Treating Tendinopathy

Perspective on Anti-inflammatory Intervention and Therapeutic Exercise

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KEYWORDS

• Tendinopathy • Anti-inflammatory • Eccentric loading • Therapeutic exercise

KEY POINTS

- Tendinopathy is the clinical term for tendon overuse injuries.
- Histopathologic examination frequently reveals a noninflammatory process underlying tendinosis; however, an inflammatory infiltrate has been demonstrated in pathologic supraspinatus, subscapularis, and Achilles tendons.
- There is a poor relationship between symptoms and the onset of tendon pathology; however, a continuum from early reactive tendinopathy to late degenerative tendinosis may be identified with clinical examination and imaging.
- Anti-inflammatory medication may have a role in the early stages of tendinosis; however, several deleterious effects have been noted, including increased risk of rupture, adipogenisis, and chondrogenesis.
- Mechanical loading through exercises inclusive of eccentrics and heavy slow resistance are efficacious in the treatment of Achilles and patellar tendinopathy.

INTRODUCTION AND TERMINOLOGY

The tendon transmits force developed through muscle contraction to bone, resulting in motion and joint stabilization. Tendon is inherently exposed to large magnitude loads and has the ability to adapt in form and behavior in response to loading history. The mechanism of tendon adaptation is largely through autocrine/paracrine signaling via mechanotransduction pathways.[1] Excessive or insufficient loading disrupts tissue homeostasis and is a primary factor in the development of tendon pathology. Diseased tendon is typified by cellular dysfunction and tissue alterations, leading to functional compromise. It is alarming that most tendon ruptures occur without warning symptoms, yet postrupture tendon shows signs of chronic degeneration.[2]

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Tendinosis is a degenerative condition with a notable absence of inflammatory cells and what has been characterized as a failed healing response.[3,4] Several studies have documented the absence of an inflammatory response in human ruptured tendons,[2,5] biopsy samples,[6] or surgical debridement samples.[7,8] A paradigm shift away from an inflammatory model of tendinitis has occurred and the treatment of tendon related symptoms reassessed in light of new understanding. Because the diagnosis of tendinosis requires histopathologic examination, the term tendinopathy is advocated for the clinical presentation of tendon pain, stiffness, and loss of function related to mechanical loading.[9]

A combination of intrinsic and extrinsic factors lead to the development of tendinopathy. Influential intrinsic factors favoring the development of tendinopathy as well as tendon rupture include advancing age and gender. Men are significantly more prone to both tendinopathy and rupture. Additionally, genetics (variations in genes for tissue and cellular proteins) and limb mechanics affect risk. Extrinsic factors such as activity level, footwear, training technique, and surface type have been implicated in both Achilles and patella tendinopathy. Loading history is likely a key component in the pathogenesis of tendinopathy, with a likely interaction with intrinsic factors.

The tendon adapts to mechanical load through alterations in composition and mechanical properties. There seems to be a threshold to overuse, and the quantification of appropriate load (volume, intensity, and frequency) for optimal tendon function remains elusive. Appropriate load is anabolic. Overloading has catabolic effects on the tendon tissue, as degradation of the extracellular matrix exceeds synthesis of new tissue. Seemingly contradictory is the benefit seen with eccentric exercise. At first glance, adding load to an overloaded tissue is counterintuitive. In a degenerative condition in which cell and tissue interactions are disrupted, stimulation of the tendon cell (tenocyte) through eccentric loading is likely anabolic and therefore advantageous. Achilles and patella tendinopathy are prevalent in an athletic population and respond well to loading programs.[5]

The purpose of this article is to review the pathogenesis of tendinopathy and discuss the possibility that inflammatory mediators are at work despite the absence of leukocytes in tissue samples. We review the role of anti-inflammatory medications in the management of tendinopathy and then describe effective loading programs for the management of Achilles and patella tendinopathy.

TENDINOSIS

Although viewed as a local lesion, tendinosis affects the entire tendon. Macroscopically, tendon darkens and loses its pearly white appearance.[10,11] The peritendinous and intratendinous tissue may be affected in isolation or concomitantly. The peritendinous tissue thickens and can cause adhesions, which may constrict the microvasculature.[10] A proliferation of fibroblasts and myofibroblasts in the paratenon is thought to be responsible.

Proliferation of neovascularity is often present, and some feel this vascular proliferation may be the pain generator of tendinopathic tendon.[12] It is important to note that a large percentage of tendinosis is asymptomatic, and 90% of spontaneous tendon ruptures can occur prior to any precipitating symptomatology. Underlying degenerative tissue is typically found.[2]

In a diseased tendon, collagen becomes disorganized, with visible microtearing, decreased density and fiber diameter, and loss of crimp. An increase in reparative type 3 collagen is noted. The alterations in organization and composition of collagen compromise tendon structural integrity.[13] Several variations of collagen degeneration have been described (hypoxic, mucoid, myxoid, hyaline, lipoid fibrocartilagenous, and