

THE TENDINOPATHY CONTINUUM

Title: Is tendon pathology a continuum?

A pathology model to explain the clinical presentation of load-induced tendinopathy.

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Reference: British Journal of Sports Medicine 2009; 43; 409-416

INTRODUCTION

In the past 10 years research into tendon pathology has seen our understanding grow. The new term “tendinopathy” has replaced the old term of “tendonitis” as a result of this research and a better understanding of pathophysiological mechanisms. What follows is a summary of this landmark paper on our developing knowledge of tendinopathy. I have attached a link to the full article which is well worth a read and another more recent article regarding the changeable feast that is tendinopathy research.

CLINICAL QUESTIONS

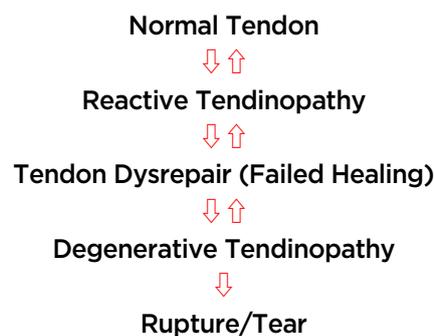
What is happening at the injured tendon site and is this common to all tendinopathy, and if not, how do we determine the type of tendinopathy and how is it best treated?

CAUSES OF TENDINOPATHY

Tendon injuries are usually the result of increased loads and overuse. This leads to changes within the tendon, which make it harder for it to cope. The injuries may occur in the mid-portion or, more commonly, in the insertion. At both sites the pathological changes of the tendon appear to be the same. Despite the common pathological changes within the tendon, different treatment approaches are used specific to the site of the problem, and this has been shown to have better outcomes.

THE CONTINUUM OF TENDINOPATHY

It is proposed that under periods of excessive load a tendon will pass through the following continuum:



REACTIVE TENDINOPATHY

Pathophysiology: This is a non-inflammatory proliferative response, which is a short-term adaptive thickening in an attempt to reduce stresses. This takes place in the short-term to allow the tendon to cope with loads through thickening and stiffening. The tendon can revert back to normal structure if overload is reduced or sufficient time is given between loading.

Imaging: will show fusiform swelling and increased diameter (US/MRI).

- US – diffuse hypoechogenicity.
- MRI – minimal or no increased signal.

Clinical Manifestations: Generally follows a period of acute overload (i.e. a burst of unaccustomed physical activity) or a direct blow. More common in the young athlete and produced through increases in training load or commencement in training if previously sedentary.

TENDON DYSREPAIR

Pathophysiology: Similar to reactive tendinopathy this is an attempt at tendon healing, however there is greater matrix breakdown. There is also possible increases in vascularity and associated neuronal ingrowth (neovascularisation).

Imaging: US and MRI will show increased matrix disorganisation and evident swelling.

Clinical Manifestations: This stage appears in chronically overloaded tendons. This has the potential to appear over a range of ages and loading environments. On examination, these tendons are thickened with more localised changes in one area of the tendon.

DEGENERATIVE TENDINOPATHY

Pathophysiology: In this stage there is progression of both matrix and cell changes, there are even some areas of cell death. Large areas of the matrix are disordered, filled with vessels (neovascularisation) and breakdown. Considerable heterogeneity exists between parts of the tendon.

Imaging: Extensive compromise of the tendon can be seen on US and MRI

Clinical Manifestations: More commonly seen in the older patient/athlete, but can also be seen in younger patients with a chronically overloaded tendon. Therefore, there is real potential to see this in a young, elite athlete. However, the more classic presentation is the middle-aged recreation athlete, with focal swelling and pain. They often describe repeated bouts of tendon pain. If allowed to progress this stage can inevitably lead to rupture. Analysis of ruptured tendons have shown these degenerative changes in 97% of cases.

IMPLICATIONS FOR TREATMENT

Pain has been shown to vary considerably at all stages along the continuum and therefore this will not aid in determining the stage of pathology. However, pain levels should be integrated with other clinical information to determine the stage of pathology in order to determine appropriate treatments. In general, increased loads will lead to increased pain and it is thus important to determine pain levels on assessment to enhance outcomes. To make things more challenging, variable stages of pathology may coexist in different localities on a single tendon.

If you identify the injury in the early stages (reactive tendinopathy or early tendon dysrepair) then load management/reduction will allow the tendon time to adapt and recover. This will result in lower levels of pain. In these stages if aggravating activities are continued and eccentric exercises are added without sufficient recovery, poorer outcomes will result. Also, use of NSAIDs may be beneficial at this stage, as their ability to impede healing can reduce abnormal adaptation.

In degenerative stages exercise appears to be a positive stimulus for tendon restructuring. Pain at this stage of the pathology has shown not to affect the overall outcomes achieved by exercise. This emphasises the importance of eccentric exercise treatments, despite pain response. Possible adjunct treatments, such as frictions and ultrasound, may be useful at this stage of pathology as their rationale is to stimulate cell activity. Prolotherapy, aprotinin, sclerosing therapy, glyceryl trinitrate and injection itself may all have theoretical implications in degenerative tendinopathy.

CONCLUSION

This model presents a clinical framework of tendinopathy that aids the sports physiotherapist to select more appropriate treatment options and will ultimately enhance the overall effectiveness of treatment programs and athlete outcomes.

Full article: [PDF](#) *Tendon Pathology Continuum - Cook & Purdam, 2008*

Other reading: [PDF](#) *Tendinopathy - Cook, 2012*

<http://bjsm.bmj.com/content/45/5/385.full>