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Research Article

Epidemiology and Management of Tendocalcaneal Injuries: Evidence Based Clinical Practice

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Abstract

Tendocalcaneous, also known as 'Achilles tendon' is one of the strongest tendon in the human body and plays a pivotal role in lower limb movement. Achilles tendinopathy is a common problem facing the young as well as the old and often occurs during the third or fourth decades of life. Achilles tendon conditions adversely affect persons in form of morbidity, pain, limited movement and adverse quality of life. Also, it takes its toll on the federal system in terms of logistic burden on health care, rehabilitation and insurance systems, commercial work loss days, enforced early retirement set-backs and loss of sports personnel. Hence, adequate knowledge about tend calcaneus conditions and their management is important to anatomists, orthopedic surgeons, physiotherapists, practicing physicians and nurses alike. This Research article describes the pathophysiology of Achilles tendon conditions, it's epidemiology and management. A distinctive feature of this paper is the objective comparison between 'operative 'versus 'non operative/ conservative' approach in dealing with Achilles tendon conditions (tendinitis and tendinopathy). Both lines of management and their outcomes have been comprehensively reviewed through many evidence based researches in order to present a clearer picture to the clinical community of the risks and benefits involved and deciding the practicable approach in unique patients. Also, an everlasting debate among practicing musculoskeletal care givers regarding 'Early functional rehabilitation' versus 'Traditional immobilization' in surgical tendon repair cases after acute tendon rupture has been focused through conclusions and recommendations of relevant and notable researches.

Keywords: Tendinopathy; Tendocalcaneous; Achlles Tendinitis; Achilles Tendinopathy; MeSh terms; Achiles Tendon; Tendinopathy; Degeneration

Abbreviations

NHS: (National Health Sciences); TRIP: (Turning Research into Practice); CIHHAL: (Cumulative Index to Nursing and Allied Health Literature).

Introduction

Tendocalcaneous, also known as 'Achilles tendon' is one of the strongest tendon in the human body and plays a pivotal role in lower limb movement. Achilles tendinopathy is a common problem facing the young as well as the old and occurs in both elite and recreational athletes. Tendon ruptures are thrice more common among males [1]. It often occurs during the third or fourth decades of life and is specially common among those who lead customarily sedentary lifestyles [2] except on the weekends; the so called "weekend warriors". Achilles tendon conditions take their toll on the person affected in form of morbidity, pain, limited movement and adverse quality of life. The immediate family of the patient also suffers from psychological discomfort, time, care giving and financial issues. Also, it takes its toll on the federal system in terms of logistic burden on health care, rehabilitation and insurance systems, commercial work loss days, enforced early retirement set-backs and loss of sports persononell.

Hense, adequate knowledge about tendocalcaneus conditions and their management is important to anatomists, orthopedic surgeons, physiotherapists, practicing physicians and nurses alike.

This Research article elucidates the functional anatomy of Achilles tendon and describes the pathophysiology of Achilles tendon conditions (tendinitis and tendinopthy), it's epidemiology and management. A distinctive feature of this paper is the expansive comparison between 'operative 'versus 'non operative/conservative' approach in dealing with Achilles tendon conditions. Both

lines of management and their outcomes have been comprehensively reviewed through many evidence based research studies in order to present a clearer picture to the clinical community of the risks and benefits involved and deciding the right and practicable approach in unique patients. Also, an everlasting debate among practicing musculoskeletal care givers regarding 'Early functional rehabilitation' versus 'Traditional immobilization' in surgical tendon repair cases after acute rupture has been dealt with by presenting conclusions of relevant and notable researches.

Methodology of review

A literature search was performed using various medical database: NHS, TRIP database, Cochraine library, PubMed, MedLine, Embase, CINHAL, Global health, Web of Science, Scopus, PubMed, Clinical Queries during the time period of January to April 2018 using the following key word search strategy: (Ankle, Sports, Achilles tendon OR Tendo calcaneus OR Tendinopathy OR tendinitis OR tendonitis OR tendon rupture) AND (Achiilles tendinopathy prevalence OR incidence OR epidemiology OR etiology OR operative, nonoperative management). Studies (reviews, meta-analysis, case control, prospective cohort, cross sectional studies, case studies) reporting on Achilles tendinopathy, tendinoses, etiology, epidemiology, traumatic injuries, management, rehabilitation, incidence, prevelance were included.

Studies on upper limb orthopedic / muscular conditions: epicondylitis, rotator cuff tendinopathy and hip or knee joint conditions: patellar tendinopathy tenosynovitis, Non-english studies, reports on tennis or golfers elbow were excluded. Reviews older than 20 years, animal, and cadaveric studies were also excluded.

The search returned 1500 articles, of which 121 were included based on best match of the pre-set inclusion criteria. The items of the inclusion criteria used to filter studies were:

- Was an a priori design provided?
- Was there duplicate study selection and data extraction?
- Was a comprehensive literature search performed?
- Was the status of publication (i.e., grey literature) used as an inclusion criterion?
- Was a list of studies (included and excluded) provided?
- Were the characteristics of the included studies provided?
- Was the scientific quality of the included studies assessed and documented?
- Was the scientific quality of the included studies used appropriately in formulating conclusions?
- Were the methods used to combine the findings of studies appropriate?
- Was the likelihood of publication bias assessed?
- Was the conflict of interest stated?
- What were the items of the inclusion criteria in these studies?

Two researchers independently conducted the search according to the preset criteria and pooled in a repository of selected articles. The search was further refined according to the characteristics of the cohort (athletes, workers, general population, and patients with comorbidities), sample size, age group, duration and type of tendon condition and the reported prevalence and incidence data, management techniques used. The main body for information was derived from Cochrane Database of Systematic Reviews, Journals: Bone and Joint Surgery Am, Clinical Orthopedics and Related Research, Knee Surgery, Foot and Ankle surgery. Sports Traumatology, Arthroscopy, The American Journal of Sports Medicine, The British Journal of Sports Medicine, Orthopedic trauma and rehabilitation journal.

Results

42 studies qualified for the present review article. 10 Systematic review, 4 critical review (considering publication status and methodology of selected studies), 6 meta-analysis (more recent meta-analysis offering best currant evidence were preferred), 9 cohort studies, 6 case reports, 5 randomized control trials (those using superior methodologies), 2 Conference publications. (Figure 1) reference-manager software -Ref Works and End Note were used to import citations and duplicates were removed. The reference-manager program Mendeley (Elsevier) was used during manuscript preparation.

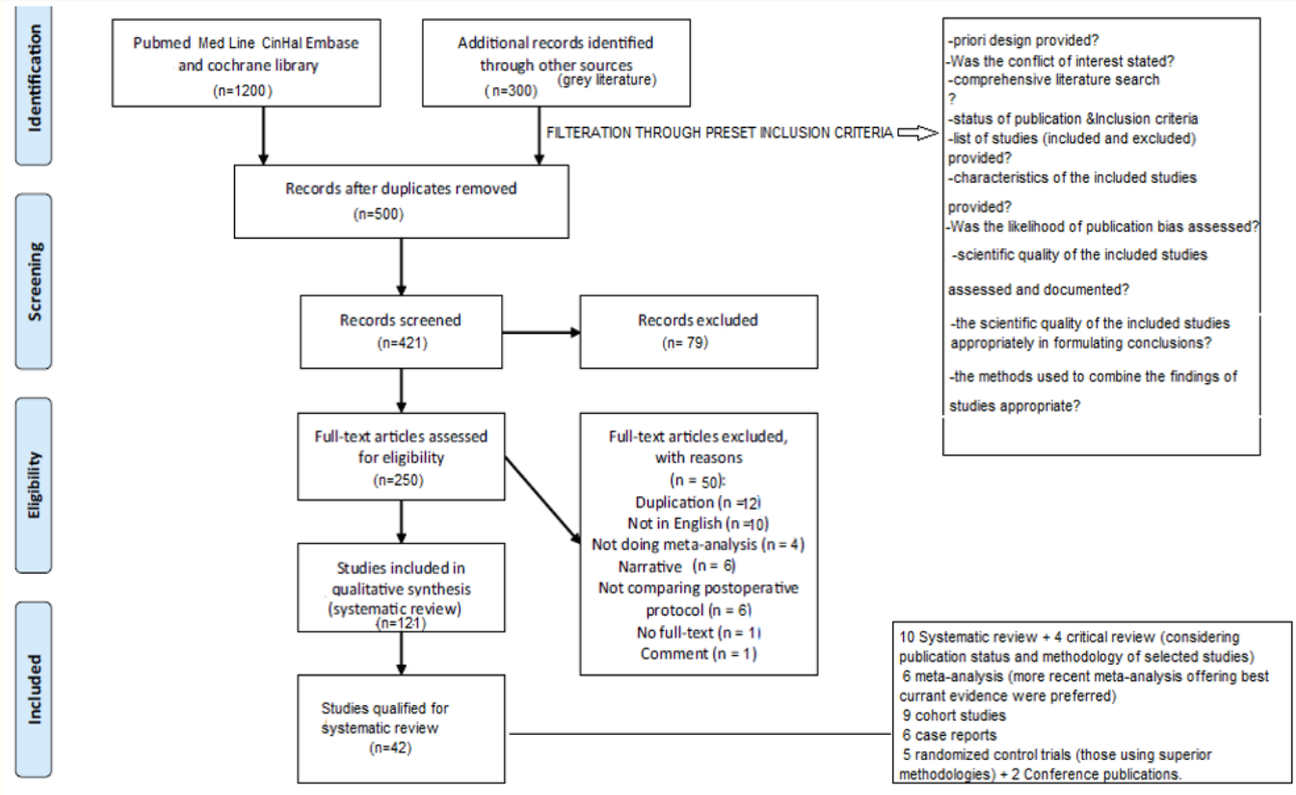

Figure 1: Flow-pathway demonstrating article filtering and systematic analysis for literature review.

*CINAHL, Cumulative Index to Nursing and Allied Health Literature; EMBASE, Excerpta Medica Database; MEDLINE, Medical Literature

Analysis and Retrieval System Online; RCT, randomized controlled trial. ''Cochrane Library Database'' refers to any of the following: Cochrane database; Cochrane Musculoskeletal Injuries Group Specialized Register; Cochrane Bone, Joint, and Muscle Trauma Group Specialized Register; Cochrane Database of Systematic Reviews; Cochrane Central Register of Controlled Trials.

Discussion

The Achilles tendon: history of its name

Renowned as the largest and strongest tendon in the human body, the 'Achilles tendon' derives its name from the legendary Greek mythological character 'Achilles' who was the hero of the Trojan war and was said to be undefeatable. The Dutch anatomist 'Verheyen' [3] coined the name in 1963. The strength and endurance associated with the name of Achilles underlies the history of naming tendo-calcaneus as the 'Achilles tendon'. The tendon has a high capacity to withstand tensional forces of locomotion. Anatomically, the Achilles tendon is formed by the common tendinous insertion of two main superficial calf muscles: gastrocnemius and soleus (along with plantaris) into the calcaneal bone (Figure 2).

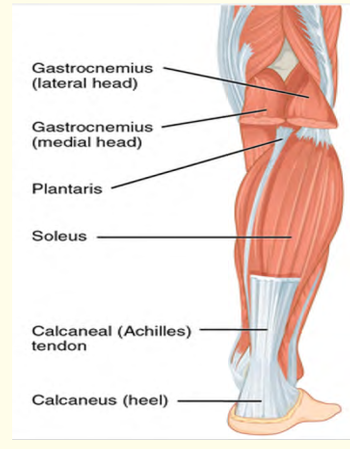

Figure 2: Achilles tendon anatomy.

(Source Greys anatomy for students volume 12 edition 5)

Functional anatomy and histology

The tendon contributes to the insertion of superficial calf muscles to the heel bone and backs the main propulsive force of calf muscles during walking. The tendon is mainly fibrous in nature (collagen fibers) and has high tensile strength. It starts in most people slightly below the mid-point of the posterior compartment of leg and ends by attaching at the back of calcaneus (calcaneal tuberosity). Being about 15 cm long; the narrowest part of the tendon lies about 4 cm above its bony insertion. At the insertion point, the gap between tendon and bone is occupied by calcaneal bursa, fat and areolar tissue. The tendon functions through calf muscles as a planti-flexor and knee flexor. It can bear 4 times the body weight during walking and 7 times the weight during running [4].

The normal tendon is avascular and fibrillar; containing 90% type I collagen and 10% elastic fibres aligned longitudnally. 90-95% of cellular element of the tendon exhibits two cell types; tenoblasts and tenocytes [5]. These cells are well organized in a healthy tendon. 5-10% of cells are chondrocytes (at enthuses) and a few synovial cells (in synovial tendon sheath). The extracellular matrix contains glycosaminoglycans, glycoproteins and proteoglycans. The high hydrophilicity of the matrix contributes to overall elasticity of the tendon. Other collagen types present are Type III, V, XII. Type III collagen promotes tendon healing and development. This holds implications for mechanical strength of the tendon. Type V collagen inserts into the core of type I collagen and forms template for fibrillogenesis which modulates fibril growth. Collagen type XII has binding ability [6] and it integrates neighboring matrix components like proteoglycans, fibromodulin, and decorin.

Collagen forms microfibrils, fibrils, and fibers. Fibers group to form a fascicle and fascicles unite to form bundles. The bundles are enclosed by an endotenon sheath which carries blood vessels, lymphatics, and nerves.

Blood supply to the tendon is varies according to age of the person and morphological segment of the tendon [7]. The blood supply to middle portion of the tendon is derived from the surrounding paratenon. In young people, the most abundant blood supply zone is located at the tendon insertion. Those older than 30 years, exhibit the tendon origin as the most intensely vascularized zone. The tendon zone -2 to 6 cm above its 'insertion point' into the calcaneus is the least vascularized zone in all age groups. Therefore, it has limited reparative ability from stress or injury.

The nerve supply to the tendon is derived from overlying superficial nerves or the tibial nerve and its branches. Afferent receptors are: Golgi tendon organs (mechanoreceptors; most abundant and detect pressure and strech stimuli); Ruffini corpuscles (pressure receptor); Vater-Pacinian corpuscles (movement receptor) and free nerve endings (pain receptors).

Pathology of Achilles tendinopathy

In tendinopathy patients, the Achilles tendon is stiffened, rough, and brownish. Histological examination shows no inflammatory cells. Microscopic examination demonstrates an increased number of tenocytes (showing irregular shape and high apoptosis rates) and high concentration of glycosaminoglycans in ground matrix, fragmented collagen, and neovascularization [8]. Since there is no 'real inflammation; rather the changes are more representative of degeneration; the term 'tendinopathy' seems preferable over misleading terms like 'tendinitis' or 'tendinoses'. In advanced cases, there is chronic mucoid and/or lipoid degeneration of the tendon with a variable amount of fibrocartilaginous metaplasia and calcium hydroxyapatite deposits.

In chronic Achilles tendinopathy there is increased expression of type III collagen, fibronectin, tenascin C, aggrecan, and biglycan [8]. These changes might be an adaptive reaction to changes in mechanical loading because repeated minor strain is considered as the major precipitating factor in tendinopathy. Healthy tendons are fairly a-vascular. Neovascularization and accompanying neo-innervation specially in the mid portion of the tendon is one feature of Achilles tendinopathy. These neo-nerves are the source of chronic pain. Interestingly, oxygen saturation in tendon does not show any difference between tendinopathy and normal tendon models [9].

Natural Healing process of Achilles Tendinopathy

Tendon repair occurs in several phases.

The *inflammatory phase* lasts a few days showing erythrocytes and inflammatory cells migration to the injury site within the first 24 hours and release of vasoactive factors. There is increased vascular permeability, initiation of angiogenesis, tenocyte proliferation and collagen fiber production.

Secondly, the *proliferative phase* begins; lasting for few weeks. Synthesis of type III collagen peaks in this stage. Water and glycosaminoglycan concentrations are elevated. Tenocyte proliferation marks the tendon repair stage [10].

The re-modeling phase starts after roughly 6 weeks when the healing tissue is redesigned. This is primarily a 'fibrous consolidation phase' and repair tissue substance transforms from cellular to fibrous elements. Collagen fibers align along the direction of the

mechanical loading [11]. Then, approximately after the tenth week postinjury, the maturation phase starts; with gradual change of fibrous tissue to scarlike tissue over the tendon and it continues within the course of one year.

Failed healing response in the tendon presents with three continuous stages (reactive phase, dis-repair phase and degenerative tendinopathy phase). Inflammatory response is not found in these three stages [12]. Histological samples of such models show increased tenocytes and glycosaminoglycans in the ground substance, disorganization and fragmentation of the collagen fibres, and compensatory neovascularization.

The source of pain in Achilles tendinopathy

Pain is constant and its origin is multi-factorial [13]. Increased prostaglandins in matrix, neovascularization, neo-nerves (neonueronal cholinergic systems), tenocyte structural and functional changes, complex catabolic and necrotic changes; all jointly contributing to pain sensation. Chemical irritants like cytokines and interleukins, neuropeptides and neurotransmitters such as glutamate are constantly elevated in tendinopathy and cause pain.

Etiology of Achilles tendon disorders

Owing to perplexing terminology adopted for Achilles tendon disorders, Khan., *et al*. recommended in their 2002 BMJ publication that "The clinical syndrome characterized by a combination of pain and swelling (diffuse or localized) in or around the Achilles tendon, accompanied by impaired movement should be called Achilles tendinopathy" [13]. Now, Achilles tendinopathy is used often by clinicians as a 'blanket' term for "tendinitis", "tendinosis", and "tendon rupture" (Figure 3).

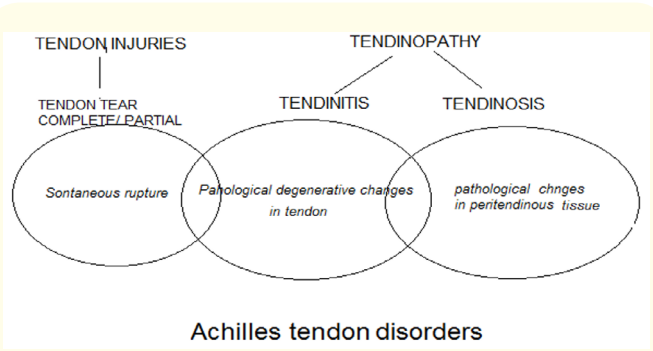

Figure 3: Achilles tendon conditions qualifying for the 'Achilles clinical syndrome'.

Achilles tendinopathy is a debilitating condition related to overuse injury from chronic or acute exposure to repetitive micro-traumas. Several 'intrinsic' and 'extrinsic' factors have been reported to contribute to the development of the condition. Genome wide association studies have revealed genetic factors undelying development of the condition.

The intrinsic and extrinsic risk factors for Achilles tendinopathy either play alone or in combination. Intrinsic factors mainly comprise of biomechanical or physiological factors like lower extremity abnormalities, systemic comorbid conditions (diabetes, hypertension), increasing age, obesity, inflammatory arthropathies, corticosteroids etc. Extrinsic factors arise from locomotion and environmental issues like excessive mechanical overload, sports training errors, abrupt changes in loading, mileage, interval training and scheduling, excessive up or downhill training, hard or slippery grounds, foot wear misfit with poor shock absorption.

Some investigators avoid the term ''degeneration'' since it can be misinterpreted as being an 'irreversible' pathologic process, whereas tendinosis likely is reversible. Hence, the term degeneration is merely representative of findings seen in histo-pathologic samples. Achilles tendon can undergo spontaneous ruptures or overuse injuries. Most problems in any given population can be traced back to sports and exercise-related stress. Sometimes, 'excessive loading' induced injury ensues.

At other times, spontaneous degeneration of the tendon can occur; [15] often without any predisposing systemic disease. Rarely, (~2%) comorbid systemic conditions like rheumatoid arthritis, gout, ankylosing spondylitis, chronic uremia, hyperparathyroidism etc. can manifest as Achilles tendon symptoms, often presenting with tendon ruptures.

Most systematic reviews report tendinopathy (55%-65%) as the most commonly reported disorder among foot and ankle conditions; followed by insertional problems: retrocalcaneal bursitis and insertional tendinopathy (20%-25%) [14]. Younger patients with Achilles like symptoms present with co-existing calcaneal apophysitis commonly known as Sever's disease. Sports or overuse injuries can occur either from intrinsic or extrinsic factors; or a combination of both (Table 1).

Predisposing Factors Related to Achilles tendinopathy			
Intrinsic factors		**Extrinsic factors**	
General factors	**Local (anatomic) features of lower limb**	**General factors**	**Sports-related factors**

• Gender *(males)*
• Age *(old)*
• Obesity
• Physical constitution *(weak)*
• Blood group *(ABO)**
• HLA-types
• Predisposing diseases and or Co morbid conditions
• Blood supply: (*Ischemia)*
• Oxygen concentration in blood: (*Hypoxia)*
• Body temperature: (*Hyperthermia)*
• Inherited disorders
• *(Ehlers-Danlos syndrome*)
• Endocrine
• Metabolic diseases
• *(amyloidosis)*
• Rheumatologic disease
	• Valgus / Varus foot
	• Plantar arch
	  • *pes planus (flat)*
	  • *Pes cavus (concave)*
	• Lower limb length *discrepancy >20 mm*
	• Ankle joint flexibility *(Decreased or increased)*
	• Sub-talar mobility *(limited)*
	• Musculo-tendinous tightness *(Decreased or increased)*
	• Muscle hypoplasia
	• Muscle weakness**
	• Mal-alignment of bones at ankle joint
	• Faulty foot movements
	  • *Hyper-pronation*
	  • *Increased inversion*
	  • *Decreased dorsiflexion*
		• Therapeutic agents
		  • Corticosteroids (local and systemic)
		  • Antibiotics *Fluoroquinolone*
		  • Weight-lowering drugs
		• Narcotic drugs
		  • *Cannabis*
		  • *Heroin*
		  • *Cocaine*
		• Environmental conditions *(Extreme cold, hot, humid, windy, high altitude)*
			• Excessive loading of lower extremities***
			  • Speed *(High/vigorous/ repetitive movements)*
			  • Foot wear *(Faulty)*
			  • Training surface *(Hard/ irregular)*
			  • Training errors *(Fatigue, poor technique, over distance, poor equipment)*
			• Anabolic steroids

Table 1: Predisposing Factors Related to Achilles tendinopathy.

*ABO blood types are associated with spontaneous tendon ruptures.

**If the muscle is weak or fatigued, the energy-absorbing capacity of the whole muscle-tendon unit is reduced, and the muscle no longer protects the tendon from strain injury

*** Overuse injuries: These are associated with vigorous physical activities. The occurrence of Achilles tendinopathy is therefore most frequently observed (upto 10% higher) among long distance runners, mountaineers, tennis and badminton players, footballers and soccer players.

Repetitive tendon strain may lead to cumulative fiber microtrauma. If the reparative capacity of the tendon tissue is exceeded, inflammation, edema, pain and tendon degeneration can ensue. In acute trauma, extrinsic factors predominate, while chronic overuse injuries are multi-factorial in origin with both extrinsic and intrinsic factors interplaying. However, the exact mechanisms by which these factors contribute to the pathogenesis of Achilles tendinopathy still remain ambiguous. An overuse injury caused by repetitive strain on the tendon can lead to loss of endurance and tensile strength. Consequently, tendon fibers begin to disrupt, resulting in pain and inflammation. Biomechanical orthopedic factors like limb length discrepancies as little as 6 to 10 mm can lead to tendon strain and clinical symptoms. Architectural defects, biomechanical misalignment of lower limb bones cause horizontal, axial, radial and rotational alterations on collagen fibers of the Achilles tendon. All of these can predispose towards tendon rupture. A discrepancy >20 mm has been observed as having significant correlation with Achilles tendinopathy [16]. However, the true magnitude of such biomechanical alterations is yet not well-known.

Muscle mass, tone, power, endurance, and flexibility are important for locomotion, and thus, can play a pivotal role in either prevention or acceleration of sports related tendon injuries. A weak or fatigued muscle can limit the energy and stress absorbing capacity for the whole 'muscle tendon' unit which in turn fails to protect the tendon from strain injury and subsequent inflammatory changes. This concept, however, is open to speculation because studies do not provide conclusive evidence on whether muscular factors precede Achilles tendon conditions or they are consequential to tendon injuries.

Extrinsic factors, like lower limb overuse, too much uphill or downhill work, extreme loading of the lower extremities and sports training errors; all lead to excessive strain and loss of tensile strength of the tendon [17]. Inability of the tendon to rebound back to its original state leads to microscopic degeneration and disruption of tendon fibers.

Extreme environmental conditions, such as too hot or too cold weather, hard grounds, slippery / icebound surfaces also have also been proposed as accelerating factors for Achilles tendon problems. But there is lack of objective evidence from long term prospective studies or reliable meta-analysis regarding the true extent of 'extrinsic risk factors' role in Achilles tendon disorders.

Spontaneous tendon ruptures are invariably related to sudden traumatic injury or long standing wasting changes in the tendon over an extended period of time. Such degenerative changes are often bilateral. Histopathologic evidence on ruptured Achilles tendons show hypoxic and mucoid degeneration, poor vascularity, tissue and cell necrosis, calcification, lipomatosis and irregularly arranged, degenerated collagen fibers.

Paradoxically, tendon "overuse" is not the only damaging force behind Achilles tendon conditions. Tendon "underuse" as in professional white collar jobs sedentary life style results in circulatory stasis, tendon hypoxia, degeneration and spontaneous tears [18].

Epidemiology of Achilles tendon disorders

During the past decade, with a general increase in popularity of modern world sporting activities, the incidence of Achilles tendon overuse injuries is on the rise. Complete, spontaneous ruptures have increased in industrialized countries [19]. The most common clinical diagnosis of Achilles tendon conditions lies under the umbrella of 'overuse injuries' or tendinopathy. Two main categories of Achilles tendinopathy classified according to anatomical location include 'insertional' (at the calcaneus-Achilles tendon junction) and 'non-insertional' tendinopathy (2 to 6 cm proximal to the insertion of the Achilles tendon into the calcaneus).

There are clear degenerative changes in the tendon before actual rupture occurs [18]; but these changes are often so subtle and a-symptomatic that many Achilles tendon ruptures take place suddenly without any preceding warning signs [14,15].

This disorder is more likely among amateur or professional athletes who participate in running and jumping. Up to 9% of recreational runners may be affected by the condition and up to 5% of professional athletes minght end up loosing their careers owing to the condition [17].

In a large cohort of 1394 nonathletes, during 2012; Waldecke and his team documented the presence of Achilles tendinopathy in 9 % of subjects out of which 4% were insertional type, 3.6% noninsertional type and 1.9% of both forms [17].

In another research by Kvist; 25% of Achilles tendinopathy cases had insertional disorder, 66% had noninsertional, and 23% had retrocalcaneal bursitis or or insertional tendinopathy [17,18].

Chronic tendinopathy is common among the old as compared to the young people. It rarely occurs in the 'younger than 14' age group owing to the naturally sturdy physique of youth [19]. Insertional tendinopathy tends to occur in more active persons, while noninsertional tendon injury are more common in the older, less active, and overweight group [19,20].

Management of Achilles tendon conditions

Non-operative Management: In the acute phase, initial rest is the most important; use of braces, immobilization cast or pneumatic walking boot should be combined with reduced activity. Immobilization controls worsening of the condition. But prolonged immobilization should be avoided to prevent degenerative changes. Specific exercises are recommended. Most patients are able to return to previous activity levels after few weeks. Orthotics (graduated shoe raise, heel lift, insoles) may be helpful in cases with identifiable biomechanical malalignment. Otherwise, braces or splints do not appear to improve outcomes in Achilles tendinopathy [21].

Ultrasound based physio therapy is often suggested to relieve pain, improve motility, reduce inflammation and promote healing. It has been suggested that ultrasound could stimulate collagen synthesis. However, there is lack of high quality evidence on the role of ultrasound to support its clinical use in Achilles tendinopathy.

Low level laser therapy has been suggested to reduce proinflammatory markers such as interleukins and tumor necrosis factor while stimulating tenocyte proliferation and collagen production. It also appears to decrease the capillary flow of neovascularization, and reserve resistance and elasticity of the tendon. But laser

therapy should be given in guided dosages. Further researches and good objective evidence is needed to validate the 'suggested' useful effects of laser in Achilles tendinopathy [22].

Non Steroidal Anti Inflammatory Drugs (NSAIDS) Since Achilles tendinopathy shows minimal inflammatory changes in the tendon itself; NSAIDs have limited use (only in the acute phase) in terms of reliving pain and reducing leg stiffness. The over all effect of NSAIDs in determining clinical outcomes of Achilles tendinopathy is debatable [23].

Corticosteroid injections reduce pain, swelling and inflammation. But these effects are local and short acting [23]. In long term, steroids induce lyses of tendon, peritendon adhesions and nociceptor alterations. Other more serious adverse effects of steroids include tendon atrophy, rupture and decreased tendon strength. Therefore, the symptomatic benefits of corticosteroid injections far outweigh the potential risks [17]. Many systematic reviews state that eccentric exercises are beneficial in the early treatment of non-insertional Achilles tendinopathy.

Eccentric exercise

The exact mechanism of how exercise works is not well understood. Theoretically, eccentric exercise serves in reducing pain and improving healing process through improved circulation, strengthening of calf muscle and lengthening of myotendinous unit [24]. The tensile force generated within the tendon during the exercise reduces neovascularization. Over time, the neo-vessels are obliterated, along with their associated neo-nerves and pain receptors, which lead to the relief of symptoms.

The harms of exercise differ among subjects and include muscle soreness, exacerbation of tendinopathy and muscle injuries if sufficient recovery period is not allowed and muscle is already weak or fatigued. Therefore, exercise should be supervised by trained health personnel, for maintaining correct biomechanics and regulated loading.

Extracorporeal shockwave therapy presents with conflicting results for Achilles tendinopathy management. It has been suggested to cause alteration of dorsal root ganglia, selective dysfunction of unmyelinated sensory nerve fibers, initiation of tendon regeneration and an overall promotion of healing response and pain reduction. High level efficacy of Extracorporeal shockwave therapy in chronic Achilles tendinopathy [25] has been confirmed in recent randomized placebo-controlled trials. However, determining the most effective dosage and duration of shock wave therapy still remains a subject of clinical discussion.

Platelet-rich plasma injections at the tendon injury site have been hypothesized to facilitate healing as it is rich in several different growth factors and cytokines that increase the expression of collagen and stimulate healing of soft tissues [26]. However, controlled trials demonstrating tendon healing with or without plasma injections show controversial clinical results. Therefore, evidence supporting the use of platelet plasma in the management of Achilles tendinopathy is inadequate (level I), till the benefits are validated more strongly in future level III researches.

Intratendinous hyperosmolar dextrose (prolotherapy) could reduce 'pain at rest' and during 'tendon-loading' in patients with chronic tendinopathy. Also, with dextrose, a reduction in the size and severity of hypoechoic regions and intratendinous tears has been observed [27].

Cryotherapy might play a role in moderating heightened capillary blood flow, reducing metabolic rate of the tendon and pain relief but available evidence is not concrete.

Sclerosing agents Some researchers suggest the use of sclerosing agents to be injected at multiple sites around the tendon to induce fibroblast proliferation and collagen synthesis. Therefore, a stronger, more organized tendon might be expected by using such regimes [28].

Aprotinin injection Some studies have documented Aprotinin injection showing improved clinical results in Achilles tendinopathy. Aprotinin is a broad spectrum serine protease inhibitor and speeds up the healing process but allergic reactions to Aprotinin are common [29].

Deep friction massage coupled with tendon mobilization and stretching has also been suggested as being helpful in the treatment of Achilles tendinopathy and paratendinopathy. 'Friction' raises the tendon's protein output while 'stretching' helps re-establish tissue elasticity and an overall strain reduction in the muscle-tendon unit [30].

However, all of these unique treatment modalities are prone to speculation and lack strong level III evidence of improved clinical outcomes.

Operative management

Non-insertional achilles tendinopathy

Surgery involves resection of degenerated tissue to stimulate healing by encouraging the body's natural homeostatic restorative mechanisms through controlled, low-grade trauma. Often, tendon grafts full or partial or whole tendon reconstruction is done; de-

pending on the extent of damage and chances for functional recovery. Many 'tried and testsed ortho clinics' have suggested that noninvasive treatment should be tried for up to 4months before resorting to any surgical type of intervention [31].

Conventional surgical treatment involves open release of adhesions with or without resection of the paratendon. Augmentation and restoration is suggested in cases with more than half of tendon tissue eroded or debrided [31]. Operative treatment modalities vary according to the extent of damage. The intervention may range from minimal invasive tendon stripping, open tenosynovectomies, percutaneous longitudinal tenotomies, open debridement and tubularization, and tendon augmentation with flexor hallucis longus

Long-term outcomes of operative interventions are encouraging as accounted by notable level 1 studies but level 3 researches are still needed to provide a more fair evidence in favour of surgery of Achilles tendinopathy.

Complications for operative treatment of Achilles tendinopathy [32] are a common occurrence as would be the case in any other surgical procedure. Some common complications include wound necrosis, superimposed infection, sural nerve injury, thrombosis, haematoma, seroma etc. The complications are defined in further detail in table 2. The overall morbidity rate in terms of post-operative complication has been roughly estimated as 11% and consequent re-operation rate as 3% [32].

Defining postoperative complications in Achilles tendon rupture	
Infection	Clinical signs of wound infection, i.e. redness, swelling, pain and functional impairment.
Deep infection	Infection beyond skin or subcutaneous fat needing surgical treatment in the operating theatre
Disturbed wound healing	Keloid formation or hypertrophic scar, secondary wound healing, protruding PDS knot.
Sural nerve injury	Any sign of altered sensibility in the sural nerve area diagnosed by surgeon or surgical resident (using touch and pin prick test).
Scar adhesion	Clinical signs of adhesion of skin to underlying tissue layers. Clear wound retraction at ankle movement
Deep vein thrombosis	Clinical and ultrasonographic signs of deep vein thrombosis of the ipsilateral lower leg.

Table 2: Defining Postoperative complications in Achilles tendon rupture.

Insertional Achilles Tendinopathy

This approach is retained for patients who do not respond to conservative treatment. Most clinicians consider a 3 to 6 months waiting and observing period before deciding if the conservative approach has failed and an operative intervention is mandatory [19]. Surgery for insertional tendinopathy includes the following: removal of deteriorating tendon and calcified areas, removal of inflamed retrocalcaneal bursa, resection of the posterior calcaneal prominence and reattachment of the insertion as required. Most of these procedures can now be performed endoscopically. Available clinical data suggests that up to 50% of damaged tendon can be safely debrided or detached without compromising strength of insertion or risk of rerupture [20]. However, greater than 50% tendon debriment or detachment from calcaneus, needs suture anchors to re-attach and support the residual tendon. In worsening cases with extensive disease or when greater than 75% of tendon is excised; augmentation is advisable with a tendon transfer/graft (usually with flexor halluces longus tendon and semitendinosus tendon).

Operative Treatment of Achilles Tendon Ruptures: Is it superior to Nonoperative Treatment?

Multiple studies have strived to determine whether 'operative' or 'nonoperative' treatment of Achilles tendon ruptures leads to superior outcomes. A trend observed during recent years suggests a steady decline in the number of Achilles tendon ruptures that are treated operatively [33]. Also, an increase in non-operative treatment has been observed. Surrounding this shift in treatment paradigm of Achilles tendon injuries lies a paradoxical evidence from studies that have demonstrated 'no significant difference' in tendon strength or re-rupture rates in 'operative' versus 'nonoperative treatment' when accompanied by an accelerated rehabilitation protocol [33]. Conversely; some researchers quote a significant increase in re-rupture rates of nonoperatively treated Achilles tendon injuries [32-34].

Till date, the clinical community has not been able to reach a consensus on the 'standard benchmark treatment' of Achilles tendon ruptures. As generally known; operative treatment of Achilles tendon ruptures decreases re-rupture rates and might allow earlier return to work. But surgery also increases the risk for certain complications like haemorrhage, wound infection, fibrosis, adhesions, bio-mechanical foot alterations, motion range limitations, sural nerve damage, altered sensations, deep vein thrombosis, scarring and kleoids etc.

The American Academy of Orthopaedic Surgeons (AAOS) released their Clinical Practice Guidelines in 2010, making weak recommendations for treating acute Achilles tendon rupture either operatively or non-operatively while also highlighting 'lack

of good evidence' surrounding this subject. The AAOS-Clinical Practice Guidelines also unanimously cautioned against higher probability of potential postoperative complications and wound issues in diabetics, smokers, neuropathy andimmune compromised poor wound healing conditions. These recommendations suggest that surgery is an option for decreasing re-rupture rates in Achilles tendinopathy; but postoperative complications must be carefully weighed out. The 'risk versus benefit' may subjectively differ among patients and the decision to operate or to give conservative management lies with the best judgement of the practicing clinician.

Mc Mohan., *et al*. [33] compared approaches for Achilles tendon rupture repair and the outcomes of 'open' versus 'percutaneous minimally invasive' surgical repair. The results showed no significant differences in tendon re-rupture rates, tissue adhesion formation, and deep infection.

Superficial infection is a frequent complication afflicting patients undergoing surgical treatment. The minimal invasive percutaneous technique offers much lower rates of superficial wound infections. Hense, percutaneous techniques must be considered as a first line of management for Achilles tendon ruptures. Similarly, the ability to bear weight (when and to what extent?) must be objectively outlined in non-operative groups.

The research by Zhao., *et al*, in 2011 [34] concluded that patients who undertook surgery had lower tendon re-rupture rates and benefited from shorter 'work return' period but they also exhibited a higher incidence of minor/ moderate complications. However, regarding an overall 'major' or 'total' complications assessment among the two groups; no significant differences were recorded.

A double blind randomized controlled trial by Barfod., *et al*. in 2014 [35] compared 'immediate weight bearing' after injury versus 'no weight bearing' in cases of ankle orthosis for upto 6 weeks postoperative. They found no difference in functional outcomes between the two groups. Therefore, early weightbearing could be an acceptable aspect while framing management algorithms for non-operative treatment of Achilles ruptures.

Soroceanu., *et al*. [36] compared three groups of Achilles tendon rupture patients in 2012. They noted that patients in the "operative group" had lower re-rupture rates than the "non-operative group -without functional rehabilitation" while the "nonoperative group-with functional rehabilitation" exhibited no difference in re-rupture rates. This validates the pivotal role of rehabilitation exercises in conservatively manages cases. Also, there was no difference in strength or calf circumference among all three groups.

Another notable study by Van der [38] and his team interestingly observed no difference in re-rupture rates between the operative versus non operative group.

A point of clinical debate

The discordance of postoperative functional outcomes among various studies opens a field for further evidence based exploration regarding 'non-operative' versus 'operative' treatment of Achilles tendon ruptures.

With passing time and recent advances in the medical field, rehabilitation protocols, weight bearing instructions and surgical techniques, have evolved. Majority of modern studies document that functional rehabilitation 'does not' increase the rate of re-rupture or other complications and may infact lead to lower tendon re-rupture rates.

If the primary goal of the clinician is to decrease the re-rupture rate, most current evidence favors surgical treatment over nonsurgical treatment. But, if the goal is to prevent post-operative complications (other than re-rupture); then the evidence tips in favour of nonoperative/conservative management.

Hence, in patients with Achilles tendon rupture; the treatment decision should be personalized and individualized. Patients with high activity levels or challenging jobs may gamble their chances with post-operative complications rather than suffering from inevitable and cumbersome re-rupture. Others, who are less active or those with comorbidities may choose the non-invasive route to alleviate the burde of infection, adhesions, and other post-operative problems.

The "take home" message

Variable conservative and surgical treatment options for Achilles tendinopathy are available. But there is no 'gold standard of treatment' due to controversial clinical outcomes documented by numerous studies. Observations, suggestions and recommendations vary widely between numerous studies. We still need high quality evidence based investigations that could standardize outcome scores to evaluate patients after operative or nonoperative treatment. This would make for easier comparisons between studies and help guide universally standardized treatment regimes. In a nutshell; the only conclusive statement that can be made at this point in time is that operative treatment decreases re-rupture rates and allows earlier work resumption but it also inevitably increases surgical complications when compared to nonoperative treatment. In the future, more level III researches will be needed to prove the effect of these treatment options.

Rehabilitation: 'Early functional rehabilitation' versus 'traditional immobilization' for Achilles tendon repair after acute rupture?

Achilles tendon rupture has an overall incidence of 18 per 100,000 per year. Although conservative treatment has its advantages, surgical intervention is treatment of choice for athletes and young people. Traditionally, surgical management of acute Achilles tendon rupture was combined with ankle immobilization for 6 weeks. The customary rehabilitation protocol involved rigid 'below-knee non-weight bearing cast' immobilization, followed by mobilization of ankle joint and strengthening exercises later. Until late 1980's, this remained the standard approach.

Over recent years, early postoperative functional rehabilitation has been increasingly recommended. Clinical trials using early postoperative ankle mobilization and functional rehabilitation have shown lower re-rupture rates [38,39]. However, the Best Practice Guidelines for postoperative rehabilitation in acute Achilles tendon rupture still remains disputable as multiple randomized controlled trials comparing 'early functional rehabilitation' with 'cast immobilization' have led to conflicting outcomes [40]. This could be due to myriad confounding factors ranging from non homogeneous study designs, different cohorts, operative and post operative care differences, non standardized surgical protocols, different data analysis tools used etc.

The Cochrane database of systematic review is internationally recognized as "the highest standard" in evidence-based health care resources and is updated every two years to provide the most current evidence to decision makers. Based on cochraine derived 'best practice data' from high quality meta-analysis using Jadad decision algorithms. (Jia-Guo., *et al*. 2017. www. nature. com/ scientificreports/); we can say that the scale still tips in favour of 'early functional rehabilitation and mobilization'.

The explanation offered for such observation lies in the pathophysiology of Achilles tendon healing. The healing process is divided into three phases: inflammation, proliferation and remodelling. During these phases, the tensile strength of the tendon gradually increases. However, the new scar tissue still remains biomechanically inferior; resulting in increased stiffness and limited visco-elastic properties. Animal studies involving early rehabilitation limits excessive adhesion formation, improves biomechanical properties of the scar tissue and successively enhances tendon gliding function. Ankle joint motion exercises remove local oedema, improve tissue perfusion and prevent joint stiffness and disuse atrophy of calf muscles.

Re-rupture following surgical repair is a probable complication for early locomotion and many surgeons argue that early function may increase re-rupture rate [37]. Nevertheless, the results of high standard studies demonstrate that early functional rehabilitation after Achilles tendon repair do not increase the re-rupture rate. Rather,it improves patient satisfaction and facilitates earlier return to activity [38-40]. Several randomized control trials and superior meta-analysis comparing 'early functional rehabilitation' versus 'traditional immobilization' following surgical repair of acute tendon rupture are available. Existing evidence suggests that 'early functional rehabilitation' is superior to 'cast immobilization' [24,36-38] in terms of patient satisfaction and 'pre-morbid activity' return time period. No significant differences have been observed regarding 'triggering of major complications' or the 'employment / sports return' time. Thus, early functional rehabilitation is suggested as the preferred post-operative strategy for acute Achilles tendon ruptures.

McCormack R and is team,in 2015, [39] published in the British Journal of Sports Medicine certain noteworthy points regarding rehabilitation practices adoped in Achilles tendinopathy cases. They performed high quality intervention meta-analysis on 10 randomised controlled trials over 570 patients comparing "Early postoperative ankle motion and weight bearing" (bracing rehab) versus "Traditional ankle immobilization with a non-weight bearing rigid cast" (cast rehab). Clinical outcomes between the 'bracing group' and the 'cast group' following surgical repair of acute tendon rupture; showed that early functional rehabilitation was safer, with higher patient satisfaction and less complications rate as compared to traditional ankle immobilization, with a non-weight bearing cast.

Adopting the best practice approach considering 'Risks' versus 'Benefits

Therefore,based on available best evidence, adopting the safest approach considering 'Risks' versus 'Benefits'; our recommendations for best clinical practice guidelines emerge as follows:

As compared to cast immobilization(casting), early functional rehabilitation (bracing)

- Is safe.
- Results in higher patient satisfaction.
- Leads to earlier return to function.

How might it impact clinical practice?

It is safe to start functional rehabilitation early following Achilles tendon repair.

- Early functional rehabilitation can improve patient satisfaction and facilitate earlier return to activity following Achilles tendon repair.
- Postoperative immobilisation is not necessary or helpful.

Conclusion

Achilles tendinopathy is not just a 'foot condition'; rather, it is a clinical syndrome characterized by a combination of morbid symptoms including lower limb pain, swelling, limited motion, joint performance and impaired daily life activities. Etiology of Achilles tendinopathy is multifactorial and both extrinsic and intrinsic factors play intertwined roles. The tendon shows deteriorative changes at the cellular level; with high glycosaminoglycans concentration in ground matrix and an increased number of tenocytes. The mainstay of strength and endurance of any tendon in the human body is the strategic alignment of healthy collagen fibres. In Achilles tendinopathy; this very foundation is disturbed and there is disorganization and fragmentation of collagen fibrils, followed by compensatory neovascularization and neo innervation. However, these new blood vessels or nerves neither improve perfusion nor sensation. Conversely, they pave a derogatory path for further damage in the form of bleeding points, hematomas and sensory disturbances. The source of pain in Achilles tendinopathy is multifactorial and its pathophysiological mechanisms are complicated and difficult to understand. Management of Achilles tendinopathy is variable; depending on the condition of the patient and the best clinical judgement of the doctor based on 'risk versus benefit' evidence available from medical literature. Conservative treatment usually precedes surgical options for routine cases. However, there is yet no 'gold standard' treatment manifesto for Achilles tendinopathy because of contradictory clinical outcomes between various studies done on different cohorts. It still remains a point of debate as to 'whether' and 'to what extent' race, genes, age, occupation and gender play a role in incidence, progress and clinical outcomes of Achilles tendinopathy. In future, more level III researches are needed to substantiate the effect of these interrelated factors on each other during the course and management of this condition. Futuristic researches could focus on whether it is time to incorporate genes mapping in preventive and rehabilitative programs of Achilles tendinopathy.

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