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Review

Integrating Tendinous Pathophysiology Into Rotator Cuff Tears And Greater Trochanteric Pain Syndrome: A Narrative Review

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Abstract

This narrative review aims to use the similarities between the shoulder and hip joints to better understand why rotator cuff (RC) tendinopathy and hip abductor tendinopathy occur and inform about diagnosis and treatment of both orthopedic complaints. A search of the literature was conducted using GoogleScholar and Pubmed and initially followed a systematic review protocol, but the nature of the topic, current literature and data necessitated a narrative review. Reports that discussed pathomechanics of RC and gluteal tendinopathy individually, together and with other muscles groups were reviewed. It was found that the methods measuring and describing the processes of tendinopathy differ significantly, for each individual joint and between all joints. A review of a large body of quantifiable measures and theoretical ideas regarding tendinopathy was performed to address this lack of consensus in current literature. Initial literature yielded 74 articles. After review, only 43 articles were used from a broad range of approaches and methodologies. The review found a body of evidence suggesting that fibrocartilage overgrowth and compressive forces over bony structures cause tendonous pathology of the RC and hip abductor tendons. These findings support the idea that tendinopathy is often caused by intrinsic factors rather than the traditional view of external factors. Earlier treatment and improved outcomes without surgery are possible using current imaging technology to identify these intrinsic factors that affect tendinous properties.

Introduction

The similarities between the musculature of the shoulder joint and the hip joint have long been acknowledged. The tendons surrounding the hip joint are often referred to as the "rotator cuff of the hip".[1–4] This comparison is fitting because of how similar the joints are in anatomy, pathology, and treatment. The muscles that of the rotator cuff(RC) of the shoulder and hip abductors that have drawn the most comparison include the subscapularis, supraspinatus, infraspinatus, teres minor, gluteus medius and gluteus minimus. Current literature is replete with connections between these muscle groups.

The RC has been more thoroughly studied than the hip abductors primarily due to the prevalence of RC injury. Tears of the hip abductors are often discussed in terms of findings in RC literature. Treatments for hip abductor injury and lateral hip pain are even discussed in terms of effectiveness in the RC rather than the gluteus medius or minimus.[2] Lateral hip pain affects 1.8 out of 1000 patients annually in the United States.[5] For many years, pain of the lateral hip has been poorly understood resulting in a diagnosis of trochanteric bursitis without further clinical investigation.[2] Recent research suggests that the differential is far more broad. Greater trochanteric pain syndrome(GTPS) has gained traction as one of the newly considered diagnosis for lateral hip pain and in some studies is considered more prevalent than Trochanteric bursitis.[2,6,7] In one particular study, MRI was used to confirm diagnosis of trochanteric bursitis in 24 female patients. It found that 45.8% of patients had gluteus medius tears and 62.5% had isolated gluteus medius tendinopathy with no findings of trochanteric bursitis[8]. As a result of many studies like this, trochanteric bursitis is no longer accepted as the principal cause of lateral hip. It is now just a part of the broad differential of lateral hip pain[8,9]. Causes of lateral hip pain are often injuries that lead to hip abductor tears . Hip abductor tears are due to degenerative wear that occurs commonly in adults over 40 years old, more commonly in women, and rarely occur because of trauma.[5,10] In exploring the role of GTPS in hip abductor pathophysiology we can start to understand its similarity to RC tendinopathy.

In orthopedics, pain of the RC and limited shoulder range of motion are among the most common complaints and the more heavily studied conditions. It is currently considered the third most common musculoskeletal complaints in the united states.[11,12] RC cuff based complaints have a lifetime prevalence of 67% in the Unites States and are also highly age dependent with a global prevalence between 5-10% in patients 20 years old or younger and above 60% in those older than 80 years old (77).

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Despite its prevalence its pathophysiology is still not well defined. Dr. Charles Neer first described impingement of the supraspinatus muscles in 1972. He proposed that extrinsic acromial impingement was the principal cause of the symptoms experienced so commonly and recommended acromioplasty and coracoacromial ligament release as treatment.[13,14] At the time, his idea was well accepted and followed but has since been challenged. In fact, many recent studies suggest that rotator cuff tendinopathy and eventual tears have very little to do with acromial impingement but rather due to intrinsic impingement and apoptosis.[13–18] Extrinsic acromial impingement as the cause of RC tendinopathy is further questioned by a 2012 report from the American Academy of Orthopedic Surgeons that declared "routine acromioplasty is not required at the time of rotator cuff repair".[19]

Describing RC injury or gluteus medius/minimus tear in terms of impingement or trochanteric bursitis limits the thinking of researchers and physicians alike.[14] Arguments and evidence in support of the prevalence of acromial impingement and trochanteric bursitis are still present but the frequency and impact of these arguments has since diminished. The original insights have led to new theories and ideas surrounding tendinopathy and tears of the hip abductors and RC. Researching these gaps in current literature is important to providing the highest quality of care based upon the most accurate understanding of these complaints. The aim of this paper is to review current literature on the anatomy, pathology, and biomechanics of each muscle group to understand the cause of both sets of symptoms more fully. Elaborating on the causes of these orthopedic complains can lead to better treatment and as a result better patient outcomes.

Methods

A comprehensive search was conducted using two electronic databases, Google Scholar and PubMed, to identify articles related to the pathomechanics of RC and gluteal tendinopathy. The search terms included keywords such as "Rotator Cuff," "Gluteal Tendinopathy," "Greater Trochanteric Pain Syndrome," and "Hip Abductor Tendons." Articles that discussed the pathomechanics of RC and gluteal tendinopathy individually, together, and with other muscle groups were included in the review. The initial search results were screened by title and abstract for relevance and yielded 74 articles by two authors. These articles were then reviewed in detail with a focus on identifying the causes of tendonous pathology in the RC and hip abductor tendons. After a thorough screening process, 43 articles were used to provide the insights found in this review. These articles were from a broad range of approaches, methodologies and demographics, including biomechanical studies, clinical trials, and case reports. When necessary, articles were critically evaluated to determine the quality of evidence presented and to extract relevant data. Not all papers were used for statistical reasons but rather for insight and theories in order to collect a broad range of ideas to consider in this narrative review. The nature of the topic demanded this approach given its limited current research. Scale for the Assessment of Narrative Review Articles (SANRA) was used to guide this review.

Results

Anatomical Comparison of the Hip and Shoulder Joint Musculature

The supraspinatus originates from the supraspinous fossa. It crosses underneath the acromion, passes over the glenoid and bicipital groove to attach to the greater tuberosity on the anterior lateral side of the humerus deep to the deltoid. It is a primary initiator of abduction of the shoulder. Gluteus minimus originates from the area of the ilium between the anterior and inferior gluteal lines. Gluteus medius originates from the area of the ilium between the anterior and inferior gluteal lines. The gluteus medius inserts into the lateral and superior aspect of the greater trochanter. Gluteus minimus inserts onto the anterolateral aspect of the greater trochanter located deep to the Iliotibial band band/tensor fascia lata. Both muscles function to abduct and medially rotate the hip are the primary muscles of concern for tendinous degeneration. Both tendons course posterior medially to inferior lateral and attach just millimeters after taking a sharp turn over bony prominence. This is one of the major contributors to the pathology of these muscles ***Table 1***.

Infraspinatus and teres minor attach proximally to the infraspinous fossa. The infraspinatus courses superior to medial and the teres minor courses laterally. The infraspinatus inserts on the greater tuberosity inferior and posterior to the supraspinatus insertion. It shares fibrous insertion with the supraspinatus. For this reason, the infraspinatus tendon is also of concern for RC based injury. The teres minor inserts on the greater tubercle just inferior to the infraspinatus insertion. The piriformis originates from the pelvic surface of the sacrum and inserts on the superior border of the greater trochanter. All three of these muscles function to externally rotate their respective joints.

The subscapularis originates at the subscapular fossa and inserts onto the lesser tuberosity of the humerus. The Iliopsoas is composed of the psoas and the iliacus which originate at the transverse process and vertebral bodies of T12-L5 and the iliac crest respectively. These muscles join as a common tendon that attaches onto the lesser trochanter. Both muscles originate posterior from their insertion. All these muscles function to internally rotate the joint.

The long head of the biceps brachii originates at the supraglenoid tubercle and inserts at the radial tuberosity. Its proximal tendon courses through the bicipital groove as it crosses the glenohumeral joint. It most prominently flexes the arm but also functions to flex the shoulder. The rectus femoris originates at the anterior inferior iliac spine and inserts onto the tibial tuberosity via the patellar tendon. This muscle primarily flexes the hip and extends the knee. Both muscles are unique because they course anteriorly across two joints and influence motion of both.

The deltoid originates from the distal third of the clavicle, the acromion and the spine of the scapula. All three parts of the

muscle converge to form the deltoid tendon and insert on the deltoid tuberosity of the humerus. The tensor fascia lata originates at the anterior superior iliac spine and anterior one third of the outer lip of the iliac crest. Parts of the gluteus maximus fuse with the tensor fascia lata to form the iliotibial band which inserts on the lateral aspect of the tibia via the iliotibial tract. These muscles are the most superficial and lateral of their respective joints and serve as a means of compression over the RC and hip abductors respectively. This results in higher compressive forces over the tendons.

Table 1. **Comparing Function of Shoulder and Hip Joint Musculature.**

Hip	Rotator Cuff	Function
Gluteus medius/gluteus minimus	Supraspinatus	Abduction/stabilizer
Iliopsoas*	Subscapularis	Internal rotator
Piriformis*	Infraspinatus/ teres minor	External rotator
Rectus femoris	Long head of biceps brachii	Crosses over the anterior portion of both joints of the limb**
Iliotibial band/tensor fascia lata	Deltoid	Overlies the bony prominence

Legend: *One of many. **In the lower extremity this includes the knee and the hip, in the upper extremity this includes the elbow and shoulder. Shoulder and hip joints have muscles that are identical in function, directionality, and contribution to pathology of the joint.

Biomechanics

Tendons are naturally developed to withstand large tensile loads but there is little known about their ability to withstand compressive forces. Tendons are so strong in resisting tears from tensile loads that tears occur at the muscle tendon junction before the bony insertion under extreme testing conditions.[20] The supraspinatus and the gluteus medius tendons are wrapped over the greater tuberosity and the greater trochanter respectively until attaching to their insertions. To contract, both muscles must transmit forces nonlinearly from attachment to insertion and exert force onto the joint. This vector relationship is not uncommon anatomically but what sets these tendons apart is the extreme change in direction that occurs just before insertion and the large forces exerted through muscles. Compressive forces alone do not result in pathology. This is because of the bursa that is between the bone and tendon. Rather, compressive forces can be a catalyst when combined with excessive tensile forces.

The nature of the gluteus medius tendon differs based upon insertion location. The anterolateral insertion is identified as the thinnest portion and the most prone to injury and tendinopathy.[1,9,21–24] Gluteus medius tears are mostly degenerative in nature, starting as undersurface tears at the anterior border of the lateral facet footprint and progressing posteriorly to become full-thickness tears.[25] The supraspinatus tendon is also split into anterior and posterior portions. The anterior portion has a higher modulus of elasticity and still more prone to pathology.[26] Tears at both joints primarily occur on the undersurface of the tendons.[6] The tendinous tissue along the bony prominence is stretched less compared to the more superficial side during contraction and therefore experiences less tensile load but experiences a greater compressive force along the bony prominence. Differences in tensile load along cross-sectional areas of the tendon is often referred to as stress shielding.[10,27,28] This is further intensified by increased iltiotibial band tightness exerting that exerts pressure onto the greater trochanter over the gluteal tendons. The same scenario is seen with acromial pressure on the tendon of the RC. Hyperadducted positions are implicated in the pathology of both joints because of the increased force down onto bone when abduction is performed exiting hyperadducted positions. Abducting from an acute angle of the tendon over the bone produces more downward force than in a larger angle over the bone.[10,29–31] Due to these differences in the nature of the forces exerted on the tissue they take on different morphology within the tendon. This is the main thought as the leading cause for these tendinopathies.[20,27,30–34] One of the major issues in current literature is whether these challenges are pathological or adaptive to these tendons.

Metaplastic Changes in Fibrocartilage

According to a review done by Cook et al. and findings in Benjamin et al., tendinous tissue that inserts onto the bony prominences are considered fibrocartilage.[27,33] Tendons under influence of compressive forces undergo metaplasia consisting of a transition from type I collagen to type II collagen, large proteoglycan aggregates, and neurovascular infiltration. All of these changes lead to tissue with increased capacity to withstand compressive forces. These metaplastic changes present new questions such as whether these changes are natural to all tendons in response to compression or are they specific to certain tendons and do they ultimately have a negative impact.

The review presented by Cook et al. reports these changes as native to the tissue. Rather than being a pathological response, fibrocartilage is just a subset of tendons specific to areas under compression. The principal theory proffered by Cook et al. to explain the damage that leads to tendinopathy is that compressive forces lead to reduced water content of the tendon which is essential to its ability to withstand compressive forces.[27] Grigg et al. shared research showing decreased tendon thickness in tendinopathy of the Achilles tendon that supports this idea.[35] This research is a good foundation for supporting this theory but will need further application to the gluteus medius and rotator cuff.

Benjamin et al. support the idea that this form of tissue is native to "wrap around tendons".[33] This suggests that these metaplastic changes occur strictly in wrap around tendons. Regardless of this, they still question whether this process is pathological or adaptive in nature.[33,34] Another important observation made by Cook et al. is that the bony prominence that the Achilles tendon rubs on is in close proximity to the tendinous attachment to the calcaneus.[27] This is of concern because the tendon is undergoing a transition from normal tendon to that which attaches to the bone with fundamentally different types of tissue. They briefly describe how

the proximity of these two zones and increased fibrocartilage metaplasia causes instability and tearing of the tendon. This expansion of fibrocartilage outside of its normal zone is supported more previous reports because tears occur primarily in the tendon in proximity to the enthesis, this insight will be elaborated on later in this review.[20,36]

Continuum of Tendinous Pathology

Cellular and extracellular intratendonal differences have been investigated since 1978 when Gillard et al. found that tendons adapt to compressive and tensile loads.[37] To prove this they surgically detached tendon from over a bony prominence to an insertion with a direct course and observed a gradual loss of the fibrocartilage tissue. Afterwards they reattached the tissue and saw an increase in fibrocartilage. This is evidence of an adaptive measure by tendons to compensate for compressive forces but is very outdated and may need reevaluation. Even the model of tendon morphology between normal, fibrocartilage and pathology used by Cook et al. is from a paper written in 1996.[27]

As in most physiological processes, tendinopathy is considered a process that fluctuates along a continuum of presentations based upon more recent models.[36,38] Cook et al. presents tendinopathy in three stages and the initial stage of reactive tendinopathy fits those pathological stages elaborated on already.[38] Namely, compression leading to a change in composition of the tissue with higher type II collagen and increased proteoglycans. The second stage is tendon disrepair which is characterized by greater matrix breakdown and hyperplasia with chondrocytes and myofibroblasts resulting in increased protein production including proteoglycan and collagen deposition causing decreased organization in the matrix. The last stage is degenerative tendinopathy. This stage includes areas of cell death due to apoptosis and trauma or tenocyte exhaustion. There is little capacity for reversibility of pathology at this stage.

In comparison, Macdonald et al. argue that the first stage of tendinopathy is not a characteristic of adaptation but rather a pathological reaction to acute overload or stress.[15] Cook et al. describe this process as acute and easily reversible which implies it to be a normal response to new stimuli within the tendon. They even call it a response to compression but do not consider it in the context of wrap around tendons like the RC of the gluteus medius.[38]

In a follow up report, Cook et al. suggest that all models of tendinopathy can be divided into three categories.[34] The category most in line with pathology of the RC and gluteus medius is the "tendon cell response" model. It views tendinopathy as a direct result of tenocyte activation in response to compression. It displays the exact morphology of those mentioned in development of fibrocartilage in wrap around tendons. The problem they find is knowing when these changes go from healthy adaptation to damaging pathology. They cite a study using the Achilles tendon in Australian football players that shows this adaptation as a healthy part of increased tendon load and use but the study also showed 3 of the 18 of the participants progressed to a painful tendinopathy which complicated their conclusion of normal adaptation.[39]

Tendon Cell Response Model - RC and Gluteus Medius

Direct evidence of the tendon cell response model in the RC and gluteus tendons is minimal. Almekinders et al. share evidence about the gluteal tendon being thinner in pathological states but does not comment on its compositional makeup.[20] Allison et al. elaborates on muscle atrophy and weakening in gluteal tendons but they are unsure if the atrophy is a result of the tendinopathy or if the atrophy comes before the tendinopathy.[31] Another model mentioned by Cook at al. explains tendinopathy as part of a lack of electrical stimulation from chronic damage that is correlated with atrophy of muscle. This model is not investigated in a particular muscle, but it is broadly applied to all muscle tendinopathy, so knowing how this may apply to the RC or gluteal tendons is conjectural. This draws into question whether the tendinopathy indirectly results in reduced muscle function. Allison et al. also consider disuse atrophy from patients involuntarily reducing use of the muscle plays a role in causing the atrophy of muscle.[31]

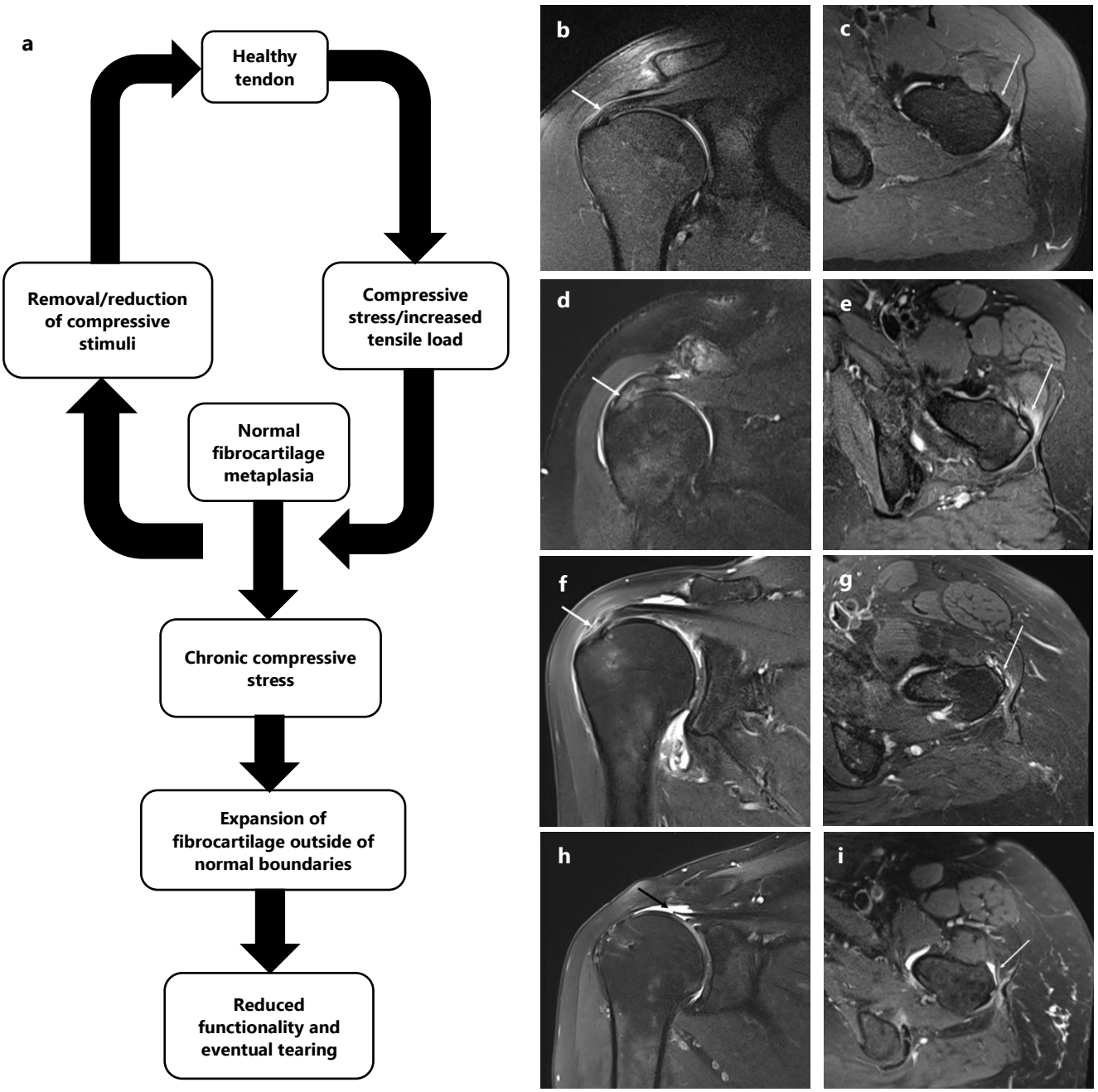
Strong evidence for compressive forces being the principal cause of gluteal tendinopathy is supported by the findings of a study investigating why females experience a much higher degree of gluteus medius tears.[29] They found that on average females have a much lower femoral neck shaft angle, giving them a slight coxa vara compared to males. This causes a more acute angle over which the gluteus medius passes over the greater trochanter which translates to greater compressive forces over the bony prominence.[10,29–31] Evidence suggests that this is the cause of higher rates of gluteus medius tears in adult females. In addition, repetitive movements into and out of hyper adduction are implicated in worse outcomes and symptoms for gluteal pathology. Contraction of the gluteal muscles in hyperadducted positions would maximize the force of the tendon onto the bone due to a lower angle of contraction between insertion and attachment as stated earlier in this review.[20,30,32,33]

In a study done by Soslowsky et al. tensile and modulus testing was performed on rat supraspinatus where they compared repetitive tensile loads used to simulate acromion impingement and overuse stress on the tendon.[40] They found that acromial impingement alone was not enough to induce tendinopathy but required both tensile overuse and impingement to replicate rotator cuff pathology. They also found that the thickness of the tendon and diameter of the tendon was significantly lower and the modulus was significantly lower. This supports the theory regarding dehydration of the tendon and is in line with lower modulus being a likely effect of these combined forces. They did not consider the impact of compressive forces intrinsic to the tendons as they considered them to be a non-physiological component of the supraspinatus.

Perhaps the greatest insight as to why these tendons ultimately fail has been shown from fibrocartilage metaplasia as expressed by Cook et al.[27] They demonstrated that tenocyte overactivity extends outside of the margins of fibrocartilage to zones of the tendon that are essential to sustaining tensile load and the transition into enthesis. This type of change requires destruction of existing tissue and deposition of new matrix and cellular

metaplasia. The expansion of these zones is detrimental to other essential functions of the tendons. Just as the expansion of the fibrocartilage is necessary, the expansion of normal tendinous tissue becomes a demand of the tendon as less normal tendon is present. These two essential tissue types and functions compete and ultimately decrease the overall functionality of the tendon leading to damage. The tendon is stuck between repair, metaplasia and proper function. Tissue consisting of proteoglycan and type II collagen along the bony surface and normal tendinous tissue proximal to the bony surface coexist within a microscopic section of tendon. This continuum of change and adaption with a single tendon ultimately limits the tendon's ability to function and repair as needed. Impaired function and repair leads to the damage that is seen in the RC and hip abductors and eventual tears *Figure1*.

Figure 1. **Progression of Rotator Cuff and Hip Abductor Tendinopathy.**



Legend: Side by side progression of tendinopathy of the RC and gluteus medius and minimus. Injuries to both tendons occurs on spectrum that is reversable at early stages but may lead to chronic complications. (a) Healthy tendons that are exposed to compressive and tensile stress compensate with fibrocartilaginous metaplasia. Metaplasia may be sufficient to avoid damage and tearing. Alternatively, removal of the stimuli will allow continued healthy function. Under chronic over exposure to compression and tensile load, metaplasia disrupts proper function of the tendon and leads to pain and tearing. (b) Coronal T2 fat suppressed image showing a normal supraspinatus tendon. (c) Axial T2 fat suppressed image showing a normal gluteus minimus tendon. (d) Coronal T2 fat suppressed image showing a supraspinatus tendinosis. (e) Axial T2 fat suppressed image showing tendinosis of the gluteus minimus tendon. (f) Coronal T2 fat suppressed image showing a severe supraspinatus tendinosis with a high-grade

partial-thickness bursal surface tear. (g) Axial T2 fat suppressed image showing tendinosis and high-grade partial-thickness tearing of the glutes minimus tendon. (h) Coronal T2 fat suppressed image showing a fill- thickness retracted re-tear of the supraspinatus tendon. (i) Axial T2 fat suppressed image showing a complete tear of the gluteus minimus tendon and underlying subgluteus minimums bursal fluid.

Discussion

This collection of evidence supports the idea that the source of damage to the tendons is due to intrinsic adaptation. It is likely that these changes are both pathological and adaptive. Fibrocartilage is an essential adaptation to compressional forces on the tendon but can become pathological as demonstrated in Figure 1. In situations that decrease the muscle angle in females, over abducting or in overstressing the tendon, fibrocartilage overgrows and ultimately leads to decreased functionality. The need to investigate these findings is especially important to the RC and gluteal tendon because these two tendons experience the greatest compressive forces, have no definitive explanation for the cause of their pathology and are common complaints among orthopedic patients. The gluteal tendon and RC are not a point of focus in the research that defined these insights. This leaves a gap in the knowledge of these two prevalent medical issues.

Clinical Implications

Patients who manifest an overgrowth of fibrocartilage could be treated at early stages to reduce overgrowth and the damage that occurs in chronic stages of this pathology. In detecting early morphological overgrowth, we can advise and treat the patient non-invasively before the tendon is damaged enough to necessitate surgical intervention. Treatments such as lifestyle and work modifications, physical therapy and pain management can slow progression in chronic stages. The limiting factor is the ability to determine the current morphological state of a tendon in vivo. The invasion of fibrocartilage into adjacent zones would be a matter of micrometers in difference and is not currently within the capabilities of modern imaging see ***Figure 1***.

One alternative to this would be to measure the tensile modulus, compressive modulus and thickness of the tendon over time. This relates to the idea of how the morphology changes the water content and ability of the tendon to withstand certain forces.[20,27,34–36,38,40] Tendon thickness would be indicative of certain stages of pathology. Early fibrocartilage induces water retention and tendon thickening. Later tendinous damage would manifest as a thinner tendon as water is poorly retained from over compression and damage. Ability to withstand tensile load would decrease as normal tendon is broken down in favor of fibrocartilage. This would manifest as a reduced tensile modulus.

Shear-wave elastography (SWE) and other ultrasonographic techniques are used currently to measure these very parameters in tendons.[41–45] SWE is widely available but rarely used in clinical practice. These methods of imaging enable physicians to avoid invasive and expensive imaging in favor of measures that tell us about the morphological state of the tissue. The principal measure that is relevant to tendinopathy is tendon shear elastic modulus otherwise known as stiffness. The higher stiffness in the tendon, the less metaplastic change has occurred in the tendon. Dirrichs et al. used intraindividual and interindividual comparisons of tendinopathies to compare SWE, ultrasound and power doppler.[41] This study showed that SWE significantly improved the identification of painful tendinopathy across a wide age group 20-71years old and different tendons including Achilles, patellar and humeral epicondylar tendons.

Zhang et al. compared the patellar tendon stiffness and cross-sectional area of sedentary and active 18-35 year old men.[45] They found a significant decrease in stiffness and increase in cross-sectional area in those who were more active and putting increased stress on their tendons. The authors showed that the evaluated tendon demonstrated a normal response to tensile load. Taking this in light of findings in the healthy active population we can correlate this with a healthy metaplastic change in the tendons. The findings of Dirrichs et al. and Zhang et al. in some ways contradict each other but also support each other. The next step for this diagnostic tool is to use cross sectional area, stiffness and patient presentation can be used to create diagnostic criteria.

The next logical question to be addressed is whether these cross-sectional area and stiffness parameters lead to tendinopathy impacting RC and gluteal tendinopathy treatment. Performing experiments to correlate the tendon morphology to physical parameters and matching them to stages of pathology within patients could potentially change the patient experience and reduce the need for surgery in patients in the long term.

Limitations

This review is limited due to the nature of the topics and current literature available for comparison and analysis that necessitated a narrative review. It relies primarily on a collection of theories, ideas and anatomical understanding to build its argument. This can be a foundation for a discussion that can lead to many other research opportunities to test these theories spark innovation in orthopedic care of Hip tendinopathy and shoulder tendinopathy.

Conclusion

Theories on the root cause of RC and hip abductor tendinous degeneration have evolved over time. The anatomy, biomechanics and pathophysiology of these tendons is so similar that we can use them to further our understanding. In doing so is it apparent that increased tensile load and compression over bony prominences lead to metaplastic changes within the tendon. These changes are natural adaptive responses that become pathological with chronic overload of the tendon. The metaplastic changes decrease the capabilities of the joints to function normally and lead to tearing of the tendon. This process points to intrinsic factors of the tendons as being the principal cause of these complaints as opposed to extrinsic factors. These intrinsic factors can be measured using modern SWE techniques. In understanding and identifying early signs of degeneration that are intrinsic to RC and hip abductor tendons that were previously unknown it may be possible to slow or even stop the progression of tendon degeneration before a tear becomes evident.

Summary – Accelerating Translation

This study explores the similarities between the muscles in the shoulder and hip joints, and how this can provide a better understanding of the cellular and mechanical problems associated with Greater Trochanteric Pain Syndrome and Rotator Cuff Tendinopathy. This is a literature review and analysis of relevant insights and connections that were found applicable to the aims of this project. By comparing these two conditions along with current theories regarding of tendinopathy it is suspected that factors within the tendons themselves, rather than external factors, lead to pain and tearing. Shear Wave Elastography, a non-invasive ultrasound technique, can measure these intrinsic factors and can be used to test these theories. The study suggests that earlier and more affordable diagnosis and treatment of tendinopathy could be achieved by testing the insights of this review.

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