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Title: Iliotibial Band Syndrome: A Friction Syndrome or a Compression Syndrome?

Running Title: Iliotibial Band Syndrome Etiology

Abstract

For a long time, Iliotibial Band Syndrome (ITBS), a common injury seen in athletes of all different backgrounds, was believed to be due to friction between the iliotibial band (ITB) and the lateral femoral epicondyle of the femur. However, Fairclough et al. 2006 recently proposed a new compression model that looks at the compression of a highly innervated fatty tissue layer beneath the ITB, instead of the pain stemming from the ITB itself. This new model has led researchers to rethink previous beliefs and conduct more research to better understand the syndrome. Under further examination, MRI's back up Fairclough et al. by revealing the presence of inflammation beneath the ITB in a layer of highly innervated fatty tissue that becomes tightly compressed during 30° flexion, an angle that can be reached by an activity as simple as climbing a flight of stairs. Further research has been performed to reevaluate two precursors that have been found to be associated with ITBS in the past, increased peak hip adduction and increased peak internal knee rotation, to determine whether it would be possible for them to lead to the new compression syndrome. Due to the ability of both of these precursors to pull the ITB tighter than it normally would be, it was concluded that they could increase the pressure the ITB places on the highly innervated fatty tissue layer, resulting in the compression syndrome.

Key Words: iliotibial band syndrome, knee rotation, hip adduction, MRI, athletes, friction, compression

Significance

Iliotibial Band Syndrome (ITBS) is a common injury of athletes from many different backgrounds. ITBS has long been believed to be a friction syndrome associated with the Iliotibial Band (ITB) rubbing in an anterior-posterior manner over the lateral femoral epicondyle of the femur (Khaund 2005). More recent research has proposed that ITBS is actually due to the compression of a highly innervated fat layer inferior to the ITB itself (Fairclough et al. 2006). Although much remains unknown regarding ITBS, researchers have been able to establish some potential precursors that may play a role in the development of ITBS. Some of the commonalities noted among ITBS patients include increased peak hip adduction and increased peak knee internal rotation (Aderem and Louw 2015). Researchers have studied how these potential precursors could lead to the onset of the friction believed to be occurring in ITBS, but little is understood about how the precursors may contribute to the newly proposed compression model of ITBS. It is important that we also understand how these precursors cold lead to the compression syndrome of ITBS so that we can improve prevention methods for athletes. In my research, I evaluated the hypothesis of ITBS onset by the compression of a fatty tissue layer, and discussed factors that are believed to be precursors to developing ITBS.

Background

Iliotibial Band Syndrome is a condition where the patient experiences a sharp needle-like pain around the lateral femoral epicondyle, or slightly inferior to it (Lavine 2010). It often presents itself by the presence of an inflamed ITB and, in some cases, a bursa. The significance of the presence of bursae remains unknown, because they are not present in the majority of ITBS cases (Fairclough et al. 2006). However, in a study where a bursectomy was performed on patients with bursas, the pain associated with ITBS was completely relieved (Harris et al. 2009). This

leads to two possible interpretations, either the pain the patient was feeling was actually due to the bursa instead of ITBS, or there are several different forms of ITBS. Other researchers have proposed that bursa are not present, but instead the lateral recess of the knee may be misinterpreted to be a bursa in an MRI scan (Fairclough et al. 2006). Figure 1 demonstrates how the lateral recess might appear as a pocket of fluid, or a bursa. This hypothesis is supported by the fact that bursas have only been found in MRI scans, and never in a dissection of a cadaver with ITBS.

Due to the associated pain, ITBS greatly decreases an athlete's peak performance. This motivated researchers to look for the cause of ITBS to reduce the likelihood of the syndrome occurring in more athletes. While observing the movements of ITBS patients, Noehren et al. (2014) discovered that there was a trend among ITBS patients. They noted that ITBS seemed to be most prevalent in people who had increased peak hip adduction, and increased peak internal knee rotation. These findings gave researchers a starting point in determining the causes of ITBS, and opened new windows for more possible treatments by altering the gait of patients with ITBS.

Recent research has reconsidered the etiology involved with ITBS, and the newest model proposes that it is not a friction syndrome but rather a compression syndrome. MRI scans show an increased intensity in the layer of fatty tissue beneath the ITB, confirming the presence of inflammation in that fatty layer rather than in the ITB itself (Fairclough et al. 2006). This tells us that the pain is not coming from the actual ITB, but from irritation that is occurring in the fatty layer beneath it. At certain degrees of knee flexion, most severely 30°, the layer of highly innervated fatty tissue beneath the ITB becomes compressed and can lead to the pain and inflammation associated with ITBS (Hong and Kim 2013). My research aimed to determine the validity in the compression model and how the factors that have been determined to be

precursors to ITBS, increased peak hip adduction and increased peak internal knee rotation, could lead to the development of ITBS if it is a compression syndrome.

Research Goals and Hypotheses

Goal 1: To determine whether ITBS is a friction syndrome, or a compression syndrome.

The friction form of ITBS would rely on the ITB being a structure that has a wide range of movement. When the ITB is stretched too tightly, the ITB would be pulled and stretched anteriorly and posteriorly over the lateral femoral epicondyle (Khaund 2005). The compression form of ITBS is likely if the ITB is connected to a layer of tissue that is subject to significant changes when the leg is engaged in flexion or extension (Fairclough et al 2006).

> Hypothesis 1: ITBS is a compression syndrome due to the compression of a highly innervated fat layer deep to the ITB itself.

This hypothesis is based on evidence from several studies that provide detailed MRI images of the increased signal associated with ITBS. Additionally, the ITB is actually anchored to the lateral epicondyle of the femur by fibrous strands that don't allow it to move in an anterior-posterior manner (Fairclough et al. 2006). Therefore, the ability of the ITB to move enough to cause friction would be very unlikely.

Goal 2: To determine how precursors to friction ITBS could instead lead to compression ITBS.

Certain precursors have been found to strongly influence whether or not a person develops ITBS, and they include increased peak hip adduction and increased peak internal knee rotation (Lavine 2010). However, most studies examined how these factors lead to the friction of the ITB on the lateral femoral epicondyle. My research looked at whether or not these characteristics that are common in ITBS patients could lead to the compression of the fat layer.

Hypothesis 1: Increased peak hip adduction is still a valid precursor to the onset of compression ITBS.

Hypothesis 2: Increased peak internal knee rotation is a valid precursor to compression ITBS.

Both of the above hypotheses are supported by the conclusion that the ITB is strained when the legs are not moved in a proper manner (Fairclough et al. 2006). Increased peak hip adduction results in an improper stance, and therefore is allowing for the compression of the fat layer when the ITB is tightened. Likewise, increased peak internal knee rotation produces similar effects by pulling the ITB tighter due to the stretching of the ITB in a more medial fashion (Lavine 2010). Tension on the ITB for an extended period of time from either of these factors would result in inflammation from the compression of the fatty tissue layer beneath the ITB.

Discussion and Conclusions

Friction vs. Compression

Distinguishing between friction and compression is a difficult process, because both models would respond to many of the same treatments. Each model of ITBS involves the same symptoms, inflammation and pain. However, the causes of these symptoms are different between models. Most forms of treatment for ITBS target the inflammation as the source of pain. For example, several treatments have been found to have positive effects on ITBS patients including stretching of the ITB, deep transverse frictional massage, cortisone injections, trigger point therapy, and shockwave therapy (Weckstrom and Soderstrom 2016). A few of these types of therapy target the inflammation directly, like cortisone injections and shockwave therapy, but others like deep transverse frictional massage and stretching indirectly improve inflammation by preparing the muscle to respond to excessive stretching better in the future. Although very

beneficial to ITBS patients, this information provides little to no help in diagnosing the real cause of ITBS because these treatments target symptoms that are found in both models, instead of looking at where these symptoms originate from (friction or compression). Different treatments would much more beneficial to understanding ITBS if they aimed to reduce friction via lubrication of the problematic area, or prevented inflammation and pain in the area believed to experience compression by inhibiting the Pacinian Corpuscles (nerves responsible for sensing pressure). Successful trials with either of the previous treatments would validate one model over the other, and allow us to have better insight in developing more specific prevention methods, instead of continuing to use methods based on treating general symptoms.

Recent studies have used MRI scans to pin point the location of the inflammation and provide some insight on which model of ITBS is more valid. Instead of appearing on the actual ITB itself, the higher intensity signal (a marker of inflammation) is actually seen just beneath the ITB in a layer of fatty tissue (Hong and Kim, 2013). Figure 2 displays the high intensity signal in an MRI scan, which indicates the area experiencing inflammation and therefore irritation. This is a strong indicator that the inflammation that results in the pain associated with ITBS is due to the compression of that fat layer beneath the ITB.

Additionally, researchers have performed multiple MRIs on ITBS patients at different angles of flexion to determine what types of movements are causing the inflammation (Fig. 3). Results demonstrate that at around 30° flexion, the layer of highly innervated tissue beneath the ITB gets compressed very tightly (Fairclough et al. 2006). To put this into perspective, a 30° angle of flexion is reached every time a person does something as simple as walking up a stair. This would explain why ITBS is most commonly seen in athletes who run or bike for very long periods of time. Every time that individual bends their knee to 30° flexion, that fat layer

compresses. Repeated compression could cause the tissue layer to become inflamed, which is observed in the MRI images below in Figure 2.

Although MRI scans have proven to be very useful for the understanding and diagnosis of ITBS, standard dissections of the ITB area are also informative. Dense fibrous strands that anchor the ITB to the femur were found in a dissection of five cadavers (Fairclough et al. 2006). These fibrous strands do not allow the ITB to move in the anterior-posterior manner, making it impossible for the ITB to rub over the lateral femoral epicondyle in the way that is suspected in the friction form of ITBS.

The MRI scans alone provide strong evidence that would lead us to conclude that ITBS is more likely to be a compression syndrome than a friction syndrome. A friction syndrome would present with inflammation in the actual ITB from rubbing over the lateral femoral epicondyle (Fig. 1), which is not observed. Additionally, the studies performed on the effect of different degrees of flexion on the anatomy of the ITB and fat layer provide reasonable explanation for why the pain felt with ITBS is felt during certain movements. As Figure 3 demonstrates, there was no change in the ITB during flexion that is constant with the ITB rubbing over the lateral femoral epicondyle, which is an absolute requirement of the friction model of ITBS (Fairclough et al. 2006).

Biological Precursors

Although several factors have been investigated, two factors that have demonstrated an influence on the development of ITBS are increased peak internal knee rotation and increased peak hip adduction. Tateuchi et al. (2015) found that the ITB is stretched tighter when either of these two movements happen. Increased peak hip adduction results in the femur remaining internally rotated more than the average leg, and this results in the ITB being pulled tighter (Tateuchi et al.

2015). The continued abnormal tightening of the ITB restricts the blood flow through the highly innervated fat layer deep to the ITB, and the result is inflammation. The same result is seen when a person demonstrates increased peak internal knee rotation. This does not directly confirm that these two factors increase a person's likelihood of developing ITBS, but it does provide a rationale for how these factors would bring about the symptoms associated with the compression form of ITBS.

Increased peak hip adduction and increased peak internal knee rotation are both movements that would cause the ITB to be pulled more medially during knee flexion than it would in a person with "normal" movements. It has been found using MRI imaging that the medial sloping of the ITB reduces the space occupied by the fatty tissue, and results in tighter compression (Fairclough et al. 2006). This explains why people who exercise regularly with these two incorrect movements seem to be more likely to develop ITBS. Fairclough (2006) explains how these two factors pull the ITB in a more medial position, and leads to the conclusion that both increased peak internal knee rotation and increased peak hip adduction are reasonable precursors in the compression form of ITBS.

Although recent research on ITBS has come a really long way, there is still a lot more to look at. With the new compression model of ITBS, it would be interesting to research whether an individual's body fat percentage would influence their susceptibility to developing ITBS. Theoretically, a decreased body fat percentage would result in a thinner higher innervated fatty tissue layer, reducing the amount of compression that occurs. However, there is no research out there on this idea currently. Additionally, more research on treatments needs to be performed. Treatments for ITBS are not very effective due to the lack of understanding of the syndrome, and many athletes are not finding any significant relief from their pain they are experiencing. We

have a way to treat symptoms temporarily, but the symptoms always return due to the root of the problem not being addressed. Due to how commonly Iliotibial Band Syndrome is seen, it's important that research is continued to enhance our understanding and ability to help people who suffer from the syndrome.

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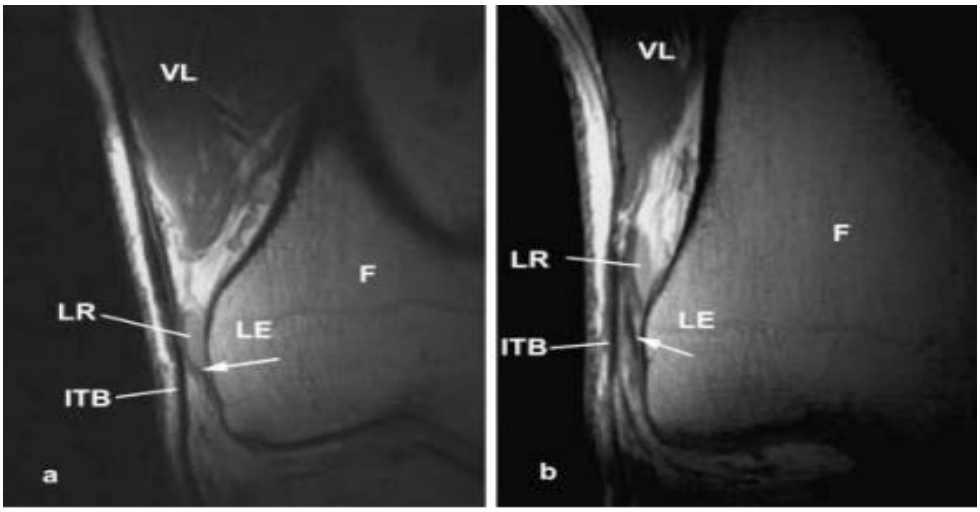

Figure 1. Image shows the lateral recess (LR) of the knee, that is believed to be commonly mistaken as a bursa in ITBS patients. The lateral femoral epicondyle (LE) that is believed to be the structure involved in the friction model of ITBS is also pictured. Adapted from Fairclough et al. 2006

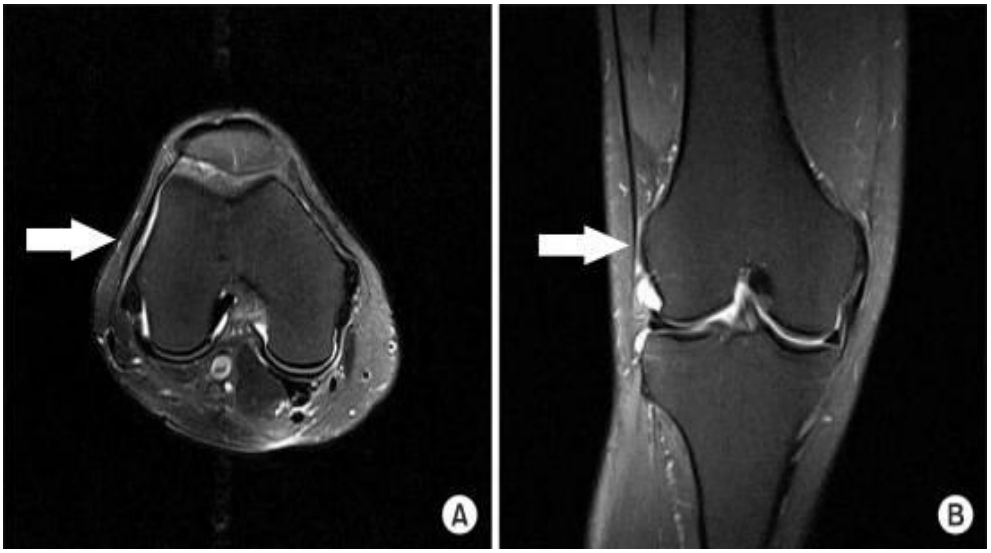

Figure 2. Pictured is an MRI of an ITBS patient with increased signal (inflammation). Inflammation beneath the ITB (black stripe by pointer of arrow) is observed, but not on the actual ITB itself. Adapted from Hong and Kim 2013

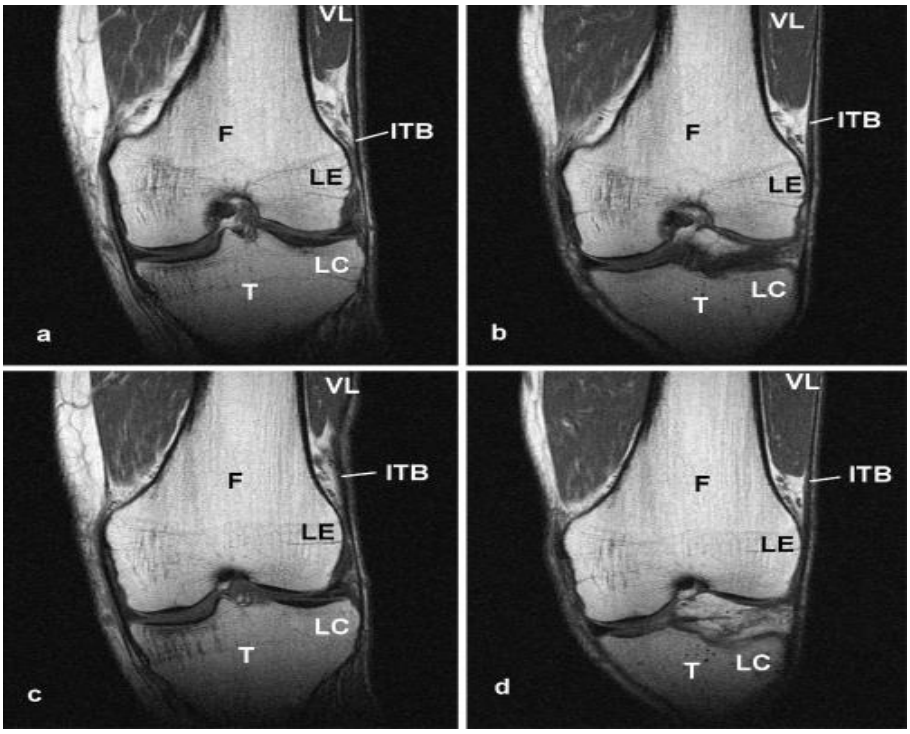

Figure 3. A and C demonstrate a fully extended knee in an ITBS patient, and B and D show the same knee at 30° flexion. When the knee is at 30° flexion, the VL (vastus lateralis) extends and compresses the tissue layer just beneath the ITB (small white pocket). Note that the ITB in A and C slopes laterally (outward), where in B and D, the ITB slopes medially, and results in the compression that we associate with ITBS. Adapted from Fairclough et al. 2006

Literature Cited

Aderem, J., and Q. A. Louw. 2015. Biomechanical risk factors associated with iliotibial band syndrome in runners: a systematic review. BMC Musculoskeletal Disorders 16: 356.

Fairclough, J., K. Hayashi, H. Toumi, K. Lyons, G. Bydder, N. Phillips, T. M. Best, and M. Benjamin. 2006. The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. Journal of Anatomy 20: 309-316.

Harris, S., E. T. Savidge, M. M. Reinold, J. Zachazewski, and T. J. Gill. 2009. Treatment of recalcitrant iliotibial friction syndrome with open iliotibial band bursectomy: indications, techniques, and clinical outcomes. The American Journal of Sports Medicine 37: 1417-1424.

Hong, J. H., and J. S. Kim. 2013. Diagnosis of iliotibial band friction syndrome and ultrasound guided steroid injection. The Korean Journal of Pain 26: 387-391.

Khaund R. 2005. Illiotibial band syndrome: a common source of knee pain. American Family Physician 71: 1473-1475.

Lavine, R. 2010. Iliotibial band friction syndrome. Current Reviews in Musculoskeletal Medicine 3: 1-4.

Noehren, B., A Schmitz, R. Hempel, C. Westlake, and W. Black. 2014. Assessment of strength, flexibility, and running mechanics in men with iliotibial band syndrome. The Journal of Orthopaedic and Sports Physical Therapy 44: 217-222.

Tateuchi, H., S. Shiratori, and I. Noriaki. 2015. The effect of angle and moment of the hip and knee joint on iliotibial hardness. Gait and Posture 41: 522-528.

Weckstrom, K., and Soderstrom, J. 2016. Radial extracorporeal shockwave therapy compared with manual therapy in runners with iliotibial band syndrome. Journal of Back & Musculoskeletal Rehabilitation 29: 161-170.