of collagen fibers
Tendinopathy	Grey or brown Thin, fragile and disorganized Loose texture	Localized widening of the tendon. Local hypoechoic areas Irregular fiber structure Neo-vascularization correlate with changes	Disorganized collagen bundle Increased ground substance consisting of proteoglycans and glycosaminoglycans (GAG) Large mucoid patches and vacuoles between fibers Round with darker-staining tenocyte nuclei Markedly increase number of nuclei with loss of parallel alignment Increase of vascular and nerve ingrowths	Angulation, bubble formation of collagen fibers. Variations in the diameters and orientation of collagen fibers. Hypoxic changes in tenocytes (lipid vacuoles, enlarged lysosomes and degranulated endoplasmic retinaculum)