

The Pathology & Healing of Tendinosis

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Tendonitis is a misnomer. With microtraumatic overuse injury, we are dealing most of the time with tendinosis. While the stages of pathology of the macrotraumatic inflammatory response are well-known, the tissue pathology of tendinosis needs more clarity.

In conditions such as tennis elbow, and overuse injuries in general, there is a fibroblastic and vascular response (tendinosis), rather than an immune blood-cell inflammatory response.¹ In a normal tendon, collagen fibrils are embedded in a matrix of proteoglycans, glycosaminoglycans and water, surrounded by a loose connective tissue containing visceral and parietal layers. Vascularized tendons (de Quervain, etc.) are surrounded by epitenon with capillaries and penetrating vessels.²

In tendinosis there is disorganized collagen, fibroblastic and vascular hyperplasia. Kraushaar and Nirschl,² with electron microscopy, showed abnormal collagen with an "uneven mixture of thick and thin fibrils." In regions of severe tendinosis, the collagen fibers did not show evidence of combining with or becoming normal tendon. Leadbetter¹ saw collagen fibers with microtears and signs of repair. The initial response in tendinosis is a fibroblastic hyperplasia whose job it is to produce collagen. Some of the fibroblasts return to the mesenchymal state and form cartilage cells, bone-forming cells and vascular endothelium; other fibroblasts develop chemotactic mobility and intracellular contractile elements.² "This process represents the intrinsic capacity of tendons to attempt to heal."² They blame the failure of healing on a lack of an effective vascular system.

Interestingly, the fibroblasts can use local chemical mediators to manufacture rudimentary blood vessels, i.e., angiogenesis. The basic question for manual treatment of soft tissue is that it can aid in the healing of an "osis." The authors of the article² state: "The presence of red blood cells inside the abnormal vessels found in regions of tendinosis suggests that vascular hyperplasia may lead to communication with an extrinsic healing response, provided that the immune system receives signals of a need for the healing process." They state that rehabilitation exercise can stimulate regional hyperemia and create cyclical tensile loads that stimulate the remodeling of collagen.

They suspect that extracellular alignment and crosslinking of collagen fails in tendinosis.³ A fibroblastic-driven process is expected to integrate old and new collagen to contribute to the final stability of the matrix.^{1,3} It is now proven that augmented soft tissue mobilization (ASTM) performed by Graston technique⁵ will stimulate fibroblastic proliferation.⁴ The absolute evidence of how a tendinosis can heal is not completely understood; as usual, the clinical benefits of a technique often precede the explanation.

A final point refers to the cause of pain in the noninflammatory tendinosis. Many practitioners immediately think of inflammation as the sole cause of pain. According to Leadbetter,² the pain of inflammation occurs in the macrotraumatic or synovial irritation; degenerative pain from excessive cyclic loading results in matrix molecular damage, loss of tissue strength and increased

deformation with loading, which causes pain due to the stimulation of mechanoreceptors. The structural changes that occur from abnormal mechanics (motion, vibration, fatigue) create biochemical and/or nutritional changes. The result is chemical irritation and immunologic factors that irritate the nociceptive sensors and cause pain. The pain may be related to the chemical characteristics of the matrix (pH, lactic acid and prostaglandins levels). Another possibility of the cause of pain in tendinosis is chemical irritation from regional anoxia, and the lack of phagocytic cells to remove noxious products of cellular activity, such as lactic acid.¹ This is noninflammatory pain - so why rush to treat with anti-inflammatories?

References

1. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). *J Bone & Jt. Surg* 1999. 81-A(2):259-278.
2. Leadbetter WB. Cell-matrix response in tendon injury. *Clin Sports Med* 1992;11(3):568-569.
3. Geiberman R, Goldberg V, An KN, Banes A. Tendon. In *Injury and Repair of the Musculoskeletal Soft Tissues: Workshop*. Savannah, Georgia, June 1987, pp1-40. edited by S.L-Y Woo and J.A. Buckwalter. Park Ridge, Ill. Amer Acad of Orthop Surg, 1988.
4. Davidson CJ, Ganion LR, Gehlsen G, et al. Rat tendon morphologic and functional changes resulting from soft tissue mobilization. *Medicine & Science in Sports & Exercise* 1997;29(3):313-319.
5. Hammer WI. Graston technique, a necessary piece of the puzzle. *Dynamic Chiropractic* 2001;19(20).

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APRIL 2002