

SCIENTIFIC

PHYSICAL THERAPY

Patient with dull, aching pain across the lower back and burning sensation in the left medial thigh.

CASE STUDY

By Matt Hatscher P.T.

HISTORY:

The patient is a 64 y.o. white male who is 5'8" and weighs 185 lbs. He currently works full time as an insurance broker and spends most of his time sitting at a desk or driving in his car. He suffered a herniated disc at the end of September '03 while playing golf. Originally, he experienced pain in his lower back and an intense burning sensation in his L medial thigh. He underwent an L4-L5 far lateral discectomy 6 weeks ago and reports less intense burning sensation in his L thigh and dull, aching pain across his lower back. Sx increase with standing, walking >0.5 mile, and going downstairs. Sx improve if he lies on his L side. Sx are also worse at the end of the day. He has not been given or been doing any exercises since the surgery and is not taking any medications. The patient reports a history of clavicle and L hand fx, and benign tumor in throat. His primary goal is to return to playing golf without pain.

SYSTEMS REVIEW

Structural inspection: L iliac crest higher than R, 1" well healed scar on L side of spine at L4- L5, minimal atrophy at bilateral erector spinae.

SI and hip joints all WNL

No significant findings pertaining to cardiopulmonary, integumentary, or mental status.

In this issue:

Patient with dull, aching pain across the lower back.

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Tendinitis?

By Didrik J Sople, PhD, L.Ac.

New Research on Stretching

By Didrik J Sople, PhD, L.Ac.

TESTS AND MEASURES

AROM: Lumbar

(Stiffness subjectively reported with all motions)

Flexion: Moderate limitation

L SB: Moderate limitation

L rotation: Moderate limitation

Flex/L rotation: Moderate limitation

Extension: Minimal limitation

R SB: Moderate limitation

R rotation: Moderate limitation

Flex/R rotation: Moderate limitation

Ext/R rotation: Moderate limitation

Ext/L rotation: Moderate limitation

PROM: Lumbar

Same limitations as active with subjectively reported tightness

Resisted tests:

4+/5 strength in all planes without pain

Palpation:

No tenderness to palpation at scar, spinous process, facet joints, or ligaments. Mild tenderness to palpation at L erector spinae and multifidi. Moderate guarding of L QL

Neurology:

Myotomes: 5-/5 at L3 and L4 distributions on L

Dermatomes: Slightly decreased sensation with the dull and sharp at L L3

DTR's: 1+ at L patellar

(-) skin rolling test B along spine

Special tests:

All compression tests (-)

Leg length tests (-)

Slump and Cram's test (-) for sciatic tension

Discomfort and tension with femoral nerve tension on L

Shearing (+) at L3-L4 and L4-L5

Joint mobility: 2/6 at L3-L4 in FB, 4/6 at L4-L5 in R rotation

EVALUATION AND DIAGNOSIS

Medical diagnosis: Far lateral L4-L5 discectomy

PT diagnosis: Disc degeneration at L3-L4 and L4-L5 secondary to trauma and surgery resulting in impaired nerve function (motor and sensory) and weakness/atrophy of lumbar erector spinae and multifidi.

LITERATURE REVIEW

Hodges, et al 1999

Retrospective review of 25 pts who underwent surgery for far lateral disc herniations at L3-L4 or L4-L5. VAS scores improved from 7.7 to 4.2 and Oswestry scores improved from 50.7% to 34.7% without disrupting spinal stability.

Epstein, NE. 2002

Far lateral disc herniations constitute 7-12% of all disc herniations. Occur primarily at L3-L4 and L4-L5. Syndromes reflect compression of superiorly exiting nerve root and ganglion. Complaints include severe radicular pain accompanied by very positive mechanical signs. Neurological deficits are seen over 75% of the time. Conservative management is occasionally successful (10%) but surgery is usually required.

Porchet, et al 1999

Follow up (mean of 50 months) of 202 pts who underwent far-lateral approach for disc herniations. 31% report excellent results, 42% report good results, 20% report fair results, and 7% report poor results.

The following literature supports lumbar stabilization and dynamic exercises following discectomies:

Kjelby-Wendt G., Styf J. 1998

Yilmaz, et al 2003

Dolan, et al 2000

Danielsen, et al 2000

RECOMMENDATIONS

Initial treatment has begun with STM to the L QL and multifidi to reduce guarding. Additional manual treatment has been forward bending joint mobilization at L3-L4 with 10 sec holds for plastic deformity at end range. Clinical exercise was initiated with sidelying caudal rotation at 3x25 at 4 kg to promote coordination and endurance of the multifidi and optimal stimuli for the disc. Fall out lunges (3x15 at 4#) and prone hip extension (3x10) for improved recruitment, coordination, and stability. Home exercise program included 1) lower trunk rotations for optimal stimuli to the disc, coordination, and vascularity 2) SKC stretches for improved mobility in the lumbar spine, and 3) fall out lunges for improved paraspinal recruitment.

Expect to have reduction in guarding with improved recruitment and coordination in 2 weeks.

TREATMENT INTERVENTIONS AND PROGRESSIONS

Stage 1 (Weeks 1-2): STM and joint mobilizations with exercises as stated above. Initiate sit ups for abdominal strengthening. Primary goal is to improve endurance and coordination in pertinent muscles (multifidi, TrA, erector spinae, gluts). Also providing compression/decompression with modified tension in the line of stress to the disc.

Stage 2 (Weeks 2-3): Continue manual treatment as necessary. Progress to standing rotational exercises at 3x25 at 60% then to 3x15 at 85% for strength training. Continue to progress hip strengthening with lunges and steps. Progress abdominal strengthening as tolerated with 3 x 15 on slant board. Also begin to initiate diagonal patterns with pulleys at 3x25 at 60% for coordination.

Stage 3 (Weeks 3-5): Progress pulley diagonals to 3x15 at 85% for strength training. Also, may add quick reversal for eccentric training. Continue closed chain activity for LE strengthening. Progress abdominal training. Begin swinging golf club at home without contact and progress to hitting balls at the range.

DISCUSSION/CONCLUSION

Patient has responded well to exercises. After 2 sessions pt has returned to his gym and states he is able to walk on treadmill for 30 minutes. Subjectively, he has an easier time moving and it is not as difficult getting in and out of his car. Still continuing to have L medial thigh pain and difficulty going downstairs.

As pt is very motivated and has responded well to the early stage of exercise, I believe he will be able to meet his goal and return to golf in 2-3 months.

Tendinitis?

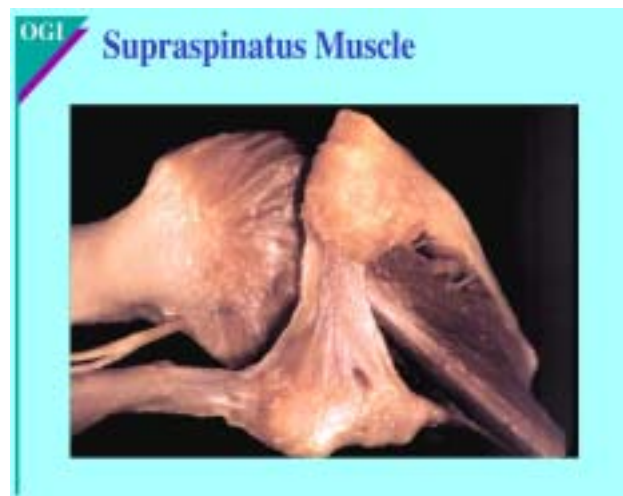
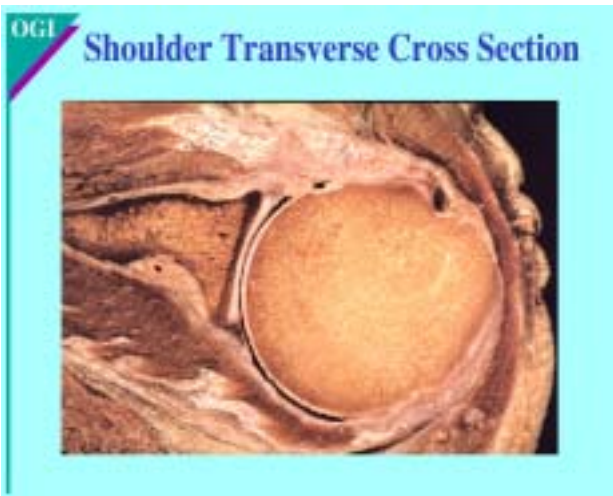
Reviews of a presentation at the
Ola Grimsby Institute Competency Forum, 2004
and some of the recent literature on tendinopathies.

by
Didrik J. Soplér, Ph.D., L.Ac.

One of the excellent presentations at the last OGI Competency Forum was titled "New Concepts of Tendinopathy and Their Clinical Applications", and the speaker was Karim Kahn, M.D., Ph.D. from the University of British Columbia, Vancouver, Canada.

Dr. Kahn has, together with other researchers, studied tendon disorders, so called "tendinitis" quite extensively the last few years. How common is it to suffer from tendinitis of the elbow, shoulder, knee and heel? Quite common according to the diagnosis used on painful conditions in these areas. If this is correct, the pain from these conditions is caused by a local inflammatory reaction in the tendon or tendons where the pain is located. When a biopsy of the tissue in an inflammatory condition is examined microscopically, it should show evidence of inflammatory cells. What is so interesting however is that no such evidence has been documented. Kahn and colleagues state in an article where they examine the cause of common tendinopathies that the most significant feature is the absence of inflammatory cells.¹

These researchers state that the term tendinitis is an incorrect way of describing symptomatic Achilles, patella, extensor carpi radialis brevis and rotator cuff tendons. They instead suggest the term tendinopathy to be used for these common tendon conditions.



Histologically a normal tendon when compared with a tendon in these conditions looks very different. The pathology of overuse tendinopathy is non-inflammatory with a degenerative or failed healing response.² When tissue samples of patients having surgery for tendon pain were examined microscopically, collagen separation was found.³ Also found was an increase in tenocytes with myofibroblastic differentiation (tendon repair cells).

What is still the most common way of treating these conditions? Anti-inflammatory drugs, because most practitioners still believe they are treating "tendinitis", a local inflammatory condition.

Does it work? No, a review of the role of various anti-inflammatory medications found limited evidence of short term pain relief and no evidence of effectiveness in providing even medium term clinical resolution of clearly diagnosed tendon disorders.⁴

Tendinopathies are conditions with cellular pathology and is best treated as such. The cellular metabolism is abnormal and the tissue does not heal as it should. Tissue like this has a lower tolerance for stress. These conditions are looked at as overuse conditions. Some of the patients have however not necessarily participated in very strenuous activities.

Treatments which have shown to produce results in tendinopathies is an approach providing stimulation to the tissue. Eccentric exercises have proven to be effective in treating both Achilles and chronic patellar tendinopathy.^{5, 6}

There have been attempts trying to explain why we develop tendinopathies. In one study, human patellar tendon fibroblasts were exposed to cyclic stretching in a laboratory setting.⁷ They found that after 4 hours of stretching, there were increases in prostaglandin E2 and leukotriene B4 levels. The researchers also found that blocking of the prostaglandin E2

lead to increased levels of leukotriene B4 and visa versa.

They stated the clinical relevance would be that use of nonsteroidal anti-inflammatory drugs might increase the levels of leukotriene B4 within the tendon and potentially contributing to the development of tendinopathy.

In a follow up study, human patellar tendon fibroblast were treated with prostaglandin E2 in cultures.⁸ The researchers found that certain concentrations of prostaglandin E2 inhibited collagen production of the tendon fibroblasts. Their conclusion was that the production of prostaglandin E2 in tendons might play some role in the acellularity and matrix disorganization seen in exercise-induced tendinopathy.

When looking critically at the two studies, there are potential problems when it comes to clinical relevance. Both of the studies were done in a laboratory setting. The stretching of the tendons was

done for four hours at certain frequencies. It is questionable if this scenario is relevant to real life situations. When Prostaglandin E2 was found to inhibit collagen production certain concentrations were used. At lower concentration, no inhibition took place. Again, is this relevant to a real clinical situation?

In light of recent research from Sweden, it does not look like it is clinically relevant. In an attempt to figure out reasons for tendinopathy, one study examined the concentrations of glutamate (a neurotransmitter) and prostaglandin E2 in chronic painful tendinosis (Achilles, patellar, extensor carpi radialis brevis).⁹ The researchers found a significantly higher concentration of glutamate, but the prostaglandin E2 was normal confirming the absence of a local inflammatory reaction.

It was first believed that glutamate since it is an excitatory neurotransmitter could be a reason for tendon pain, but that proved not to be so in another study. Successful treatment of Achilles tendinosis using eccentric exercises showed no decrease in glutamine levels after the treatment compared to pretreatment levels.¹⁰



There seems to be an anaerobic condition in the area with tendinosis. When lactate levels were measured in patients with Achilles tendinosis the lactate level in the abnormal tendons were approximately twice as high as in the normal control tendons.¹¹

What is the reason for the pain in tendinopathy?

There may be several reasons for the pain in these types of conditions. One interesting finding is the occurrence of neovascularisation in the area of structural tendon changes in painful tendinosis. This has not been found in normal pain-free tendons.

When a sclerosing agent was injected into 10 painful tendons showing neovascularisation, 8 of the 10 patients were satisfied with the result of the treatments which significantly reduced pain with activity.¹² There were no remaining neovascularisation in these patients. In the two patients who were not satisfied with the treatments, neovascularisation still remained.

In a study investigating the presence of substance P in tendon insertions of the medial and lateral epicondyles of the humerus, the researchers found indications that substance P has an effect on the nerves in this area.¹³ They stated that the results gave further evidence for possible neurogenic involvement in the pathophysiology of tennis elbow and in medial epicondylalgia.

In view of this information what is the best form of treatment for tendinopathies, eccentric exercise or injections of a sclerosing agent?

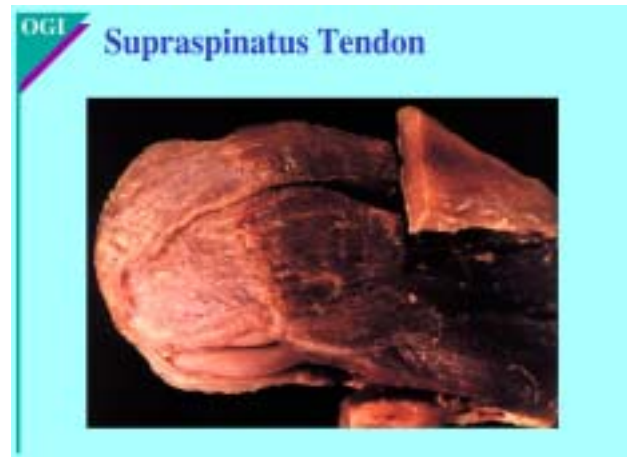
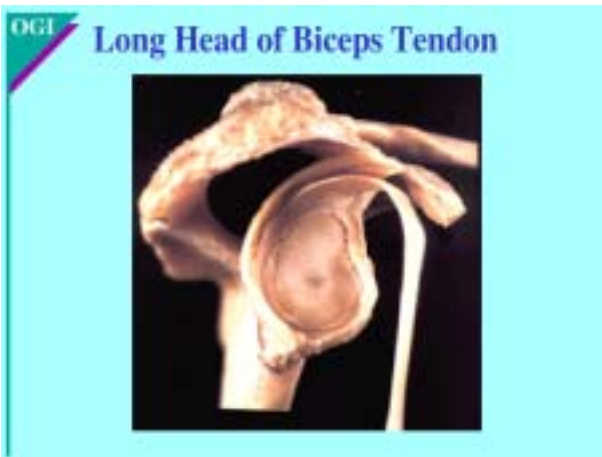
A recent study on chronic Achilles tendinosis showed some very interesting results.¹⁴ Eccentric calf muscle training decreased localized tendon thickness and normalized tendon structure in most of the patients. The patients who still had residual pain, also had remaining structural tendon abnormalities.

In another study where eccentric exercise was used in treatment of chronic midportion Achilles tendinosis, the presence of neovascularisation was also investigated.¹⁵ Before the treatments, there was detected local neovascularisation in the area with tendon changes. In 34 of 36 tendons with good clinical results of treatment there was a more normal tendon structure. In 32 of the 36 tendons there was no remaining neovascularisation. In 5 of 5 tendons with poor clinical results there was still detected remaining neovascularisation in the tendon. In 2 of the 5 tendons with poor results there were still remaining structural abnormalities.

According to the information provided in these studies the treatment of choice would be eccentric exercise since that showed to eliminate both neovascularisation as well as other tendon abnormalities.

Eccentric exercises do not only work for Achilles tendinopathy. A very recent study showed it worked well for painful chronic patellar tendinopathy.⁶ Eccentric squat exercises standing on a 25 degree decline board showed better results when compared to eccentric standard squats with the ankle joint in a standard position.

Eccentric exercise can be used as an effective treatment modality for tendinopathy. The anatomical location of the tendon does not seem to matter.



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New research on stretching

by

Didrik J. Soplér, Ph.D., L.Ac

Stretching has been a controversial issue for many years. A very recent study published in the August 2004 issue of *Med Sci Sports Exerc* investigated the effects of static stretching on force and jumping performance.¹

The objective was to examine whether static stretching decreased isometric force, muscle activation and jump power while improving range of motion.

12 subjects were tested before and after 30, 60, 90, and 120 minutes of static stretching of the quadriceps and plantar flexors. As a control condition, they were also tested at a similar period, but without stretching. The measurement taken during isometric contractions were maximum voluntary force, evoked contractile properties (peak twitch and tetanus) surface integrated electromyographic activity if the agonist and antagonistic muscle groups and muscle inactivation measured by the interpolated twitch technique. Vertical jump height was measured as unilateral concentric only (no counter movement), as well as drop jump height and contract time.

Range of motion was measured with seated hip flexion and prone hip extension. Plantar flexion – dorsi flexion was also measured. The results showed that after static stretching, there was a significant 9.5%

decrease in the torque or force with maximum voluntary force of the quadriceps and a 5.4% decrease with the interpolated twitch technique. The force remained significant decreased for 120 minutes at 10.4% which paralleled a significant increase of 6% in sit and reach range of motion after 120 minutes. There were no significant changes in jump performance after the stretching.

Another study published in August, 2004 in the same journal investigated the effect of static stretching on force, balance, reaction time and movement time.² Sixteen subjects were included in the study. They were tested before and after static stretching of the quadriceps, hamstrings and plantar flexors or similar duration control condition. The protocol involved a 5 minute cycle warm-up followed by three stretches to the point of discomfort of 45 seconds each with 15 seconds rest periods for each muscle group. It included maximum voluntary isometric contraction force of the leg extension, reaction and movement time of the dominant lower limb and the ability to make 30% and 50% maximum voluntary isometric contraction force with and without visual feedback.

This study did not find any significant differences in the decrease in maximum voluntary isometric con-

traction force between the stretch and control condition or in the ability to match sub maximal forces.

The balance scores decreased however significantly with 9.2% after stretching as compared with the control condition increased 17.3%. There was also a significant difference in reaction and movement time. After stretch, the reaction time increased with 4% and the movement time with 1.9% while in the control condition the reaction time decreased with 5.8% and movement time decreased 5.7%.

Looking at the overall results of these two studies, there does not seem to be any advantages with stretching prior to athletic performance, at least not if stretching is performed according to the guidelines used in the United States.

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