

SOFT TISSUE / TRIGGER POINTS

Early Soft-Tissue Lesions May Be Causative of Osteoarthritis

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We constantly try to prove that chiropractic care is preventative and that maintenance visits are necessary. While the absolute proof of this premise is not yet substantiated, one area to evaluate is the effect of soft-tissue evaluation and treatment to prevent osteoarthritis.

We constantly read about injections of chondrocytes into knee joints and new "chondro-protective" drugs to treat joint cartilage. While treating the cartilage may be beneficial, most idiopathic primary

osteoarthritis does not occur because of an underlying cartilage defect.¹

Osteoarthritis cannot be considered a cartilage disease. The cartilage defect may be due to disease of any of the tissues around the diathrodial joint, such as the subchondral bone, synovium, capsule, periarticular muscles, sensory nerve endings, meniscus, supporting ligaments and especially the enthesis (i.e., the bony attachment of a tendon or ligament).

Slemenda, et al., found that the quadriceps weakness that is associated with osteoarthritis of the knee

may be a cause of the osteoarthritic knee.² Their studies showed that quadriceps weakness preceded osteoarthritis and may be pathogenic. They stated that quadriceps action serves as a brake, retarding the rate of the descent of the leg at the end of the swing phase. Quadriceps weakness could accelerate damage to articular cartilage in the knee. Patients with painless osteoarthritis that may appear on X-ray probably should be given quadriceps exercises as prevention. At the very least, everyone's quadriceps should be tested for strength.

Shrier hypothesized that when muscles are unable to contract adequately due to age, fatigue, disuse atrophy, decreased proprioception or strain, more force is transmitted to the bone, leading to

sclerosis.³ The microtrabecular damage of the bone and eventual sclerosis could create stress on the articular cartilage with eventual joint space narrowing.

Another tissue cause of joint osteoarthritis is ligaments. A frequent site is the trapeziometacarpal joint. This joint is often overstretched and used as a weight-bearing instrument for treating soft tissue. This joint is not built for weight-bearing and becomes unstable due to eventual ligamentous laxity. The collateral ligaments both in the interphalangeal and knee joints are associated with osteoarthritis. Varus or valgus malalignment of the knee is a risk factor for osteoarthritis.

Schouten, et al., found a fivefold increase in the risk of progressive knee osteoarthritis in patients with a history of bow legs or knock knees in childhood.⁴ Tan, et al., determined that small-joint collateral ligaments and tendons played a central role in the early stages of hand osteoarthritis.⁵ Using MRI in early hand osteoarthritis where the radiographs were normal, striking abnormalities were seen in the

collateral ligaments and capsules of the proximal interphalageal (PIP) and distal interphalangeal (DIP) joints but not in the articular cartilage or subchondral bone. They found an apparent relationship among the ligaments, bone erosion and bone edema in small joints. Ligaments also were found to be abnormal in normal joints adjacent to clinically involved joints and older, normal joints.

Tendons also may be involved before the cartilage. The extensor tendons over both the DIP and PIP joints were abnormal in 80 percent of early osteoarthritis cases. Even before the ligaments were involved, there was thickening and degeneration in the tendon, identical to changes observed in the

ligaments.⁴ The bone at the distal phalangeal insertion of the extensor tendon was where osteophyte formation occurred. Prerequisites for joint degeneration in osteoarthritis may include ligament, muscle or tendon damage (especially where the tendon inserts).

Palesy concluded that within the musculoskeletal system, the tendino-periosteal attachment of the

muscles and ligaments is very susceptible to injury from trauma.⁶ He feels that these entheses are structurally flawed, richly innervated and metabolically active areas that are the "weakest links" and most reactive sites in the musculoskeletal system. Lesions at these areas may be responsible for a wide range of symptoms in other parts of the musculoskeletal system.

Benjamin and McGonagle state that entheses are of direct pathophysiological relevance in spondyloarthropathies and are associated with diffuse changes in the connective tissues and bone in

the immediate vicinity of the insertion.⁷ *Enthesis organ* is the term used to describe the insertion area since it is no longer considered just a tendon or ligament attaching to a bone. Most insertions have a fibrocartilaginous insertion (some are just fibrous). Rather than considering only its attachment to bone, all of the complex anatomy surrounding the tendon-ligament must be considered. For example, the enthesis organ for the lateral epicondyle includes the tendon, collateral ligament, annular ligament, adjacent circumference of the radial head and humeral articular cartilage. These tissues are related and fuse with each other. The same is true throughout the body regarding enthuses insertions. The authors state, "The inflammatory responses characteristic of spondyloarthropathies are triggered at these seemingly diverse sites, in genetically susceptible individuals, by a combination of anatomical factors which lead to higher levels of tissue microtrauma and the deposition of microbes."

In evaluating patients, it is important to determine what areas are weak, tight or tender. Entheses areas that are tender should be evaluated for local and distal restrictions within the surrounding fascia. In the early stages of osteoarthritis of the fingers and other joints, there may be painless stiffness or minimal nodular areas that should benefit from joint-play mobilization, friction massage, instrument-assisted soft-tissue mobilization (Graston Technique), ART and fascial release methods. This should be applied to the muscles, collateral ligaments, fascia and tendinous insertions. Applying mechanical load to abnormal tissue sets up a whole cascade of healing. Reducing the continuous stress on our musculoskeletal system is definitely preventative.

References

- 1. Brandt KD, Radin EL, Dieppe PA, van de Putte L. Yet more evidence that osteoarthritis is not a cartilage disease. *Ann Rheum Dis*, 2006;65:1261-4.
- 2. Slemenda C, Brandt KD, Heilman DK, et al. Quadriceps weakness and osteoarthritis of the knee. *J Rheumatol*, 1997;127(2):97-104.
- 3. Shrier I. Muscle dysfunction versus wear and tear as a cause of exercise related osteoarthritis: And epidemiological update. *Br J Sports Med*, 2004;38(5):526-35.

- 4. Schouten J, van den Ouweland FA, Valkenburg HA. A 12-year follow-up study in the general population on prognostic factors of cartilage loss in osteoarthritis of the knee. *Ann Rheum Dis*, 1992;51:932-7.
- 5. Tan AL, Toumi H, Benjamin M, et al. Combined high-resolution magnetic resonance imaging and histological examination to explore the role of ligaments and tendons in the phenotypic expression of early hand osteoarthritis. *Ann Rheum Dis*, 2000;65:1267-72.
- 6. Palesy PD. Tendon and ligament insertions a possible source of musculoskeletal pain. *Cranio*, 1997;51(3):194-202.
- 7. Benjamin M, McGonagle D. The anatomical basis for disease location in seronegative spondyloarthropathy at enthesis and related sites. *J Anat*, 2001;199:503-26.

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