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CURRENT CONCEPTS REVIEW

SUPRASPINATUS TENDON PATHOMECHANICS: A CURRENT CONCEPTS REVIEW

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ABSTRACT

Background and Purpose: Tendinopathy of the supraspinatus muscle is a frequent cause of shoulder pain. Although it is a common condition, the pathophysiology is not fully understood. The purpose of this clinical commentary is to provide an overview of the pathophysiology of supraspinatus tendinopathy and discuss the conservative treatment solutions.

Description: Supraspinatus tendinopathy is thought to be caused by both intrinsic, and extrinsic factors. Structural and biological changes happen when tendinopathy develops. Cellular and extracellular modifications characterize tendon healing stages that continue over time. Assessment is paramount in order to differentiate the structure involved, and to offer a proper treatment solution.

Relation to Clinical Practice: Knowledge of the general concepts regarding the development of supraspinatus tendinopathy, and of the healing process should guide physiotherapists when proposing treatment options. Physical modalities commonly utilized for supraspinatus tendinopathy such as: laser, ultrasound, and shock-wave therapy have little and contradictory evidence. Exercise in form of eccentric training may be considered as it seems to have beneficial effects, however, more research is needed.

Key words: Rehabilitation, rotator cuff, shoulder

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BACKGROUND AND PURPOSE

Tendinopathy is a generic term that indicates a condition characterized by pain in and around a tendon associated with repetitive activities, and impaired function that happens when the healing process fails to properly regenerate the tendon.[1,2] Tendinopathies account for over 30% of musculoskeletal consultations, and shoulder pain is the third most common musculoskeletal complaint.[4]

Tendon injuries of the rotator cuff (RC) are among the most common problems of the shoulder,[5,6] affecting people performing sports as well as repetitive activities related to work or daily living.[7-9] Moreover, tendinopathies of the RC increase with aging[10] affecting more than 80% of the people over eighty years of age,[11] with the supraspinatus tendon being the most commonly affected.[12-14] Although supraspinatus tendinopathy is a frequent shoulder condition, to date a definitive understanding of the associated pathology remains elusive, and there is not agreement on treatment.[15] Therefore, the purpose of this clinical commentary is to provide an overview of the pathophysiology of supraspinatus tendinopathy and discuss the conservative treatment solutions.

STRUCTURE OF THE SUPRASPINATUS TENDON

Along with the subscapularis, teres minor, and infraspinatus muscles, the supraspinatus joins to form the RC which functions to compresses the head of the humerus into the glenoid fossa of the scapula.[16] The supraspinatus muscle originates from the posterior aspect of the scapula, superior to the scapular spine and inserts on the greater tuberosity of the humerus, blending partially with the tendon of the infraspinatus muscle.[17,18] The tendon of the supraspinatus muscle is a specialized nonhomogeneous structure subjected to both compressive, and tensile forces.[19] Moreover, in order to better resist compression, and to lubricate collagen bundles during shoulder movements, there is an increased number of glycosaminoglycans within the supraspinatus tendon when compared with the distal region of the biceps tendon.[20]

Structurally, the supraspinatus muscle consists of two different sub-regions: anterior and posterior. Anterior muscle fiber bundles are bipennate, with a thick and tubular tendon while the posterior counterpart has a more parallel fiber bundle orientation, with a thin, and strap-like tendon.[21] These two sub-regions have different mechanical properties,[22] with the loading stress being higher in the anterior sub-region.[21] Studies have shown that the different regions of the supraspinatus muscle move independently to each other.[23] Additionally, these two sub-regions can be divided into three parts: superficial, middle, and deep. This division is generally associated with the development of supraspinatus tears.[24]

Anatomically, the insertional supraspinatus tendon is divided into four transitional layered zones according to the extracellular matrix (ECM) content.[25] The first zone is essentially made up of Type I collagen and a small amount of decorin. This zone may be considered the tendon proper. The second zone consists of mainly Types II and III collagen, with small amounts of Types I, IX, and X collagen forming fibrocartilage. The third zone is defined by a mineralized fibrocartilage which is composed by Types II and X collagen, and aggrecan. The fourth zone is formed by Type I collagen, with the collagen fiber orientation defining the effective bone-tendon attachment.[19]

As in other tendons of the human body, histological changes such as: vascular, cellular, and extracellular matrix modifications have been recurrently found also in supraspinatus tendonopathies.[19,26,27]

ETIOLOGY OF SUPRASPINATUS TENDINOPATHY

The model explaining tendinopathy development has been changing over the years. Currently, it is generally accepted that supraspinatus tendinopathy develops when excessive stresses exceed the healing capacity of the tendon cells (tenocytes),[28] with the tendon failing to repair properly.[2]

Supraspinatus tendon disorders have been classically described as degenerative processes starting from an acute tendinitis, progressing to tendinosis, and eventually resulting in partial or full thickness tendon rupture.[29-31] However, currently the terms tendinitis and tendinosis should be avoided and the word tendinopathy should be preferred as recent research shows that there are minimal or no inflammatory

cells in painful tendons.[32-38] To date, it appears that tendon disorders arise from a variety of different etiologic factors.[39] Lesions of the supraspinatus tendon seem to start where the loads are thought to be the greatest, in other words, at the articular surface of the anterior insertion on the humerus.[40-45] Excessive mechanical loads at the supraspinatus tendon insertion have been thought to cause an increased rate of collagen synthesis and turnover that are often related to tendon tears and ruptures.[26] Although supraspinatus tendinopathy etiology is still poorly understood, several intrinsic and extrinsic factors have been theorized as contributors to the development of supraspinatus tendinopathies.[46,47]

INTRINSIC FACTORS

Age has been shown to correlate with tendinopathy,[48,49] having a negative impact on tendon properties, especially after forty years of age.[10] With age, tendons tend to degrade,[50] decrease the ability to withstand tensile loads, and elasticity.[51,52] Furthermore, in a study with subjects of fifty-two years of age and older, Kumanagai et al[53] have shown that calcifications and fibrovascular proliferation changes, as well as a drop in total glycosaminoglycan and proteoglycan content are present in the aging supraspinatus tendon.[54]

Vascularization supply related to healing of supraspinatus tendinopathy has been investigated with contradictory results.[29,52,55-58] Although Codman[52] identified an area with poor blood supply within the supraspinatus tendon in chronic RC tendinopathies, and in small RC tendon tears, there is evidence of abundant neovascularization[36,53,59-63] that may crowd out necessary collagen, weakening the tendon properties.[64] Interestingly, the neovascularization, and the increased blood flow in tendinopathies seem to normalize during the course of conservative exercise-based treatments.[65] Structural tendon adaptation, tendon length changes, neuro-chemical alterations, fluid movement, neuro-muscular adaptations, and neuro-vascular ingrowth have been proposed as mechanisms of the beneficial effects of exercise training in tendinopathies.[66] There seems to be a genetic component as a factor for tendinopathies,[67,68] related to the occurrence of different forms of collagen genes.[69] In addition, there is a higher risk of developing supraspinatus tendinopathy in patients whose siblings sustained RC tears,[70] and in males.[38]

EXTRINSIC FACTORS

Extrinsic factors responsible for supraspinatus tendinopathy are all those anatomical and biomechanical alterations that eventually result in narrowing of the subacromial space.[71-73] Impingement syndrome as the mechanical compression of the RC tendons is thought to be one of the most important reasons for supraspinatus tendinopathy[40,41] and seems to be affected by acromial shape, acromial angle, and the presence of acromio-clavicular spurs.[47,74] However, in a recent randomized surgical trial, the efficacy of surgery to reduce shoulder impingement by improving subacromial space (shoulder decompression) has been questioned as it does not appear to provide clinically significant benefits compared to arthroscopy only or no surgery.[75]

Posterior capsule tightness may cause an anterior-superior migration of the humeral head which may alter the gleno humeral arthokinematics, leading to reduction of sub-acromial space, and shoulder impingement.[4,76-80] Although still under debate, changes in scapular kinematics have been linked to supraspinatus and RC tendinopathy[4,81-83] as well as strength deficits,[84,85] and postural alterations.[86,87] To date, it is widely agreed upon that tendinopathies are pathological processes originating from several factors rather than a single specific cause.[39]

TENDON CHANGES IN TENDINOPATHY

Several different cells types make up the tendon cell population. Tenocytes are the most represented cell type in tendons[88] and are responsible for maintenance of tendons' health as they produce collagen and ECM secretion.[89-91] They have a round shape at the fibrocatilaginous regions that becomes more elongated within the tensile-load-bearing regions in its tendon mid-substance.[92] Additionally, there are synovial-like cells, smooth muscle cells, and endothelial cells associated with blood vessels.[93]

The ECM is a complex structural entity that surrounds the tendon cells, providing the ability to the tendon to resist mechanical loads, influencing the viscoelastic properties, and assisting in healing from injury.[94,95] It is formed by structural proteins

Table 1. *Histological changes associated with supraspinatus tendinopathy.*

Cell/Tissue/Structure Involved	Description of change(s)
Tendon cells	Tenocytes become rounder [57,105,106] Increase in cells number[59,106] Chondroid metaplasia[28] Cellular apoptosis[107-109]
Extracellular matrix	Fatty infiltration/degeneration[36] Loss of matrix organization[26,36,106]
Vascularity	Increased vascularization[29,36,53,55-64]
General/Other	Increased number of small nerves[36,97-99] Increased nociceptive neurotransmitters[100-104] Presence of inflammatory cells/mediators[113-114] Hypoxia[37]

(collagen and elastin), specialized proteins (fibrillin and fibronectin), and proteoglycans.[96]

With the development of tendinopathy there are changes that seem to appear consistently. (Table 1) Generally, there is hypoxia,[37] an increased number of small nerves,[36,97-99] increased nociceptive substances and neurotransmitters such as substance P, and glutamate.[100-104] Tenocytes tend to lose their native shape,[57,105,106] become narcotic/apoptotic, assuming a fibrochondrogenic phenotype, and growing in number.[28,36,59,106-109] Specifically, Scott *et al* [28] found that in an animal model supraspinatus tenocytes become more chondroid and demonstrated increased proliferation as a result of an injury.

With regard to the ECM, there is a decrease in the collagen content, an increased ratio of type III/type I collagen, thinning of the collagen fibers, hyaline degeneration, chondroid metaplasia, and fatty infiltration.[36,106] Frequently, there is also an increased presence of hyaluronan, and chondroitin dermatan sulfate.[26] Additionally, Riley *et al*[110] have shown that in RC tendinopathy there is increased collagenase (MMP1) activity correlated to reduced gelatinase (MMP2), and stromelysin (MMP3) activity. This suggests a high level of collagen turnover that may be an adaptive response to the mechanical demands.[110]

Some debate still exists on the presence or absence of inflammatory cells in tendinopathy.[111] Some authors indicate that there are no inflammatory cells in degenerative tendons[32-38,46,92,112] while others have reported presence of inflammatory cytokines with the development of tendinopathy.[113,114] Macroscopically, tendinopathic tendons tend to present with an irregular gray/brown color, thin, soft and fragile crux in contrast to the brilliant white colored, and firm fibroelastic normal tendon.[115]

TENDON HEALING

Tendon injuries heal because of scar tissue processes that may last from twelve[88] up to twenty-four months.[116] However, the final repaired tissue differs from the native tissue, with a higher concentration of type III collagen, and a lower concentration of type I collagen, resulting in a lower tensile strength.[2,117,118]

Classically, scar formation goes through a three-phase healing process that starts off with an inflammation phase followed by a reparative phase, ending with a remodeling phase.[119] Inflammation deploys during the first seven days from injury, with a high activity of phagocytes, and initiation of type III collagen synthesis.[120,122] After a few days, and for up to six weeks, growth factors enhance cellular proliferation and type III collagen is gradually replaced by type I collagen which is thought to have stronger tensile properties.[120,121] As a result of fibers getting larger and with an improved interdigitation, the scar tissue becomes stronger as the healing process proceeds.[2] At approximately the sixth week, the remodeling phase commences, with the fibrils becoming aligned along the direction of the mechanical loads, improving the cross linking.[122] Thus, in about a year, the repaired tissue will have a scar-like appearance, and a stiff consistency.[89] Supraspinatus tendinopathy healing seems to follow this general tissue repair process even when there is no overt tendon fiber rupture. This means that other factors such as: blood perfusion, microscopic fiber damage, or other

unknown aspects may be key to influencing tendon healing.[26]

EXAMINATION AND ASSESSMENT FOR SUPRASPINATUS TENDINOPATHY

Patient history is paramount when considering the presence or absence of supraspinatus tendinopathy. Questions regarding aggravating/easing factors, duration of symptoms, physical activities, and general medical conditions should always be included. A self-reported questionnaire such as the Shoulder Pain and Disability Index (SPADI) may be utilized to monitor progression of pain, and functioning.[123] At the physical examination, pain commonly presents in the arc of motion between 60° and 120° of shoulder abduction/scapular plane abduction,[124] but does not tend to radiate.[31] Since the clinician should differentiate from other structures, provocation tests that load tendon fibers should be utilized.[31] These test are generally performed in the form of resisted abductions with the shoulder in internal or external rotation (empty/full can tests).[125] A positive lag test should alert the practitioner regarding the possibility of a total supraspinatus tear.[126]Imaging may be helpful for a correct diagnosis as supraspinatus tendinopathy generally occurs concomitant to other shoulder disorders.[127,128] Magnetic resonance imaging and ultrasound may be used to visualize the supraspinatus tendon, helping make more accurate diagnosis[129,130] as they may provide information regarding fatty of the tendon infiltration that are recognized signs of chronic tendon tears.[131]

CONSERVATIVE TREATMENT OPTIONS

Knowledge of pathophysiology, tissue properties, and tissue healing process are key factors when developing a targeted and safe rehabilitation program. Although a singular accepted treatment for supraspinatus tendinopathy has not been agreed upon, treatment solutions traditionally consist of anti-inflammatory drugs, rest, stretching, and strengthening exercises.[115]

The role of inflammation continues to be a point of controversy for intervention related to tendinoapthy.[111] Some authors who have investigated a variety of human tendons indicate that there are no inflammatory cells in degenerative tendons[32-38,46,92,112] while others have reported an increased presence of inflammatory cells in pathological tendons.[114,114,132] Therefore, corticosteroid injections, and non-corticosteroid anti-inflammatory drugs should be carefully utilized for pain relief, and for a limited time[1,133,134] as chronic tendinopathies are mostly degenerative in nature, and as such, corticosteroids may have adverse effects on tendon healing.[135-138] Complete immobilization of the tendon should be avoided as it may cause a protein synthesis reduction, an increase in collagenase activity,[138] and a catabolic biological response.[139-141]

Since alterations of upper trapezius/lower trapezius, and upper trapezius/middle trapezius ratios,[142] shoulder kinematics,[143,144] and posterior capsule tightness[4,76,80,145] have been associated with many shoulder disorders, correction of posterior shoulder tightness and restoration of glenohumeral joint and scapular kinematics are encouraged.[146] Such interventions illustrate the important role of the physical therapist in conservative management of movement system dysfunction that may be associated with supraspinatus tendinopathy.

Therapeutic modalities commonly utilized for tendinopathies may help limit or reverse the degenerative process of tendinopathy by improving repair processes,[115] and by reducing the expression of neovascularity often associated to tendon symptoms.[147] Laser therapy seems to be beneficial[148] and superior to therapeutic ultrasound.[149] However, the evidence regarding the effects of the various modalities adopted to date including therapeutic ultrasound, laser, and extracorporeal shock-wave therapy on supraspinatus tendinopathy is limited, and often contradictory.[150-153]

Mechanical loading is essential for tendon homeostasis, repair,[88,154-157] and for prevention of the negative effects of immobilization.[158,159] Graduated tendon loading in the form of isometric, concentric, and eccentric exercises should be considered in the rehabilitation program. Appropriate loading forces induce a tensile stretch to tenocytes, and activate protein kineases.[160] Moreover, stretching techniques if applied correctly (generally 30 second holds for three repetitions with 30 seconds between repetitions)[161] may help the collagen turnover of the

tendon,[115] and may promote an anabolic response.[162] The addition of manual therapies such as: friction massage, scapular and gleno-humeral mobilization, proprioceptive neuromuscular facilitation, and nerve gliding/sliding techniques seem to be beneficial for decreasing pain and improving range of motion.[163] However the evidence regarding the use of exercise therapy and manual therapy on RC tendinopathies is limited.[1,164,165]

Over the last several years, exercise in form of eccentric training has been advocated for use with a variety of tendinopathies including Achilles, patellar, wrist extensors, and rotator cuff, with good clinical outcomes.[31,166-169] Skillful dosage and implementation of eccentric loading interventions illustrate the important role of the physical therapist in management of tendinopathy with the goal of long term positive functional outcomes. Eccentric exercise consists of muscular contractions where the muscle, and the tendon, are activated while decelerating a mechanical load. If applied gradually over a period of time, these muscular contractions should generate high forces over the tendon fibers, driving biological responses within the tendon, cueing remodeling of the tendon internal architecture.[157] This process is also known as mechanotherapy or mechanotransduction.[170] Some authors have evaluated the effects of eccentric training in patients with supraspinatus tendinopathy, and shoulder disorders with promising results on pain.[167,171-173] However, due to the lack of a complete understanding of tendinopathy processes and the effects of mechanotransduction, eccentric training of the supraspinatus needs to be further investigated in terms of optimal dosage, frequency of treatment, and load progression.

CONCLUSIONS

Supraspinatus tendinopathy is a common shoulder disorder that requires further research in order to have a better understanding of pathophysiology. There is still a lack of evidence regarding the effectiveness of treatment options currently being utilized. Eccentric training may represent an appropriate, inexpensive, and easy-to-perform solution for treatment of supraspinatus tendinopathy; however, more research is warranted.

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