

Tendinosis

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Tendinitis has been proven to be a misnomer for several reasons. There is a lack of inflammatory cells. Now Tendinosis is used which has a profound effect on how the condition is treated.

Histopathology of Tendinosis

A typical healthy tendon is composed of primarily type I collagen with minimal amounts of type III collagen interspersed within the neatly arranged parallel fiber orientation of type I collagen. The healthy tendon is red and shiny and reflects polarized light under a microscope. The key findings in tendinosis are; 1) disrupted fibers within the tendon 2) Increased cellularity 3) neovascularization.

Kraushaar and Nirschl found that on cross section of collagen in an area of tendinosis showed that the collagen was of variable diameter, uneven mixture of thick and thin fibrils and in some areas did not even connect with each other to form a tendinosis structure. They concluded that the ultrasound of collagen in tendinosis is actually type III collagen, instead of the type I in a healthy tendon. The increase in type III collagen, and possible decrease in type I results in a decrease in the forces that the tendon can withstand and may eventually lead to a tendon rupture.

The other two findings present in tendinosis, increased cellularity and neovascularization has been termed angiofibroblastic hyperplasia by Nirschl. The cells present in tendinosis are mesenchyme-derived tendon fibroblasts (tenocytes) and myofibroblasts, as opposed to the humoral process of an immune based inflammatory response. These cells represent a degenerative condition.

Neovascularization found in tendinosis has been described as a haphazard arrangement of new blood vessels and Kraushaar even mentioned that the vascular structures do not function as blood vessels. Vessels have been found to form perpendicular to the collagen. Therefore increased vascularity present in this condition is not linked with increased healing.

Pathogenesis

Micro trauma seems to be the most widely accepted cause. Kraushaar describes four stages of micro trauma.

- 1) Inflammatory process that resolves
- 2) Angiofibroblastic degeneration
- 3) Structural failure
- 4) Mixture of 2/3 with calcification and osseous calcification

There also is said to be a tendinosis cycle that must be broken in order for treatment to be successful and pain-free movement can be restored. The cycle is simplified as repetitive overuse activities or microtraumas causing microtears in the tendon creating a fibroblastic hyperplasia response, which then respond by increased amount of type III collagen within the tendon. This further weakens the tendon causing more microtears and prolongs the degenerative cycle.

Treatment for this condition must first attempt to stop the degenerative cycle and then to restore proper collagen synthesis, strength and function.

Conservative Management

Krushaar lists the primary treatment of tendinosis as being pain control, preservation of motion, flexibility and strength, and the development of endurance over time. Since the exact cause of pain in tendinosis is not known, many theories have tried to explain the mechanism of pain. In order to improve treatment.

Traditional treatments involve the use of corticosteroid injections for the treatment of tendonitis. It has been proven that corticosteroid injections actually further the degeneration of tendons and increase the risk of recurrence of the condition as well as increase the risk of tendon rupture. That being said, corticosteroid injections have been shown to be effective in short term pain reduction for tendinosis. The mechanism is unclear but it is thought that bathing the area may alter or interfere with local chemicals that cause the pain stimulus in the area.

Other treatments include reduction of biomechanical stress on the tendon, relative rest, and ice since it is a vasoconstrictor (and increased vascularity is a finding in tendinosis) and a natural analgesic. Research for the use of modalities such as laser and therapeutic ultrasound is controversial at best, however it is recommended by certain sources to help stimulate collagen synthesis. Stretching and strengthening are two common methods of treating tendinosis and eccentric strengthening is gaining popularity quite rapidly.

Khan states that eccentric loading results in a stimulation of mechanoreceptors in tenocytes to produce collagen and thereby help reverse the tendinosis cycle. Alfredson used heavy load eccentric calf muscle training in the treatment of chronic Achilles tendinosis and found that all 15 participants were back to their pre-injury activities after 12 weeks. They performed 15 reps, with both the knees straight and knee bent, twice daily for 12 weeks. They did not perform the concentric portion on the injured leg, they relied on the uninjured leg to return them to the starting position.

A more recent study by Shalabi found that eccentric training of the gastrocnemius-soleus complex resulted in a decreased tendon volume and intratendinous signal when evaluated by MRI. They also concluded that the findings were associated with decreased pain scores on a VAS and is therefore an effective adjunct in the treatment of Achilles Tendinopathies.

Stretching and tendon fibroblast response

Li et al. investigated the effect of cyclic stretching of human patellar tendon fibroblasts (HPTFs). They were interested in the outcome of stretching on the expression of 5-lipoxygenase (5-LO) and the production of leukotriene B4 (LTB4). They also investigated the effect of blocking the COX pathway would have of expression on LTB4 and if blocking the 5-LO pathway lead to an increase of the COX pathway. Results of their study found that cyclic stretching of HPTFs increases production of LTB4 and increase 5-LO activity.

Stretching of HPTFs results in increase of the COX expression and thereby increasing prostaglandin E2 (PGE2). These are steps in the pro-inflammatory pathway. Another mediator of the pro-inflammatory pathway is LTB4. LTB4 is increased by the 5-LO enzyme that converts AA to LTB4. Results showed that both of those pathways are present with cyclic stretching of HPTFs which causes inflammation and possible injury to tendons.

Clinical management of inflammatory and even non-inflammatory tendonous conditions is often done by corticosteroid injections and/or NSAIDs. NSAIDs work by inhibiting the COX enzyme to decrease the production of pro-inflammatory cellular mediators. However, this study has shown that by inhibiting the COX enzymes, and thereby decreasing the amounts of PGE2, there is actually a reciprocal increase in the production of LTB4 which is also a pro-inflammatory cellular mediator. Therefore the conclusions of this paper show that taking NSAIDs for tendonous injuries can actually have a deleterious effect by increasing the production of LTB4 in greater quantities then the PGE2 is decreased via COX-inhibiting drugs.