

SCIENTIFIC

PHYSICAL THERAPY

Iliotibial Band Syndrome: updates from the literature, recommendations for conservative treatment and further research

By

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Introduction

Iliotibial band syndrome (ITBS) is the leading cause of lateral knee pain in runners and cyclists, although it has also been described in soccer players, skiers and weight lifters. ITBS is characterized by an aching pain over the lateral aspect of the knee which is aggravated by the particular sports activity when the knee is at about 30° of flexion. Examination reveals palpable tenderness and sharp pain over the lateral femoral epicondyle with occasional crepitus during repeated flexion and extension of the knee. Ober's test may show iliotibial band (ITB) tightness.

Differential diagnosis of lateral knee joint pain includes internal derangement of the knee, lateral meniscal tear, chondromalacia, lateral collateral ligament sprain, popliteal tendon strain, biceps tendinopathy, or lateral hamstring strain.

In recent years, several anatomical and MRI studies have revealed new insights with regards to the anatomy, biomechanics and the potential causative factors that play a role in ITBS, thereby challenging some previously made assumptions.

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Anatomy

The iliotibial band (ITB) originates in the facial components of the gluteus maximus, gluteus minimus, and the tensor fasciae latae muscles. The ITB itself is not a discrete structure, but a thickened part of the fasciae lata which envelops the lateral thigh (Fairclough et al. 2007). It inserts distally to the supracondylar tubercle of the femur and the lateral intramuscular septum, and it continues distally to attach to Gerdy's tubercle. Proximal to the lateral femoral condyle, the ITB is separated from the femur by a wide layer of fatty tissue that extends to the vastus lateralis muscle. At the level of the lateral femoral condyle, the ITB contacts the lateral femoral epicondyle and the inserting fibers of the lateral collateral ligament.

Vieira et al (2007) observed an iliotibial tract arrangement in superficial, deep, and capsular-osseous layers. Insertions have been described as follows: at linea aspera, at the upper border of the lateral epicondyle, at the patella, and at Gerdy's tibial tuberculum and across the capsular-osseous layer. They concluded that the iliotibial tract can be considered as an anterolateral stabilizer of the knee. Based on gross anatomy, Fairlough et al. (2006) also made a clear distinction between a tendinous part of the ITB proximal to the lateral femoral condyle and a ligamentous distal part between the lateral epicondyle and Gerdy's tubercle.

Muhle et al. (1999) and Fairclough et al. (2006 & 2007) did not show the presence of a bursa beneath the distal ITB, neither on MRI nor upon microscopic examination of cadavers. However, a thin layer of richly innervated and vascularized fatty tissue was found between the ITB and the lateral femoral condyle and the lateral femoral epicondyle. The lateral synovial recess was found to be located anterior and proximal to the lateral femoral epicondyle at full extension of the knee and at both 30° and 60° of knee flexion.

Enhanced gadolinium MRI studies of two runners with ITBS showed increased signal deep to the ITB in the region of the lateral femoral condyle, suggesting edema in the fatty tissue deep to the fascia lata (Fairlough et al. 2006).

Biomechanics

Orchard et al. (1996) proposed a biomechanical model to explain the pathogenesis of iliotibial band "friction" syndrome (ITBFS) in distance runners. They suggested that friction between the posterior edge of the iliotibial band and the underlying lateral femoral epicondyle occurs near foot strike, predominantly in the foot contact phase. The flexion angle of the knee at foot strike was on average 21.4° (+/- 4.3°) with friction occurring at or slightly below the 30° of flexion.

Downhill running predisposes the runner to ITBFS because the knee flexion angle at foot strike is reduced. Sprinting and faster running on level ground are less likely to cause or aggravate ITBFS because, at foot strike, the knee is flexed beyond angles at which friction occurs.

In cyclists, improper seat position, improper cleat position, leg-length discrepancy, and training errors could all contribute to ITBS. Holmes et al. (1993) found a high seat position which caused less than 30° of a knee flexion angle at the bottom center of the pedaling stroke in 13 of 61 cyclists with ITBS. Eighteen cyclists were noted to have fixed cleats that were excessively toed-in with respect to the cyclist's anatomy. This causes an exaggerated abduction of the knee on the pedaling upstroke and adduction of the knee on the down stroke. Seven cyclists with ITBS had a leg length discrepancy of one-quarter of an inch or more.

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High-resistance training and high-mileage cycling seem to be contributing factors in ITBS. At a crank angle of 170°, a knee flexion angle of approximately 33° occurs in healthy volunteers with a foot-pedal force of 231 N, which is only 18% of the forces occurring during running, when the ITB is in the “impingement zone”. Repetition of the knee in the “impingement zone” during cycling may therefore play a more prominent role than force in the onset of ITBS (Farrell et al. 2003). Especially, since during cycling with an average pedaling cadence of 80 RPM knee flexion and extension occur approximately 4800 times an hour (Holmes et al. 1993).

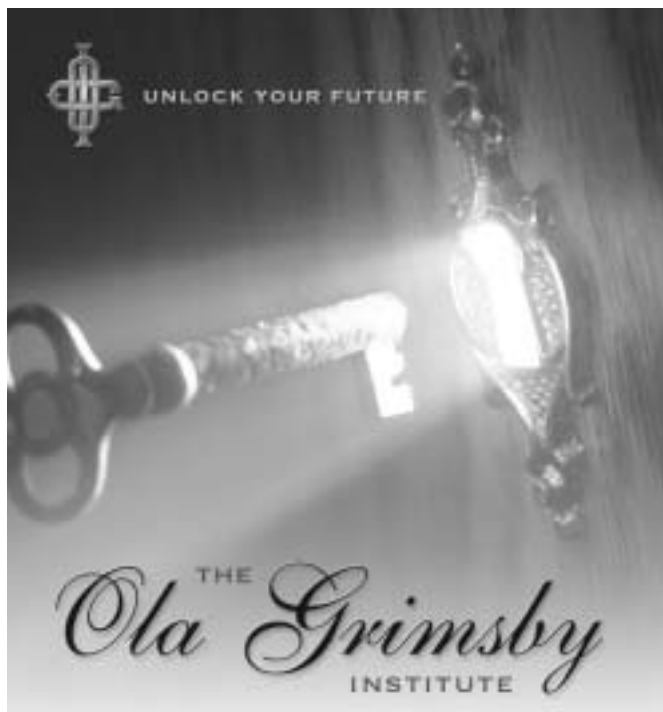
Weakness or inhibition of the lateral gluteal muscles and hip abductors may be a causative factor in ITBS in runners. When these muscles do not fire properly (eccentrically) throughout the support phase of running, there is a decreased ability to stabilize the pelvis and eccentrically control femoral abduction. In an interesting prospective study performed by Noehren et al. (2007) a group of 400 runners was followed for 2 years after collecting 3-dimensional data on lower extremity kinematics and kinetics during running. Within the two years, 18 of the 400 runners developed ITBS. These runners with ITBS landed in greater hip adduction throughout stance with greater peak adduction angles. However, peak abduction moments were no different compared to a control group. They did not find a significant difference in knee flexion angles at heel strike between groups. The ITBS group landed in greater internal rotation and remained more internally rotated throughout stance. In addition, the ITBS group had a significantly higher peak knee internal rotation angle. After analyzing segmental rotation in the lower extremity, tibial internal rotation was lower by 2.2° in the ITBS group, whereas femoral external rotation was significantly greater, thus causing greater knee internal rotation. At the rearfoot, the ITBS group landed in slightly more inversion, but there was no significant difference in rearfoot inversion compared to the control group.

This increased external rotation of the hip may be related to muscle imbalances at the hip. Co-contraction of the internal and external rotators of the hip is necessary to provide stability to the femoral head in the acetabulum during loading. The gluteus minimus, anterior fibers of the gluteus medius, and the tensor fascia latae all serve to abduct and internally rotate the femur. Insufficient activity of these muscles can lead to the increased femoral external rotation (Noehren et al. 2007).

Grau et al. (2007) tested isokinetic concentric, eccentric, and isometric peak torque of the hip abductors and adductors at 30 degrees/sec between 10 healthy volunteers and 10 injured runners with ITBS. They found no significant differences between the two groups in any of the muscles tested at this particular speed. They concluded that weakness of hip abductors does not seem to play a role in the etiology of ITBS in runners, since dynamic and static strength measurements at 30 degrees/sec did not differ between groups, and differences between hip abduction and adduction were the same.

When examining lower extremity mechanics of the iliotibial band during exhaustive running in 8 individuals with ITBS, Miller et al. (2007) found an increased knee flexion at heel strike, and an increased foot inversion and higher maximum knee internal rotation velocity at the end of the run, compared to runners without ITBS.

Fairlough et al. (2006 and 2007), based on anatomical cadaver and MRI studies, found that the ITB gets compressed against the lateral femoral epicondyle at 30 degrees of knee flexion as a consequence of tibial internal rotation, whereas it moves laterally and is pulled away from the lateral epicondyle during knee extension. The ITB is prevented from rolling over the epicondyle by its femoral anchorage and because of it being part of the fascia lata which envelopes the lateral thigh. This creates the illusion of movement of the ITB during flexion-extension of the knee, because of changing tension in its anterior and



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posterior fibers during knee flexion. With progressive knee flexion, tension shifts from the anterior to the posterior fiber bundles of the ITB. Based on gross anatomy, Fairclough et al. (2006) also made a clear distinction between a tendinous part of the ITB proximal to the lateral femoral condyle and a ligamentous distal part between the lateral epicondyle and Gerdy's tubercle. MRI study of an injured runner with ITBS showed evidence of edema in the fatty tissue deep to the fascia lata. They concluded that overuse and associated pain experienced in ITBS may be more associated with compression of highly vascularized and innervated fat and loose connective tissue that separates the ITB from the lateral femoral epicondyle with slight medial movement of the ITB during component knee internal rotation as the knee flexes, rather than from repetitive friction as previously described in runners by Orchard et al. (1996) and in cyclists by Farrell et al. (2003). They also suggested that the ligamentous part of the ITB could assist in limiting internal rotation of the tibia.

Conclusion

Iliotibial band syndrome (ITBS) is a common cause of lateral knee pain in runners and cyclists and often occurs as a result of increased training intensity. Recent anatomical studies more clearly identify the structural anatomy of the ITB and its distal insertion sites, as well as the presence of a highly innervated and vascularized layer of fat between the ITB and the lateral aspect of the femur as a likely anatomical cause for the pain associated with ITBS. It also provides an explanation with regards to the biomechanical factors that play a role in this syndrome as it associates component knee internal rotation with increased compression of this layer of fat by the ITB as the knee moves from full extension into flexion.

Conservative treatment of ITBS should place emphasis on correction of training errors and modification of training intensity. Runners should decrease their distance and weekly mileage, and avoid downhill running and running on an angled track. Cyclists should also decrease their weekly mileage by at least one-half, pedal with less resistance and on a flat terrain, decrease their seat height to assure proper knee flexion angles, switch to floating pedal systems and make adjustments in cleat position to prevent hip adduction and excessive knee internal rotation.

Ellis et al. (2007) identified four randomized controlled trials with regards to the conservative treatment for ITBS. They concluded that non-steroidal anti-inflammatory drugs, deep friction massage, phonophoresis and corticosteroid injection show limited evidence that conservative treatment of ITBS offer any significant benefit in the management of ITBS.

Further research on the prevention and treatment of ITBS is warranted and should specifically explore ways that facilitate the internal and external forces that control knee internal rotation, hip rotation, and hip adduction during sports related activities.

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Adjunct treatment for neuropathies.

By
Didrik Sople, Ph.D., L.Ac.

Neuropathy is a difficult condition to treat, but there is now evidence that two substances available as nutritional supplements are effective in the treatment of neuropathy. These two substances are alpha lipoic acid and acetyl-L-carnitine.

There can be many reasons for developing neuropathy. Alpha lipoic acid and acetyl-L-carnitine have shown to be effective in treating compression neuropathy, diabetic neuropathy and neuropathy caused by chemotherapy.

One of the most common symptoms of peripheral neuropathies is pain. The reparative processes and inflammation are contributing factors to this pain. Some of the mechanisms by which acetyl-L-carnitine is working is by inducing regeneration of injured nerve fibers, reducing oxidative stress, supporting DNA synthesis in the mitochondria and enhancing nerve growth factor concentrations in neurons (Vanotti A, et al, 2007).

In an experimental chronic compression neuropathy study on rats of the sciatic nerve, it was found that when acetyl-L-carnitine were co-administered with decompression it enhanced the clinical and histopathological recovery (Kotil K, et al, 2007).

Acetyl-L-carnitine has been documented to be effective for diabetic neuropathy. A study involving 1257 patients over a time period of 52 weeks showed significant improvements in sural nerve fiber numbers, regenerating nerve fiber clusters, vibration perception and pain (Sima AA, et al, 2005).

The investigators concluded that acetyl-L-carnitine is effective in alleviating symptoms, particularly pain, improve nerve fiber regeneration and vibration perception in patients with established diabetic neuropathy.

One of the side effects of chemotherapy can be neuropathy from the toxicity. Acetyl-L-carnitine has also shown to be effective in the treatment of neuropathy from paclitaxel and cisplatin therapy.

When the effectiveness of acetyl-L-carnitine was investigated in cases of chemotherapy induced neuropathy it was found to improve sensory neuropathy grade in 60% of the patients, motor neuropathy in 79% and total neuropathy score improved in 92% (Bianchi G, et al, 2005).

Alpha lipoic acid is an antioxidant which has shown impressive results in diabetic neuropathy in as short a time as 3 weeks. The study documenting results in 3 weeks used an oral dosage of 600 mg of alpha lipoic acid t.i.d. (Ruhnau KJ, et al, 1999). The total symptom score used included pain, burning sensation, paraesthesiae and numbness in the feet and was recorded at weekly intervals and summarized. The Hamburg Pain Adjective list and the Neuropathy Disability Score were assessed at baseline and day nineteen. The total symptom score decreased from baseline to day nineteen by 47% in the treatment group and by 24% in the placebo group. The Hamburg Pain Adjective list score decreased from baseline to day nineteen by 60% in the treatment group and by 29% in the placebo group. There were no differences between the groups regarding adverse events. The researchers concluded that the findings indicated that oral treatment with 600 mg of alpha lipoic acid t.i.d. for three weeks may improve symptoms and deficits resulting from polyneuropathy in type 2 diabetic patients.

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In another study also on diabetic polyneuropathy the researchers used alpha lipoic acid in an oral dosage of either 600 mg, 1200 mg or 1800 mg once daily (Ziegler D, et al, 2006). The time period of the study was five weeks. The total symptom score which included stabbing pain, burning pain, paraesthesiae and asleep numbness of the feet was reduced by 51% in the 600 mg group, 48% in the 1200 mg group and 52% in the 1800 mg group. The placebo group had a reduction of 32%. This led the researchers to recommend 600 mg as the daily dosage.

These findings are quite interesting and these substances are worth while to include in a treatment program, especially alpha lipoic acid since results has been documented in as short a time as three weeks.

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Strong is better.

Research Review

By

Didrik Soplér, Ph.D., L.Ac.

Being strong may help us live longer according to a study recently published in the British Medical Journal (Ruiz JR, et al, 2008).

The participants were 8762 men between the age of 20 and 80 years old. Muscle strength was quantified by combining one repetition maximal measurements for leg and bench presses. Cardio respiratory fitness was assessed by a maximal exercise test on a treadmill. The average follow-up was 18.9 years. Adjustments were made for age, physical activity, smoking, alcohol intake, body mass index, baseline medical conditions, and family history of cardiovascular disease.

The investigators concluded that muscular strength is inversely and independently associated with death from all causes and cancer in men, even after adjusting for cardio respiratory fitness and other potential confounders.

It is also interesting to notice that muscle strength seems to affect low-grade inflammation. This was investigated and a connection was found in a study involving 416 adolescents aged 13 to 18 ½ years, 230 boys and 186 girls (Ruiz JR, Ortega FB, et al, 2008). Muscle strength score was computed as the mean of the handgrip and standing broad jump standardized values. The participants were put into categories of overweight or non-overweight according to body mass index. Body fat and fat-free mass were tested using skin fold thickness.

The results showed that C-reactive protein, complement factor C3, and ceruloplasmin were negatively associated with muscle strength in overweight adolescents. It was concluded that low grade inflammation is negatively associated with muscle strength in adolescents. These associations seem more relevant in overweight adolescents, suggesting that having high levels of muscle strength may counteract the negative consequence ascribed to body fat.

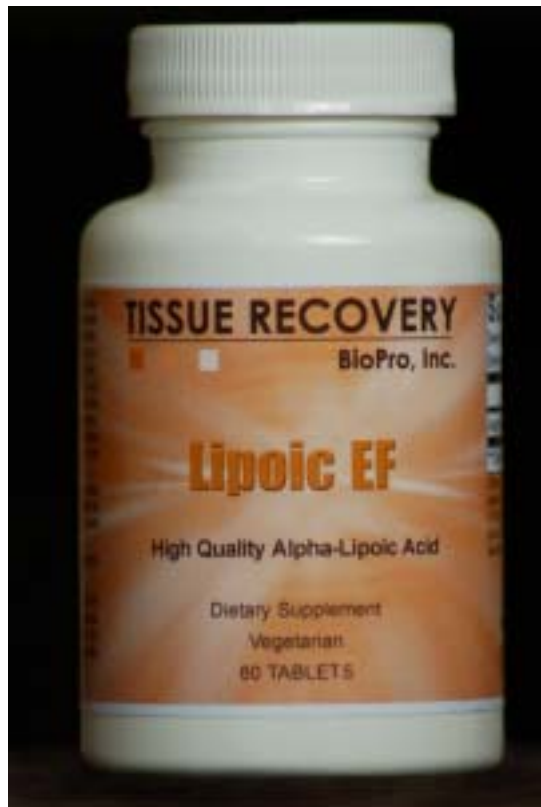
Maybe studies like these will help motivate patients to keep exercising regularly after they are finished with treatment.

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