Research

Etiology, Treatment, and Prevention of ITB Syndrome: A Literature Review

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Abstract

Background: Iliotibial band (ITB) syndrome is a common condition among athletes who participate in sports requiring repetitive knee flexion. Improper biomechanics, weak muscles, and overtraining are contributing factors to the development of ITB.

Objective: This literature review provides an overview of the anatomy, biomechanics, causes, treatment, and prevention of ITB syndrome.

Methods: PubMed (1960-2011) and Sports Science (1960-2011) were searched for sources related to ITB syndrome. Preference was given to more recent articles and journals with higher impact factors when possible.

Results: Forty three sources discussing ITB syndrome anatomy and biomechanics and another 155 sources discussing causes, treatment, and prevention were located out of which 57 were selected for review.

Conclusion: Evidence shows that a variety of intrinsic and extrinsic factors contribute to developing ITB syndrome. Intrinsic factors can often be the result of weak or inhibited muscles, such as the gluteus maximus and gluteus minimus. Extrinsic factors include training habits, including shoe type and increasing training schedules too rapidly. A consensus of literature agrees that proper biomechanics are the critical factor in prevention. Numerous treatment plans have been developed, but none have demonstrated clearly superior outcomes. The best results are obtained with rest during the acute and subacute stages followed by strengthening exercises. More research is needed using objective outcome measures.

INTRODUCTION

The iliotibial band (ITB) is a longitudinal thickening of the lateral distal deep fascia latae and the superficial one quarter of the fibers of the gluteus maximus. The iliotibial tract originates from the proximal iliac crest and inserts on Gerdy's tubercle of the tibia as it passes over the lateral femoral epicondyle. The ITB has several distal attachments, including biceps femoris, vastus lateralis, and the patella. Kaplan et al. [1] completed a study, which concluded quadrupeds do not have an ITB, and therefore an ITB is essential for erect posture.

Iliotibial band syndrome is most often classified as an overuse condition, which is caused by the ITB repetitively gliding over the lateral femoral epicondyle, and in turn inflammation arises secondary to friction. [2-9] Friction is thought to occur as the knee is flexed past thirty degrees and the gluteus maximus pulls the ITB posterior to rest atop the lateral femoral epicondyle. Furthermore, Orchard et al., [10] describes the impingement zone to be at thirty degrees just following heel strike. The heel strike phase is also known as the deceleration phase, and during the weight acceptance portion of this phase the ITB is eccentrically loaded, causing impingement. ITB syndrome is the most common cause of lateral knee pain in long distance runners, cyclists, and similar sports requiring repetitive knee flexion and extension. [1,2,3,8,11,12]

Differential Diagnosis of ITBS

Athletes will frequently complain of pain of the lateral aspect of the knee, and it is often reproduced during the clinical examination. [1,2,3,8,9,11,12] However, the differential diagnosis for lateral knee pain includes degenerative joint disease, lateral meniscal tear, myofascial pain, lateral collateral ligament sprain, biceps femoris tendinopathy, popliteal tendinopathy, fracture, or referral from the lumbar spine, sacroiliac joint, or hip. [9,13] Regularly, patients with lateral knee pain will not be able to bend their knee past a certain degree, which results in an altered gait pattern. [9] The two most common orthopedic tests to rule in or out the diagnosis of ITB syndrome are Ober's test and Nobles compression test. [1,2,3,12, 14] When the doctor performs Noble's compression test, the patient lies supine while pressure is applied to the lateral aspect of the knee. [2,3,15,]
Ober's test is used to assess tightness of the ITB, and the patient often presents with difficulty adducting the affected leg. Ober's test is performed with the patient lying on their unaffected side with the affected knee bent. [2,17] A positive Trendelenberg sign suggest weakness of the gluteus medius. Weakness of the gluteus medius results in compensation further altering lower extremity kinematics and aggravation of the ITB.[3,18,19,20]

Etiology of ITBS

Patients with ITB syndrome often present with faulty biomechanics and/or anatomical factors. There are numerous anatomical factors that may contribute to ITB syndrome including knee, forefoot, and rear foot alignments, Q-angle, ITB tightness, and the size of the lateral femoral epicondyle. There are debates between various studies when considering the occurrence of ITB syndrome in athletes with leg length

discrepancies. When forces are increased and combined with genu valgum, excessive foot pronation, or leg length inequalities increased friction may occur. [1-,3, 21,22]
Many ITB syndrome patients will show changes in lower extremity kinematics, for instance greater peak hip abduction, greater peak knee internal rotation, and femoral external rotation when compared to control groups. Along with that, the contribution of poor muscle performance, such as the hip abductors can further exacerbate faulty biomechanics. [1-,3,21-25]

A variety of altered biomechanical and anatomical factors are often the basis for development of ITB syndrome. This literature review will further discuss the many factors that may predispose an athlete to ITB syndrome and further evaluate the efficacy of treatment plans, protocols, and preventive measure that can be used in a clinical setting. The proper use of orthotics, well maintained hip abductor and adductor strength will be detailed. Along with that, Fredericson and Wolf [3] developed a rehabilitative protocol for each stage of rehabilitation and this protocol will be outlined in depth. The path of recovery may involve the correction of several contributing factors consisting of weakness of the gluteus medius, excessive hip adduction and knee internal rotation, leg length discrepancies, and excessive knee varus and valgus strain. [1,2,3] ITB syndrome is a fairly common orthopedic condition, and when properly indentified and treated will frequently result in full recovery.

METHODS

PubMed (1960-2011) and Sports Science (1960-2011) were searched for publications related to ITB syndrome using search terms treatment of ITB syndrome, prevention of ITB syndrome, biomechanics AND ITB syndrome, correction of ITB syndrome. Both authors (SS and RT) conducted independent searches and then independently screened titles and abstracts selecting articles based on relevance, preference for more current articles, preference for journals with higher impact factors and preference for articles with more citations. In the independent searches SS identified 111 articles and RT identified 93 articles. Authors then compared search results and chose 88 articles to retrieve full-text by consensus. Full text articles were compiled and screened by both authors resulting in 57 articles chosen by consensus for inclusion in the review.

DISCUSSION

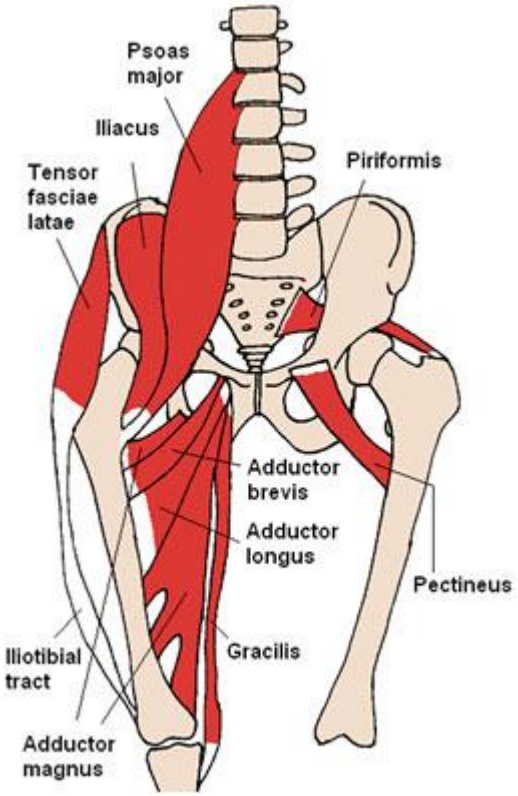
Anatomic Considerations

The ITB functions as a lateral knee stabilizer that arises off of the superficial one quarter of the fibers of the gluteus maximus and it continues as a longitudinal thickening of lateral distal deep fascia latae. This dense fibrous connective tissue goes from the anterior superior iliac spine and has two distal insertion points, while being strongly anchored to the linea aspera of the femur by way of its continuation with the lateral intermuscular septum. The first attachment site of the ITB is at the upper edge of the lateral epicondyle of the distal femur by strong obliquely oriented fibrous strands. The ITB resembles a tendon at the attachment site of the lateral femoral epicondyle with a layer of adipose tissue beneath it. [1-3,7,16,26]

The adipose tissue is highly vascular and richly innervated containing pacinian corpuscles and myelinated and unmyelinated nerve fibers, which suggest this could be the site of inflammation that causes pain when compressed. [2,16]
Between the first attachment site at the lateral femoral epicondyle and the second attachment site on Gerdy tubercle of the tibia the ITB appears to be ligamentous in structure and function. The Gerdy tubercle attachment site is tensed when the knee is in flexion and accompanied by tibial internal rotation during the weight acceptance phase of gait.[1-3,16,27,28]

Furthermore, the ITB has other distal attachment sites, which include the patella via the lateral patellar retinaculum and epicondylopatellar ligament, biceps femoris, and vastus lateralis. As a group, they form an inverted "U," which gives anterolateral support to the knee (**Figure 1**).[7,16,27]

Figure 1. Iliotibial band.



In a study of 15 cadavers, Fairclough et al. [16] examined asymptomatic individuals and athletes experiencing ITB syndrome using magnetic resonance imaging and dissection, and found that no bursa was seen near the distal point of attachment. In addition, Fairclough et al. [16] illustrates the injury as

compression of the ITB and Pacinian corpuscle containing adipose tissue on the lateral femoral epicondyle at thirty degrees of flexion with no anterior to posterior movement of the ITB resulting in an inflammatory injury, rather than a friction injury. [2]

Biomechanics

The ITB continuously moves from anterior to posterior with respect to the lateral femoral epicondyle as the knee flexes and extends during the running cycle. [1,29] The proximal portion of the ITB and tensor fascia latae helps maintain hip flexion in the swing phase by moving anterior to the greater trochanter. In addition, as the hip extends for the duration of the stance and push-off phase, the proximal ITB is pulled over the greater trochanter. Distally, the ITB is pulled over the lateral femoral epicondyle, which helps maintain knee flexion when the knee is flexed past thirty degrees.[1-3,7,30]

While in a static erect posture numerous muscles hold the ITB in place. Proximally, the ITB is posterior to the greater trochanter, this allows for the hip to remain in extension with support from the gluteus maximus and tensor fascia latae. The ITB is positioned anterior to the lateral femoral epicondyle, allowing the knee to stay in extension. [30]

Intrinsic Factors

Numerous anatomical factors predispose athletes to ITBS.[1-3,9,11,12,23,29,31,32] Anatomical factors that could predispose an individual to increased friction over the greater trochanter and lateral femoral epicondyle include, knee, forefoot, and rear foot alignments, Q-angle, ITB tightness, and the size of the lateral femoral epicondyle.[1-3,9,11,12,23,29,31,32] A recent study conducted by Ferber et al., [21] suggests that female runners with greater peak hip adduction angle and greater peak knee internal rotation were more likely to develop ITB syndrome when compared to the controls. Using a retrospective design and control group comparison, the researchers hypothesized the ITB syndrome control group would demonstrate greater stress at the knee due to internal rotation and increased tensile stress at the hip in the frontal plane. Noehren et al. [22] performed a prospective study similar to Ferber et al. [21] In which, Noehren et al. [22] analyzed female runners with ITB syndrome and contributing biomechanical factors, including knee internal rotation, hip adduction, and rear foot eversion angles. The researchers gathered data by way of three-dimensional bilateral analysis of lower extremity kinematics and kinetics. The investigators kept in contact with the subjects through e-mail over two years. The study's results revealed greater peak hip adduction, greater peak knee internal rotation angle, lower tibial internal rotation, and femoral external rotation when comparing the ITB syndrome group to the control group. Noehren et al. [22] and Ferber et al. [21] research shows excessive hip adduction and knee internal rotation in female runner experiencing ITB syndrome.

A prospective study of ITB strain was recently conducted by Hamill et al., [6] in which he used interactive musculoskeletal software to determine ITB strain, strain rate, and extent of impingement in female runners. ITB strain was calculated by taking the change in length during running divided by the resting length. Also, strain rate was measured by taking the change in strain divided by the change in time.

Seventeen patients with ITB strain and seventeen controls of the same age were studied. The investigators examined the experimental and control group's entire gait, while strongly emphasizing touch-down and peak knee flexion. When comparing the two groups' only strain rate was statically significant and this finding suggest strain rate may be a contributing factor in developing ITB strain.

Weakness of the hip abductors, mainly the gluteus medius, can result in a lateral pelvic tilt and excessive strain of the ITB. Fredericson et al.[24] evaluated twenty four runners with ITB strain, and the findings showed that all runners in the study had weakness of their hip abductors in the affected limb when compared to their unaffected limb and controls. Another study of fifty healthy marathon runners was performed by Stanford University Biomotion Lab evaluating peak hip adduction. [2] The runners were followed throughout their training season, and seven of the runners developed ITB syndrome. The results of the study showed that all seven runners had increased peak hip adduction. The study performed by Stanford University Biomotion Lab, further shows that decreased control of the hip adductors will results in an inability to properly control hip adduction through the gait cycle, thus increasing strain on the ITB.

Furthermore, weak hip abductors may also contribute to increased torque at the knee. Noehren et al.[22] established that ITB injured runners have increased internal tibial rotation when compared to the controls. In return, further strain is put on the ITB with increased pressure distally over the lateral femoral epicondyle. [1,6,]

Along with that, a ground reaction force diagram is a one way to visualize the relationship between the hip and knee in the frontal plane. Powers gives a biomechanical perspective of these frontal plane movements during a normal single-limb stance and two possible disturbances that can affect normal single-limb stance. While in a normal single-limb stance, the ground reaction force vector may pass medial to the knee joint and produce varus torque at the knee. In some cases, such as ones with excessive hip adduction the ground reaction forces may pass more medial, with a large perpendicular distance to the knee joint. The consequences are a positive Trendelenburg sign (lateral pelvic tilt), elongated lateral hip musculature, and increased varus torque at the knee. Thirdly, one may develop a compensated Trendelenburg sign where the ground reaction force vector passes lateral to the knee causing valgus stress accompanied by increased hip adduction.[2,33]

As stated previously, Noehren et al. [22] study on the lower extremity biomechanics of female runners who eventually develop ITB syndrome may also have abnormal mechanics at the foot and tibia. His primary findings were excessive hip adduction and knee internal rotation, but he also identified four subjects that also had unwarranted amount of calcaneal eversion. On the contrary, a study by Messier et al. [2] showed no significant difference in calcaneal eversion when comparing the control and experimental groups.

A study conducted by Bauer and Duke [1] examined twenty injured and twenty healthy individuals. Their research looked at the comparisons between health and injured anatomical and lower extremity running kinematics. They hypothesized that individuals with ITB syndrome would have a larger leg

length discrepancy and different running kinematics than healthy runners. All participants were between eighteen and fifty-five years old, and must have ran at least thirteen kilometers over the past year. Runners were asked to fill out a running questionnaire asking for preferred running surfaces, incorporation of other athletics such as swimming, weightlifting, bicycling, stair masters, and stadium steps, and when a subject decided to change running shoes, which included every 4-12 or every 200-600 miles. The study's results demonstrated no difference in knee flexion angle at initial contact, tibial rotation in stance deceleration, or maximum tibial rotation instance when comparing runners with or without a leg length deficiency. Also, fifty six percent of runners in this study experienced ITB syndrome in the shorter leg rather than the longer leg. In contrast, McNicol et al. [32] examined fifty two cases of ITB syndrome and found that thirteen percent had leg length inequalities, and of these thirteen percent all runners had the injury on the long leg side.

Miller et al.[34] conducted a study to evaluate the lower extremity biomechanics during an exhaustive run. His study included sixteen runner, eight with ITB syndrome, and eight age-matched controls. His results, further clarify that runners with ITB syndrome will demonstrate increased maximum foot inversion, maximum knee flexion at heel strike, and maximum knee internal rotation velocity. Collectively, the results of the previous studies show that along with excessive hip adduction and increased internal tibial rotation, abnormal foot and ankle biomechanics can play a role in developing ITB syndrome.

Extrinsic Factors

Development of ITB syndrome correlates with numerous training factors including a rapid increase in weekly mileage, excessive running in the same direction on a track, and running downhill because there is increased friction between the ITB and lateral femoral epicondyle due to decreased knee flexion at foot strike.[3,23,35,36] Orchard et al.[9] suggest that sprinting may help to prevent ITB syndrome because there is greater knee flexion meaning less time is spent in the impingement zone. The author implies that downhill and slow running contribute to the development of ITB syndrome. On the other hand, Miller et al.[34] discounted Orchard et als'[9] theory when he found that during exhaustive run, runners with a history of ITB syndrome will experience greater knee flexion during heel strike. Furthermore, extrinsic factors include type of running shoe and cycle fit. [2,9,23] Further research needs to be conducted on the topic since there are no available studies that evaluate ITB strain and sprinting.

Farrell et al.[38] analyzed kinetic data in relation to cycling kinematics and compared the data to values for running. Ten non-injured cyclists were evaluated with motion analysis and synchronized foot-pedal forces. At the conclusion of the study it was determined that cycling had a lower pedal reaction force of 17-19% when compared to running and cyclists also experienced 38ms in the impingement zone, where as runner spend about 75ms in the impingement zone. However, when investigators evaluated a one and half hour bike ride to a ten kilometer run, cycling produced more repetitions, thus they experienced more repetitive stress than runners.

A theory developed by Farrell et al.[38] proposes that when the shorter leg is fixed to a pedal, the leg is overstretched laterally and functions in less knee flexion, thereby increasing the time spent in the impingement zone.

Prevention

Runners and cyclist should train on level ground every other day.[2,5] Runners and cyclists should monitor themselves for reoccurrence of symptoms, while gradually increasing distance and frequency.[3] Combining cross training activities such as hill running, track running, swimming, and cycling is not recommended owing to repetitive knee flexion throughout the impingement zone.[2,9,23] Orthotics should be considered if your patient is a runner experiencing unwarranted calcaneal eversion and tibia internal rotation when performing functional tasks or they have an anatomical leg length deficiency of 0.5cm or greater.[3,32,37]

Cyclists suffer from repetitive flexion through the impingement zone and toe-in position, therefore it is advisable to check bicycles for proper fit.[3,38] In order to reduce passive stretching of the gluteus maximus and ITB; Wanich et al.[39] suggest lowering the seat beyond the typical height allowing for less knee extension and stress on the ITB. It is also recommend evaluating cleat position and determining if orthotics are necessary to prevent excessive tibial rotation and foot hyperpronation. Flexibility of the hamstrings and gastroc-soleus muscles are emphasized with some importance placed on flexibility of the gluteus maximus and ITB.[2,] Wanich et al. [39] treated 61 cyclists suffering from ITB syndrome by modifying their training programs. Investigators adjusted the bikes to best resemble the cyclists normal off-bicycle alignment, but lowered the seat causing the knee to flex between thirty and thirty-two degrees at the bottom center of the pedaling stroke. Other training modifications included flat terrains, controlled mileage, easy pedaling at 80 revolutions per minute, and pain free.

Clinical Presentation

Subjectively, the chief complaint by patients in a study performed by Sutker et al.[40] was lateral knee pain during exercises involving repetitive knee flexion and extension while being put under a load, as seen in the deceleration phase of running. In the following study, Sutker et al. [40] diagnosed forty eight cases of ITB syndrome following the evaluation of 1030 runner complaining of lower extremity pain. Diagnosis of the forty eight cases of ITB syndrome was concluded upon pain consistent with the patient's history and localized tenderness over the lateral femoral epicondyle. However, patients were able to hop and squat without pain.

An article by Khaund and Flynn[13] describes a clinical presentation of diffuse lateral thigh pain with sharp discomfort of the lateral femoral epicondyle and/or lateral tibial tubercle. They expand on this by stating patients may experience pain at the completion of a run or even a few moments into a run and throughout a run irritation will gradually increase. Along with that patients will often put in their history that they notice an exacerbation of their symptoms while lengthening their stride or sitting for long periods of time with their knees in flexion. In comparison, Fredericson and Wolf [3] show patients to have

a history consistent with symptoms developing after a reproducible time and distance, and typically begin runs pain free. They also note that patients often mention pain with lengthening their stride and sitting with their knees flexed for long time periods. In addition, patients will bring up pain while running down hill and in severe cases pain while walking or going down stairs.

An assessment of cases by Rene,[11] military recruits with ITB syndrome showed a limp accompanied by a straight leg gait, which contraindicates Sutker et als'[40] patients' ability to hop and squat pain free. She further notes, symptoms were aggravated with runs greater than two miles and hikes consisting of ten miles or more.

In various studies results have shown patients mention several training factors, which correlate with the development of ITB syndrome. McNicol et al. [32] found that forty two percent of fifty two subjects with ITB syndrome developed it through errors in training. Training errors included seven cases of a rapid increase in the amount of training, one case was due to over exposure to hills while running, four cases of improper footwear and surface issues, two cases of hurried initiation, and twelve cases of a single rigorous training session. Messier et al. [23] obtained similar results when he evaluated forty eight cases of ITB syndrome and found that patients experiencing ITB syndrome had less experience and had recently increased their training mileage when compared to seventy controls. In conclusion, the clinical presentation of ITB syndrome is consistent with pain along the lateral thigh, associated with excessive training, and aggravated by running while experiencing ITB syndrome, and other sports involving repetitive knee flexion and extension.

Physical Examination

Upon palpation, patients with ITB syndrome will most commonly experience tenderness and discomfort two centimeters above the joint line and the discomfort is typically exacerbated when the knee is bent at thirty degrees of flexion. Less commonly, tenderness may be palpated at the lateral joint line, popliteal tendon, lateral collateral ligament, or anterior lateral fat pad. The affected area may also present with pitting edema, crepitation, or snapping. At the angle an angle of thirty degrees, the ITB is at maximum tension, thus provoking the patient's symptoms. Furthermore, Khaund and Flynn [13] make mention of possible findings including multiple trigger points located in the vastus lateralis, gluteus medius and biceps femoris, which refer pain to the lateral aspect of the affected knee. The examiner should also check for the appropriate strength in hip abductors, knee flexors, and knee extensors.[2,3,15]

There are two common orthopedic tests used to objectively examine a patient. The first of the two is Noble compression test (**Figure 2**). When performing Noble compression the examiner should be able to reproduce the symptoms with compression of the lateral femoral epicondyle while the knee is bent at thirty degrees. However, while performing the examination the patient's knee is bent at ninety degrees and the examiner extends the knee while applying pressure just proximal to the lateral femoral epicondyle. Previously mentioned, the impingement zone is at thirty degrees, which was confirmed in a cadaveric study performed by Orchard et al. [9] and Fairclough et al. [16] Being able to properly identify the impingement zone allows the examiner to determine if the patient is suffering from ITB syndrome or

various other disorders, such as injury to the lateral meniscus, lateral retinaculum, popliteus and biceps femoris tendons, patellofemoral joint, and lateral collateral ligaments.

Figure 2. Noble compression test.

In connection with Noble compression, Ober's test (**Figure 3**) is most often used to examine ITB tightness.[2,3,17] As described by Gose and Schweizer, Ober's test is performed by: (1) positioning the patient on their side with the affected leg up (2) the examiner then flexes the knee to ninety degrees while stabilizing the pelvis, the hip is positioned in a flexed and abducted posture (3) Put the hip in to extension allowing the ITB to slide over or behind the greater trochanter (4) When the affected leg is lowered and adducts without pain it is concluded that the patient does not have ITB syndrome. In the case that the leg remains in abduction the authors details ITB restrictions as: (a) minimal (adducted to the horizontal but not fully to the table), (b) moderate (adducted to the horizontal), and (c) maximal (patient is unable to adduct to horizontal). [2,17] In continuation, the patient may or may not experience pain with the primary indicator of the Ober's test being that the affected leg remains in abduction. In addition, if a patient does experience pain this is the result of excessive friction over the lateral epicondyle while the knee is being flexed and extended. [13]

Figure 3. Ober's test.

With respect to Ober's test, it is recommended to use the modified Thomas test because both can adequately evaluate hip extension and decreased flexibility of the tensor fascia lata, ITB, iliopsoas, and rectus femoris [3,5] The modified Thomas test is performed by having the patient sit close to the edge of a treatment table, holding their thigh to their chest, and rolling on to their back, as the opposite leg hangs off the table. Clapis et al.[41] conducted a study on 42 non-injured subjects measuring subject's joint ranges using a goniometer and inclinometer while performing the modified Thomas test. All subjects were measured with a flat lordosis, which was palpated while measurements were performed, and the patient also maintained a neutral hip during the exam. The instruments were placed proximally at the midline of the pelvis and distally at the midline of the femur distally, and results showed interclass correlation measurements using the goniometer to be 0.92 and when using the inclinometer interclass measurements was 0.89. In a similar study, Harvey [42] had 117 elite athletes in sports including tennis, running, rowing, and basketball perform the modified Thomas test. In this study interclass correlation was .91-.94 respectively.[2] Harvey's findings were (1) psoas averaged -11.9 degrees (below the horizontal), (2) quadriceps was 52.5 degrees, and (3) tensor fascia lata-ITB average 15.6 degrees of abduction.

Associated with ITB tightness is lessened flexibility. Due to biomechanical factors associated with ITB syndrome the muscles that support the hip laterally should be assessed for flexibility. [2,3] Decreased flexibility has been known to be a causative factor of ITB syndrome. Frederickson et al.[43] proposed benefits to stretching by reducing the tension of the ITB, with the ability to alleviate myofascial trigger points and fascial adhesions found in patients experiencing ITB syndrome. In contrast, Messier et al. [23] examined stretching routines in fifty-six runners with ITB syndrome and seventy controls, and his results showed no differences between the two groups.

Furthermore, examination of the gastrocnemius and soleus muscles should be checked for flexibility. In the case that these two muscles are tight, the end result is decreased ankle dorsiflexion, which results in both excessive ankle pronation and knee flexion. Other causes of increased ankle pronation consist of pes planus, compensation for a forefoot varus, metatarsus adductus or femoral or tibial torsion all of which may contribute to ITB syndrome. Pes planus contributes to ITB syndrome by increasing internal

rotation hop the leg and thigh as well as exposing underlying conditions like weakness of hip abductors and hip external rotators.[2,4]

Previously mentioned in the above section, Bauer and Duke [2] and McNicol[32] performed studies on separate occasions examining leg length discrepancies in correlation with ITB syndrome. Results showed a relationship between leg length inequality, but subsequent studies have not shown whether ITB is more common in the longer or shorter leg. It appears that leg length discrepancies are a causative factor of ITB syndrome and should be monitored and treated in coordination with ITB syndrome. However, in order to properly treat ITB syndrome caused by a short leg the examiner must first determine if it is a functional or anatomical short leg. [4] If the patient has a true leg deficiency of greater than 1cm, a heel lift is recommended. [4,35,36,44]

Additionally, excessive friction caused by the ITB rubbing over the lateral femoral condyle, may be intensified directly or indirectly due to myofascial restrictions.[4] These restrictions may arises in the forms of trigger points, muscle contractures, or facial adhesions, and may be the direct cause of lateral knee pain or develop indirectly due to ITB syndrome with the subsequent result of excessive tension on the ITB. [6] In order to confirm trigger points, the doctor must use firm pressure in the area/s of the complaint, which are most frequently located in the vastus lateralis, gluteus minimus, piriformis, and distal biceps femoris muscle, and these trigger point often result in referred pain to the lateral thigh, knee, and sometimes the lower leg. [4,46] The examination is best performed with the patient lying on their side, their affected hip bent at forty five degrees and the knee slightly flexed with a pillow under the leg being palpated. In addition, if myofascial restrictions are found myofascial treatment is indicated, whether there is pain referral or not, but no myofascial treatment is indicated if there is no evidence of contracture, sensitivity, or referral.[4]

Lastly, strength tests are a crucial step in determining if the hip abductors are properly functioning and not the primary cause for the development of ITB syndrome. If during the examination the doctor discovers weakness or inhibition of the gluteus medius, the doctor must further investigate this in order to determine if a patient has developed compensation and improper firing of muscles. Improper firing may result in a patient substituting the gluteus medius for the tensor fascia-latae, quadratus lumborum, or both. Hip abduction can be achieved with flexion and internal rotation of the hip if the compensation is a result of improper firing of the tensor fascia-latae. Upon further examination, the doctor may also note excessive hip hiking due to over-activation of the quadratus lumborum. The correct firing pattern would begin with activation of the gluteus medius followed by the tensor fascia-latae and ipsilateral quadratus lumborum and erector spinae.[3]

EMG is a viable way of detecting muscle imbalance between the tensor fascia lata and the gluteus medius and maximus.[3] As described by Kendall et al.,[47] the tensor fascia lata may substitute for the posterior fibers of the gluteus medius and the hamstrings may substitute for the gluteus maximus. Functional tests allow for assessment of trunk and lower extremity strength including signs of excessive femur internal rotation, ipsilateral hip adduction, and contralateral hip drop while performing the Trendelenburg test (Figure 2).[3,18-20] Yet there is dispute on whether or not one can determine if the

weakness arises from the core stabilizers or hip musculature.[3,18,20]

Gary Gray[48] describes several functional tests that are useful for the evaluation of hip abductor strength. These tests include the single-leg balance, anterior-ipsilateral reach test (**Figure 4**). This test causes the foot to pronate, resulting in hip and lower extremity internal rotation, thus allowing the doctor to assess gluteal strength and range of motion in the sagittal and transverse planes. Fredericson and Wolff[4] further assessed this by using a measuring pole and measuring tape to see how low patients could go and how far they could reach. By measuring how far a patient could reach and how low they could go gave Fredrickson and Wolff the opportunity to examine the patient's ability to pronate the foot and the ability of the entire lower extremity to decelerate motion, examining both sides for fluidity and symmetry of both motion and total distance.

Figure 4. Single-leg balance, anterior-ipsilateral reach test.

Fredericson and Wolff[4] further examined the patients by having them perform single leg balance, frontal-plane overhead reach test (**Figure 5**). This gives the examiner the ability to examine the lateral gluteal region and its' ability to decelerate motion in the frontal plane. The frontal plane is mainly controlled by the gluteus maximus, the gluteus maximus also influences concentric femur external rotation and eccentric femur internal rotation.

Figure 5. Single leg balance, frontal-plane overhead reach test.

When performing the frontal-plane overhead reach test Fredericson and Wolff[4] had the patients stand 51-61 cm from the wall at a right angle and reach overhead using the arm furthest from the wall while the doctor examines fluidity and symmetry of motion in the frontal plane. In order to determine if a patient has tight lateral gluteal muscles caused by hip tightness in the frontal plane, the doctor will look for excessive lateral flexion of the torso. If a patient is able to pass this functional test the doctor will continue to have the patient perform the test moving them further from the wall until a threshold is met. Examining and determining if a patient has ITB syndrome can be a relatively simple and effective process but exclusion of many variables including leg length inequality, flexibility, and strength is key.

A second functional test for the gluteus maximus requires the patient to be prone with the knees flexed to ninety degrees with neutral rotation. While in this position the doctor applies pressure to the lower portion of the posterior femur. The patient should have equal strength bilaterally and should be able to fully resist without a break. As stated in the topic intrinsic factors, weak gluteal muscles will cause lateral pelvic tilt and excessive hip adduction. This can be determining factors when evaluating for ITB syndrome. [3,47,49]

Treatment and Rehabilitation

Frederickson and Wolf[4] have researched and produced an extensive treatment protocol, which focuses specifically on the treatment and rehabilitation of runners who have developed ITB syndrome. They divided treatment and rehabilitation in to four separate categories consisting of an acute phase, subacute phase, recovery strengthening phase, and return to running phase.

Acute Phase:

According to these authors, in the acute phase the primary goal is to reduce local inflammation over the lateral femoral epicondyle caused by ITB friction. Oral nonsteroidal anti-inflammatory drugs have been shown to be effective in reducing pain and inflammation. The use of modalities such as ice massages, phonophoresis, or iontophoresis have also been beneficial in reducing pain and inflammation. However, without modification of activities these treatments are ineffective. Any activity with repetitive knee flexion including running and cycling should be avoided in order to decrease stress at the lateral femoral epicondyle. In some instances all a runner needs to do is discontinue running downhill or running in the same direction. With patients who have ITB syndrome, the only recommended exercise is swimming only using their arms with a pool buoy between their legs. If after three days of treatment, there is still visible swelling a local corticosteroid injection might be recommended as it is helpful in reducing local inflammation.[4]

Subacute Phase:

Following the reduction of inflammation patients may begin a series of ITB focused stretches with a goal of lengthening the ITB. If the lateral gluteal muscles are found to be weak or are functioning improperly then other muscles including the ITB will then have to compensate causing contraction of the ITB. Therefore, it is recommended to perform contraction-relaxation stretches allowing relative lengthening of the shortened muscle groups. These exercises are performed in sets of three, consisting of a seven second submaximal contraction followed by a fifteen second stretch, with the individual's main focus on lengthening the ITB and tensor fascia-latae. When performing the standing stretch, the patient stands upright, using a wall if needed. The symptomatic leg is extended and adducted over top of the uninvolved leg. The patient exhales and slowly flexes the trunk opposite the side of the crossed leg until a stretch is felt on the side of the hip. On the side being stretched it is essential that the foot reaches optimal pronation, which allows the hip to fully load eccentrically. The area being stretched can be varied by extending or tucking the pelvis. Placing the arms overhead during the standing stretch can accentuate the stretch by increasing lateral trunk flexion. The patient may reach out with extended arms and clasped hands while bending downward, allowing for a stretch in the transverse plane (**Figure 6**).

Figure 6. Stretch in the transverse plane.

During the subacute phase and after the acute inflammation subsides, it is necessary to address myofascial restrictions. This is complementary to physical therapy and any restrictions should be attended to prior to muscle strengthening and re-education. Frequently, soft tissue treatment decreases pain and definitively treats the condition. The use of a foam roller on the tight muscles is also beneficial.

In reference to the stretches recommended during the subacute phase, Fredericson et al.[43] conducted research at the Stanford Biomotion Lab in which they evaluated the relative effectiveness of three ITB stretches. Each stretch was measured for change in ITB tissue length and the force generated within the stretched complex. The study assumes that the most effective stretch will increase the length of the tissue and overcome the external moments of the lateral complex.

The three stretches were standing stretches that can be performed without the assistance of an aid. The following stretches were chosen for their common usage, ease of implementation, and prescription. In the standing position, the participant was instructed to extend and adduct the leg being stretched across their other leg. The subject exhales, and slowly laterally flexes the trunk to the opposite side until a stretch is felt around the area of the greater trochanter. Stretch B is similar to stretch A, but the subjects hands are clasped overhead, while laterally flexing the trunk and stretching the arm on the same side as the leg being stretched. Stretch C begins like stretch B with the arms over head, but in stretch C the subjects bend diagonally downward.

All subjects denied ever having a lower-leg injury or surgery that prevented them from participating in a competitive season and each participant performed the stretches in a different sequence to prevent a warm-up phenomenon. The study tested all subjects at the same time of the day before their afternoon

workouts in order to minimize measurement error. X, Y, Z coordinates of six retroreflective markers were placed on lower body landmarks and were captured using a four-camera system. Hip and knee moments were calculated as a product of the ground reaction vector (Newtons) and the moment arm (meters) to the center of the knee and femoral head and all moments were normalized using each subject's height and weight. Subjects were instructed to stretch until they felt a "good" stretch, and hold the stretch for thirty seconds, and during the last 5 seconds of the stretch data were collected. Each subject performed 3 repetitions of each stretch, and each series of stretches that the subject performed was averaged and calculated for the average ITB length and relevant moments. Pairwise 2-tailed Student *t* tests were used to compare average measures for each stretch with statistical significance defined as a *P* value less than .05.

Results showed all three stretches to result in statistically significant improvements, but stretch B was the most effective and most consistent when comparing average adduction moment at the hip and knee and in average ITB length change. Between all three stretches a statistical significance was found in ITB length ($P<.05$). Stretch B increased the length of the ITB by an average of 11.15%, while stretch A lengthened the ITB the least averaging 9.84%. There was not statistical significance between A and B or A and C when evaluating averaging adduction moments at the hip and knee. However, there was statistical significance between B and C when comparing average adduction moments at the hip and knee ($P<.05$). [43]

The study discusses that due to myofascial trigger points, hip abductor muscle inhibition, and fascial adhesions cause increased tension on the ITB, and therefore a stretching protocol should be included in a patient's treatment plan. This helps the tissue return to their functional tissue length and decrease ITB tension. Using the Biomotion Laboratory system, the study found that extending the arms overhead with increased lateral flexion improves the overall effectiveness of the standing ITB stretch, and are simple enough to be implemented in to a clinical setting. The results suggest that there is a 1-2% difference that is physiologically detectable. [43]

Using the Biomotion Laboratory system, adding the over-head arm extension to the average ITB stretch, produced statistically significant difference in ITB length. However, there was no direct measurement of the ITB, which suggests the gluteals, tensor fascia lata, vastus lateralis could have contributed to the changes. Overall the studies errors were minimized by the use of Stanford's Biomotion Laboratory, comparisons were between individuals lowering the possibility of systematic errors, stretching was static preventing skin motion errors, and markers were placed by a highly trained staff.[43]

Recovery Strengthening Phase:

Following the subacute phase, and resolution of all myofascial restrictions, trigger points, and full range-of-motion is established, the recovery strengthening phase may begin. For all exercises it is recommended to start with five to eight repetitions and gradually build to two to three sets of fifteen repetitions, being sure to perform the exercises bilaterally even if only one leg is symptomatic. Several studies focused on concentric side-lying leg lifts that then progress to single-leg balance, step downs,

and pelvic drop exercises.[3,4] The majority of these exercises focus on strengthening the gluteus medius and maximus.

EMG studies have guided therapeutic exercises programs, by showing activation of the gluteal muscles. Distefano et al.[53] conducted a study evaluating maximal voluntary isometric contraction in 21 healthy subjects, who performed open and closed chain exercises focusing on the gluteus medius and gluteus maximus. Specific positions that encourage gluteal recruitment were chosen and include vertical tibia with lunging and forward trunk by hip flexion with squat activity. Comparable exercises included clam shell, lateral band walks, side-lying hip abduction, single-limb squats, single-limb dead lift, multiplanar lunges, and multiplanar hops. The intra-class correlation for all exercises previously mentioned, except for multiplanar hops, were 0.85-0.98 for gluteus maximus and 0.93-0.98 for gluteus medius. The requirement for a strengthening exercise proposed by the investigators was 60% or greater normalized EMG during maximal voluntary isometric contraction. The single-limb dead lift caused the greatest activation of the gluteus maximus, but maximal voluntary isometric contraction only reached 59%. The gluteus medius demonstrated 61% activity during lateral band walk, which increased to 64% when performing a single- limb squat. In addition, side-lying hip abduction produced the greatest contraction of the gluteus medius displaying 81% activity. In comparison, the clam shell without a resistance band only obtained 38-40% activation. This study used EMG patterns to support the use functional-based exercises and open chain resistance exercises to strengthen gluteal muscles.

Fischer and Houtz [54] proposed that the position of the trunk and degree of knee flexion may change the EMG in the gluteus medius and gluteus maximus. They examined 11 healthy women between the ages of fifteen and twenty-three years of age. EMG activity was measured in the gluteus maximus, sacrospinalis, medial and lateral hamstrings, and quadriceps femoris muscles. Measurements were taken while the subjects performed a floor-to-waist lift of twenty-five pounds with the knees straight and the trunk and hips flexed versus hips and knees flexed. The investigators found very little gluteus maximus activation with the knees and tibias forward, but rather strong quadriceps activation. The straight knee and trunk flexed with a twenty-five pound weight demonstrated strong activation of the hamstrings with minimal activation of the gluteus maximus and quadriceps. Along with that, the sacrospinalis muscles showed activation in both lifts. The results of Fischer and Houtz differ from Distefano, [53] but this may caused by a greater forward position of the tibia as seen in Fisher and Houtz study, respectively. [2] In addition, Distefano et al. [53] chose unilateral limb activities and multiplanar exercises while Fischer and Houtz [54] examined bilateral leg activity in the sagittal plane. Clinical significance is placed upon the correlation between proper biomechanics during functional exercise and strengthening of the gluteus maximus.

Furthermore, side-lying hip abduction and pelvic drops are exercises that have been researched specifically for use in the treatment of ITB syndrome. In the study conducted by Distefano et al. [53] side-lying hip abduction showed strong EMG activation, and the single-leg functional activity exhibited higher EMG activation when compared to double-leg closed chain exercise. Further, they made minor adjustments during functional exercises to recruit the gluteal muscles, such as more vertical tibia, forward trunk activity, and proper trunk position. It is recommended to perform the clam exercise and

lunge patterns using a resistance band because without resistance there was less than 60% gluteal activation.

A study performed by Barrios et al.[55] examined eight non-injured subjects with varus knee alignment between eighteen and thirty-five years of age. The study obtained visual faded feedback, which focused on reducing excessive knee external adduction. Each subject went through eight training sessions with faded feedback in sessions five through eight. During the sessions, the subjects were verbally instructed to "bring the thighs closer together" and "walk with your knees closer together." Clinical significance of 20% reduction in knee external adduction moment was noted. While the subjects used were non-injured, these results may be beneficial for ITB syndrome patients demonstrating excessive knee varus. Also, real-time visual feedback allows for biomechanical improvement in eight sessions and this is due to the strong cognitive component provided by visual feedback.

EMG studies, exercise research for ITB syndrome, and case studies have allowed researchers to provide a progression of exercises focused on strengthening the gluteal muscles. Recommendations begin with therapeutic exercises including an ITB stretch, side-lying hip abduction, and pelvic drops with progression to technique-driven closed chain exercises; for instance a single-leg dead lift. Single-leg functional exercises are advised because of their high vigor, and ability to strengthen the gluteal muscles, whereas bilateral closed chain exercises are of lower vigor and are mainly used to develop technique.

In addition, Fredericson and Wolf [4] expanded ITB syndrome treatment protocol with the inclusion of exercises focused on greater eccentric muscle contractions, triplanar motions, and integrated movement patterns. Such exercises include the modified matrix (**Figure 7**), which can be performed by having the patient stand tall with the left foot to twelve o'clock position and the right foot to the three o'clock position. Next, the patient puts their right arm in an abducted and externally rotated position, and then the patient rotates their hips toward the left leg and transfers their weight to the left leg, while the patient also reaches with their right arm to a point between the left hip and knee. The patient should be instructed to lower the hips as the spine flexes so that loading is felt in the hips, legs, and lower back. The patient should then return to the starting position being sure to transfer their weight back to their right leg. The authors also recommend wall bangers (**Figure 8**), in which the patient stands 15-30cm from the wall, depending on flexibility and strength of the lateral gluteal muscles. The right shoulder is closest to the wall; the patient reaches out to the left, while rotating the hips toward the left foot. The patient should maintain a neutral spine by flexing the knees and dropping the hips. As the patient 'bangs' the right hip against the wall, they should immediately recoil in order to sustain eccentric loading, and return to the stand-tall position. It is important not to allow the patient mover their right hip toward the wall.

Figure 7. Modified matrix exercise.

Figure 8. Wall bangers exercise.

In coordination with the following exercises, frontal plane lunges are suggested (**Figure 9**). With a focus on the gluteal muscles, the patient stands with their feet shoulder-width apart and steps to the nine o'clock position until a stretch is felt then immediately returns to the starting position. Variations can be added to the frontal plane lunge allowing for development of the supinators and pronators of the loading leg. This is accomplished by performing a contralateral reach to strengthen the supinators and activation of the peroneal muscles, which is the result of supination that occurs due to external rotation

of the distal lower extremity during the reach. Medial reach allows for pronation of the subtalar joint and internal rotation reaction of the tibia, femur and hip, which results in strengthening the pronators of the loaded leg.[4]

Figure 9. Frontal plane lunges.

Return-to-Running Phase:

As a general rule, resuming participation in sports depends upon being able to properly perform all strengthening exercises without pain. [3,4] Another possible outcome measure is testing the gluteal muscles for proper strength and function. [3,47,49] Previously mentioned, Ober's test can be performed to evaluate hip adduction range of motion, and the modified Thomas test may be used to assess ITB and rectus femoris flexibility. [3,17,41,42] Fredericson and Wolf [4] recommend pain free range of motion in hip adduction before returning to sports. Also, there should be a negative Noble compression test, which is confirmed by no tenderness at the lateral femoral epicondyle when the knee is bent at thirty degrees. [3,4,15]

Surgical Intervention

There are various surgical techniques developed to help decrease tension on the ITB and pressure on the lateral femoral epicondyle. The most common surgical technique removes a triangular section of the ITB that overlies the lateral femoral epicondyle, while the knee is bent at thirty degrees. This technique is also known as Z-lengthening. Most individuals are allowed to return to activities following removal of

the sutures. Outcome data is relatively poor although the majority of cases have shown successful results. Surgical treatment is a last resort due to the effectiveness of conservative treatment. [4,15, 57]

CONCLUSION

ITB Syndrome is a common injury among individuals who participate in sports requiring repetitive knee flexion. It is known that improper biomechanics caused by weak muscles such as the gluteus medius will greatly contribute to the development of ITB syndrome as a result of excess adduction. A clinician should be able to easily determine if ITB syndrome is present by performing tests such as Noble, Ober's, modified Thomas, and Trendelenburg.

A considerable amount of research has been conducted in order to understand ITB syndrome. A PubMed search generated over 100 articles discussing how patients develop ITB syndrome and the best treatment options. There is a lack of research pertaining to soft tissue mobilization techniques.

The phases of rehabilitation recommended by Fredericson and Wolf [4] demonstrate how to effectively treat ITB syndrome through a biomechanical approach focusing on integrated movements and triplanar motions to strengthen the lateral hip musculature with inclusion of massage. With an early diagnosis, a complete recovery is expected. However, for complete resolution of ITB syndrome without remission requires correction of all contributing biomechanical factors.

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