

Dextrose Shots May Jump Start Healing In Tendinopathies

BY KATE JOHNSON
Montreal Bureau

QUEBEC CITY — Hyperosmolar dextrose injected into ailing tendons may cause tissue damage that triggers a healing response, reported Michael Ryan, a doctoral candidate at the University of British Columbia in Vancouver.

Mr. Ryan and his coinvestigators have previously reported good to excellent outcomes with this approach, known as prolotherapy, in treating both infrapatellar and Achilles tendinopathies.

He reported on their most recent pilot investigation into the treatment of chronic plantar fasciitis at the joint annual meeting of the Canadian Academy of Sport Medicine and the Association Québécoise des Médecins du Sport.

The study involved 23 patients with chronic plantar fasciitis (average duration, 28 months) who had failed conservative treatment. The patients had been referred to the investigators from a local sports medicine clinic. Their level of dysfunction was extremely high—some of them could not walk without a walking boot.

The patients' injured plantar fasciae, seen on ultrasound, had the characteristic features of anechoic foci, neovascularity, regions of hypoechogenicity, and calcification/cortical defects, said Mr. Ryan. The investigators injected a 50% dextrose solution diluted with 2% lignocaine into sites of palpable pain and anechoic clefts/tears using a 27-gauge needle under ultrasound guidance.

The patients received an average of five injections 6 weeks apart, for an average treatment duration of 33 weeks. At the end of that period, 14 patients reported good to excellent results, with 12 of them reporting complete resolution of symptoms and return to function.

These clinical outcomes corresponded to structural improvements seen on ultrasound, including a reduction in the number of intrasubstance tears (from 7 to 2), hypoechoic areas (from 10 to 3), calcifications (from 7 to 1), and neovascularities (from 2 to 0).

Prolotherapy, first described by Hippocrates, is thought to work by adding injury to an already injured site, so triggering the body's natural healing response.

"By creating an osmotic shock around the site of the injection, we create an inflammatory response in the tendon, which may be absent in some patients. Individuals who are 28 months symptomatic are considered to have an abnormal immune response, and this irritation stimulates healing by bringing blood to the area and releasing growth factors," Mr. Ryan explained.

He noted that imaging may not always be necessary, which is encouraging some doctors to try prolotherapy in the primary-care setting "If the tendon is easy to palpate, such as the Achilles or the infrapatellar tendon, it's easy to find the injection spot."



An ultrasound-guided needle delivers the dextrose. Shown here is an intratendinous tear.

Therapy Still Dickey as Gout's Incidence More Than Doubles

BY SHARON WORCESTER
Southeast Bureau

DESTIN, FLA. — The incidence of gout is on the rise, and lifestyle factors are largely to blame, Dr. N. Lawrence Edwards said at the annual Rheumatology on the Beach.

For example, one study showed that between 1977-1978 and 1995-1996, the annual rate of primary gout more than doubled, from about 16 cases/100,000 population to nearly 42 cases/100,000 population. Factors that appear to play a role in this incidence spike are greater longevity, widespread diuretic and aspirin use, hypertension incidence, obesity, metabolic syndrome, and dietary trends, said Dr. Edwards, professor and vice chairman of the department of medicine at the University of Florida, Gainesville.

Weight reduction, decreased alcohol consumption, and reduced intake of purine-rich foods (which have been linked with gout) will reduce urate levels only by about 1 mg/dL. Medical treatment should be considered early in patients presenting with acute attacks, he said.

Urate-lowering therapy, which 15 years ago was reserved for use in patients with chronic gout, now is considered warranted following the first one or two acute attacks.

Uricosuric agents, such as probenecid, and the xanthine oxidase inhibitor allopurinol are used for reducing urate

levels, but uricosuric agents increase the risk of uric acid crystallization in the urine and associated stone formation. There are a number of other agents, such as ampicillin, penicillin, cephadrine, heparin, and rifampicin, that can potentially affect the action of uricosuric agents, Dr. Edwards said.

Allopurinol, which is a purine analog that is both a substrate and inhibitor of xanthine oxidase, is effective both for people who overproduce and for those who underexcrete xanthine oxidase. The drug also has the convenience of single daily dosing, and it can be efficacious in patients with renal insufficiency.

However, allopurinol isn't always effective for achieving target serum urate levels and concerns about intolerance based on reports of severe hypersensitivity syndrome, rash, gastrointestinal problems, increases in liver enzymes, and bone marrow suppression, tend to scare physicians away from prescribing higher doses, he noted.

As with uricosuric agents, drug-drug interactions also are a problem with allopurinol.

Keys to effective treatment with this drug include dosing allopurinol to achieve a serum urate level between 5.0 and 6.0 mg/dL, which allows reduction of total body urate pool and mobilization of deposited crystals, Dr. Edwards said, stressing the need to start at a low dose of 50-100 mg/day, with close monitoring of escalation. ■

Not All Physicians Have Heeded Warnings About Cardiac Risks of Pain Medications

BY KATE JOHNSON
Montreal Bureau

Physicians need a stronger message about the cardiac risks of treating chronic pain with anti-inflammatory drugs, both traditional NSAIDs and cyclooxygenase-2 inhibitors, according to Dr. Elliott M. Antman and his colleagues.

"We believe that some physicians have been prescribing COX-2 inhibitors as the first line of treatment. We are turning that around and saying that, for chronic pain in patients with known heart disease or who are at risk for heart disease, these drugs should be the last line of treatment," Dr. Antman, lead author of the statement, said in an interview.

He added that this approach should be adopted even for patients with no known heart risks, and caution should not be limited to the COX-2 inhibitors but extended to all NSAIDs. "The regulatory authorities have now put black box warnings on all NSAIDs, except aspirin, and even today many physicians are not aware [the warnings] exist."

The American Heart Association statement updates the 2005 statement and reflects this new information, said Dr. Antman, professor of medicine at Harvard Medical School, Boston. But the document, which was coauthored by six cardiologists, might not sit so comfortably with physicians who treat chronic pain on a regular basis.

"I have mixed feelings about it,"

said Dr. Roland Moskowitz, a rheumatologist and professor of medicine at Case Western Reserve University, Cleveland, in an interview. "I agree ... you have to be cautious. But that doesn't mean we can't use these medications judiciously and appropriately. They're looking at it from the cardiologist's view when it's the rheumatologists who are sitting with the patient who is in pain."



In chronic pain patients with known heart disease, 'these drugs should be the last line of treatment.'

DR. ANTMAN

The AHA document outlines a stepped-care approach to the pharmacologic treatment of musculoskeletal pain in patients with known cardiovascular disease or risk factors, starting with agents that have the lowest cardiac risk. "When acetaminophen, aspirin, and perhaps even narcotic medications (for acute pain) are not effective, tolerated, or appropriate, it may be reasonable to consider an NSAID as the next step; however, this should be coupled with the realization that effective pain relief may come at the cost of a small but real increase in risk for cardiovascular or cerebrovascular complications," wrote Dr. Antman and his colleagues

(Circulation 2007 Feb. 26 [Epub DOI:10.1161/CIRCULATIONAHA.106.181424]). "If symptoms are not adequately controlled by a non-selective NSAID, subsequent steps involve prescription of drugs with increasing degrees of COX-2 inhibitory activity, ultimately concluding with the COX-2 selective NSAIDs."

The statement outlines the spectrum of COX-2 inhibition and thus the varying degrees of cardiac and gastrointestinal risk for a wide range of NSAIDs.

Dr. Moskowitz said that most rheumatologists are already aware of the possible cardiac risks of all NSAIDs, but also have to consider gastrointestinal risks and effective pain control. "[Physicians] are frightening people away from using these things when they need to use them."

The American College of Rheumatology's guidelines on NSAID use have not yet been updated to reflect more recent concerns about cardiovascular risk (Arthritis Rheum. 2000;43:1905-15). The Osteoarthritis Research Society International's guidelines committee, of which Dr. Moskowitz is cochair, is expected to release its recommendations on the overall management of osteoarthritis in the next few months.

Dr. Antman and his colleagues disclosed no potential conflicts of interest. Dr. Moskowitz has served as a consultant for Pfizer Inc., Novartis, Merck & Co., GlaxoSmithKline Inc., and Sanofi Aventis. ■