Chiropractic Management of Tendinopathy: A Literature Synthesis

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Abstract

Objective: Chronic tendon pathology is a soft tissue condition commonly seen in chiropractic practice. *Tendonitis*, *tendinosis*, and *tendinopathy* are terms used to describe this clinical entity. The purpose of this article is to review interventions commonly used by doctors of chiropractic when treating tendinopathy.

Methods: The Scientific Commission of the Council on Chiropractic Guidelines and Practice Parameters (CCGPP) was charged with developing literature syntheses, organized by anatomical region, to evaluate and report on the evidence base for chiropractic care. This article is the outcome of this charge. As part of the CCGPP process, preliminary drafts of these articles were posted on the CCGPP Web site www.ccgpp.org (2006-8) to allow for an open process and the broadest possible mechanism for stakeholder input. A literature search was performed using the PubMed; Cumulative Index to Nursing and Allied Health Literature; Index to Chiropractic Literature; Manual, Alternative, and Natural Therapy Index System; National Guidelines Clearinghouse; Database of Abstracts of Reviews of Effects; and Turning Research Into Practice databases. The inclusion criteria were manual therapies, spinal manipulation, mobilization, tendonitis, tendinopathy, tendinosis, cryotherapy, bracing, orthotics, massage, friction massage, transverse friction massage, electrical stimulation, acupuncture, exercise, eccentric exercise, laser, and therapeutic ultrasound.

Results: There is evidence that ultrasound therapy provides clinically important improvement in the treatment of calcific tendonitis. There is limited evidence of the benefit of manipulation and mobilization in the treatment of tendinopathy. Limited evidence exists to support the use of supervised exercise, eccentric exercise, friction massage, acupuncture, laser therapy, use of bracing, orthotics, and cryotherapy in the treatment of tendinopathy.

Conclusion: Chiropractors often provide a number of conservative interventions commonly used to treat tendinopathy. (J Manipulative Physiol Ther 2009;32:41-52)

Key Indexing Terms: *Manual Therapies; Manipulation, Spinal; Chiropractic; Tendinopathy; Cryotherapy; Braces; Orthotic Devices; Electrical Stimulation; Acupuncture; Exercise; Exercise Therapy; Laser Therapy, Low Level*

Chronic tendon pathology is a soft tissue condition commonly seen in chiropractic practice.[1] *Tendonitis*, *tendinosis*, and *tendinopathy* are terms used to describe the same clinical entity. Although colloquially known as *tendonitis*, this term is misleading because this condition has not been associated with inflammation.[2] Studies have to date been unable to appreciate any intratendinous acute inflammatory cells or inflammatory cascade. As such, rather than tendonitis or tendinosis, the preferred term for this condition is *tendinopathy*, as this term makes no etiopathologic implication.[3,4]

Some common tendinopathies include rotator cuff (eg, supraspinatus) tendinopathy, calcaneal or Achilles tendinopathy, lateral and medial epicondylopathy, patellar tendinopathy, and various wrist tendinopathies such as extensor carpi radialis tendinopathy. Other less common or uncommon tendinopathies have been documented, such as that of the longus colli[5] retropharyngeal prevertebral musculature,[6] iliopsoas,[7] quadratus femoris,[8] popliteus,[9] and the pes anserine.[10]

Illness Burden

These common tendon disorders place a burden on health care resources, particularly with regard to occupational and sports-related injuries.[11,12] In 2006, the US Department of Labor, Bureau of Statistics, showed that work-related musculoskeletal disorders, which include tendinopathies, were associated with increased time away from work.[13] The average number of lost time days for tendonitis has increased from 11 days in 2003 to 14 in 2006.[13] Bonde et al[14] reported

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Paper submitted May 6, 2008; in revised form August 21, 2008; accepted September 8, 2008.

0161-4754/$34.00

doi:10.1016/j.jmpt.2008.09.014

the duration of shoulder tendinopathy disability in Danish industrial service workers to be in the order of 10 months for 50% of people with the disability. In a Canadian study, Yassi et al[15] found that the most frequent upper limb diagnosis submitted to the worker's compensation board was tendonitis. They go on to report that claimants had symptoms for an average of 8 months before reporting the injury.[15]

Chronic disability is associated with higher health care and societal costs. Baldwin and Butler[16] examined the costs and outcomes after the initial return to work of an injured worker. They found that a substantial proportion (26%) of workers with cumulative trauma disorders, such as tendinopathies, experienced further injury-related absences after the initial return to work.[16] This may lead to underestimates in the overall costs of these injuries.

Histopathology

Tendons are a dense parallel-fibered collagenous connective tissue containing an organized fibrillar matrix.[17] The tendon matrix consists primarily of type I collagen, proteoglycans, and glycoproteins. Although type I collagen is predominant, other collagens may however also be present in lesser and varying amounts. The exact composition of each tendon differs based on its function, such as extremity tendons, which have a higher percentage of their dry weight made up of collagen.[18]

Tenocytes are fibroblast-like cells within the tendon and are responsible for tissue maintenance and matrix remodeling.[17] The structure of individual tendons is determined by tenocyte metabolism, which in turn may be influenced by factors such as biomechanical loading.[19]

Animal models of tendinopathy have shown changes in the resident tenocyte and the structure of the tendon with repeated loading. A recent animal model study by Scott and et al[20] found 4 diagnostic morphologic changes in rat supraspinatus tendinopathy. Those changes were fibroblastic alterations (hyper- or hypocellularity), increased glycosaminoglycan staining, collagen disorganization or disarray, and hypervascularity.[20] Supporting the hypothesis that tendinopathy is not inflammatory, they found no extrinsic cellular invasion in the tendinopathic rats.[20] They also found no evidence of apoptosis in the tendinopathy group.

Tenocyte morphology also changed. After repetitive loading, the tenocytes appeared to have a rounded chondrocytic appearance.[20] Other authors support this observation.[11,21] Furthermore, they suggest that tenocyte proliferation may be caused by an insulin-like growth factor 1 autocrine signaling response.[20]

Other tendinous changes have been noted. These changes include hypervasularity[22]; tendinous microtears[23]; increased type III collagen, fibronectin, tenascin-C, and matrix glycosaminoglycans[24]; increased expression of chondroitin sulfate proteoglycans, aggrecan, and biglycans[25]; increased water content; increased denatured collagen; upregulation of collagen type I and type III gene expression; increased metalloproteinase activity; and altered matrix metalloproteinase gene expression.[26] Metalloproteinase enzymes have been implicated, at least in part, in the cell-mediated changes seen in tendinopathy.[24]

Risk Factors

Biomechanical risk factors have been studied extensively. Tendons are suited to sustaining great tensile loads.[18] Other loads are not as well accommodated. Corps et al[25] found tendon changes in tendinopathy to be consistent with adaptive responses to shear or compression. Repetition and forceful exertion have also been implicated as causal factors in the development of tendinopathies.[11,20,27-29]

Personal risk factors include advancing age and obesity. Increasing age has been associated with increased risk of developing tendinopathy and delayed recovery.[14,27] Frey and Zamora[30] found that patients who were overweight or obese significantly increased their risk of developing "tendinitis" in general.

The role of genetics on the development of tendinopathies is currently being explored. The COL5A1 gene and the TNC gene have been identified in Achilles tendinopathy.[31,32] Type V collagen fiber assembly and diameter are associated with the COL5A1 gene.[31,32] The TNC gene encodes for tenascin-C, which is important in regulating the tendon's response to a mechanical load.[31,32]

Although biomechanical and histologic analyses have helped shed light on the etiopathogenesis of tendinopathy, disability due to this condition appears to be complex and multifactorial. Leclerc and et al[33] found that psychosomatic problems and social support at work were predictive of wrist "tendinitis." They also found that previous upper limb disorders and depressive symptoms predicted a first occurrence of lateral epicondylitis.[33] Other studies support the key role of psychosocial factors in tendinopathy severity and disability.[34-36] Therapies aimed at reducing this condition should take these factors into account.

Diagnosis

The onset of most tendinopathies is insidious. The pain is localized and described as "sharp" or "stabbing" with activity. Often there has been a history of a recent increase or change of activity that coincides with the onset of pain. The patient may report that the pain increases with activity but diminishes shortly after a warm-up period. This is most common early in the progression of this condition. Later, however, the patient may feel a "dull" or "achy" type of pain after activity or even at rest.

Provocative palpation of the tendon tends to reproduce the patient's pain in a well-localized pattern. Tests that load the tendon similarly to inciting activities can also recreate the patient's pain and help support the diagnosis.