

The Dilemma of Knee Osteoarthritis and Iliotibial Band Syndrome

Abstract

In the majority of cases, acute exacerbation of lateral knee pains is associated with medial joint collapse and varus deformity in the absence of any joint effusion. In the elderly, this is usually associated with difficulty in walking and performing activities of daily living. However, in younger age-groups and athletes, this is usually as a result of extra-articular manifestations. This is primarily due to tight iliotibial band syndrome associated with chronic muscular spasm due to magnesium deficiency and long standing pains. Treatment of the underlying cause (primarily iliotibial band syndrome) of the lateral and posterior knee pain would result in improvement in pain. For these patients, this would also result in improved walking ability. This paper argues that a considerable volume of patients are receiving inappropriate and unnecessary treatment due to inadequate history taking and examination. We argue that healthcare providers must be vigilant in patients with clinical and radiological signs of severe osteoarthritis.

Keywords: Osteoarthritis; Lateral femoral epicondyle

Review Article

Volume 2 Issue 4 - 2015

Safaa-eldin Abaza*

Orthopaedic Surgeon, UAE

***Corresponding author:** Safaa-eldin Abaza, Sam Nashi
Consultant Orthopaedic Surgeon, Gulf Diagnostic centre
Hospital, Abu Dhabi P.O. box 30702, Abu Dhabi, UAE, Tel:
00971-50 4146390; Email: safa.abaza@yahoo.com

Received: January 3, 2015 | **Published:** April 29, 2015

Abbreviations: OA: Osteoarthritis; IGF-1: Insulin-Like Growth Factor; TGF- β : Transforming Growth factor

Discussion

Osteoarthritis (OA) is a common degenerative disorder with complex underlying pathophysiology. In nomenclature, OA is derived from 3 Greek words; 'osteo' – of the bone, 'arthro' – joint and 'itis' meaning inflammation. OA is a disorder involving the synovial joint characterized by focal areas of articular hyaline cartilage damage, mild synovitis, subchondral bone sclerosis and osteophytosis. In OA of the knee, the medial compartment is almost 10 times more likely to be involved compared to the lateral compartment due to mechanical stresses. Although poorly understood, the pathogenesis is thought to involve extracellular activation of matrix metalloproteinases leads to degradation of collagen and proteoglycans. Disturbance of tissue inhibitors and contributes to cartilage loss and subsequent OA. Synovial inflammation results in the production of interleukin-1 and tumour necrosis factor α which further exacerbates this process. Lastly, deficiency of growth factors involved in collagen synthesis, including insulin-like growth factor (IGF-1) and transforming growth factor (TGF- β) may play a role in impairing matrix repair. Unlike other connective tissues, hyaline cartilage does not possess a vascular, lymph or sensory supply. Joint pain resulting from OA usually results from subchondral nerve stimulation and micro-particle chemical synovitis and subsequent inflammation. A number of risk factors have been implicated in the development of OA. Twin studies reveal a 40-60% genetic link in the development of OA. Constitutional risk factors including ageing (prevalence of 30% in 65 year olds), female gender, obesity, and high bone density have all been heavily linked to its development and progression. Finally, biomechanical risk factors including previous joint injury, repetitive stress injury, joint laxity and mal-alignment and reduced muscle strength are deemed significant risk factors.

Interestingly, clinical severities of symptoms are poorly correlated with structural and radiological change. The

prevalence of subchondral marrow edema, knee joint effusion and narrowing of articular cartilage does not accurately correlate with the clinical picture [1,2] correlated VAS pain scores with MRI OA severity. Their research concluded that changes in synovitis were associated with knee pain, although interestingly not the loss of articular cartilage. The iliotibial band is a lateral thickening of the Tensor Fascia Lata. It originates from the anterior iliac crest outer lip, anterior border of the Ilium, and the outer surface of anterior superior iliac spine. It encloses the tensor fasciae lata, anchoring this muscle to the iliac crest and receives most of the tendon of gluteus Maximus. Its insertion is on the anterolateral of the thigh, approximately one third of the way down. It inserts proximally into the lateral epicondyle of the femur then passes in its broad expansion between lateral aspect of patella. Finally, it distally inserts on Gerdy's tubercle on the lateral aspect of tibial tubercle. ITB friction syndrome is an overuse injury and is a well-recognized cause of lateral knee pain in runners and cyclists. Approximately 12% of cases in such groups are attributed to ITB friction syndrome.

It plays an important role in the movement of the thigh by connecting hip muscles to the tibia. Between 2 to 25% of physically active individuals, and approximately one quarter of those presenting to a sports medicine clinic for knee pain were cyclists who were diagnosed with ITBS, highlighting its clinical significance [1-6]. It was originally thought that ITB is a friction syndrome with an anterior-posterior direction movement over the lateral femoral epicondyle. Anatomical studies by Fairclough et al. [7,8] disclosed the presence of anchorage of the ITB to the lateral intra muscular septum and it is impossible to move forward and backward. Instead, it can move lateral to medial leading to compression load to the loose soft connective tissue and fat rich in innervation beneath the tract, with the range of movement of the knee about 20-30 degrees.

The diagnosis of ITB friction syndrome is based on clinical examination and radiological evidence (MRI), with tenderness over the lateral femoral epicondyle. Patients report a sharp, burning

pain when the practitioner presses on the lateral epicondyle during knee flexion and extension. The Ober test for distensibility of the iliotibial band is also frequently a measurement of interest. Medial compartment cartilage loss leads to varus deformity and alter the biomechanics of the knee with varus deformity leading to increased compression of ITB over the lateral femoral condyle. A study by Farell [9-11] established the association of ITBS with genu varum in runners. It also highlighted the frequent presence of MR signs of ITBS in patients with isolated medial compartment knee osteoarthritis. This can be an explanation for the presence of lateral knee pain, when lateral knee compartment is unaffected. In 2009, Vasilevska et al. [12] stated that ITBF is unrecognized cause for lateral knee pain in patients with medial compartment knee osteoarthritis. Either ITBFS has been shown to cause lateral knee pain in athletes, it may be a consequence of gait changes induced by knee OA and may occur together with symptomatic knee OA

Conclusion

Advanced varus deformity of the knee due to a reduction in cartilage thickness and severe degeneration of the Meniscus is associated with chronic muscular spasm. This is primarily due to Magnesium deficiency because of age and associated chronic illness e.g. diabetes. Both may be the cause for stretching and tightness of the ITB, and therefore the reason for acute ITBS with subsequent lateral and posterior knee pain. This may radiate proximally and distally simulating the signs and symptoms of sciatica. Patients usually present with both acute iliotibial band syndrome and varus medial osteoarthritis but without synovitis or effusion. We are of the firm view that treatment of acute iliotibial band syndrome with local steroid injection and magnesium supplements may suppress the pain and improve the patient's ability to mobilize in the presence of varus deformity.

References

1. Barber FA, Boothby MH, Troop RL (2007) Z-plasty lengthening for iliotibial band friction syndrome. *J Knee Surg* 20(4): 281-284.
2. Burgkart R, Glaser C, Hyhlik-Dürr A, Englmeier KH, Reiser M (2001) Magnetic resonance imaging based assessment of cartilage loss in severe osteoarthritis: accuracy, precision and diagnostic value. *Arthritis Rheum* 44(9): 2072-2077.
3. Center for disease control and prevention (CDC) (2003) Public health and aging: projected prevalence of self-reported arthritis or chronic joint symptoms among persons aged >65-United States, 2005-2030. *MMWR Morb Mortal Wkly Rep* 52(21): 489-491.
4. Cicutini FM, Wluka AE, Stuckey SL (2001) Tibial and femoral cartilage changes in knee osteoarthritis. *Ann Rheum Dis* 60(10): 977-980.
5. D'Ambrosia RD (2005) Epidemiology of osteoarthritis. *Orthopedics* 28(2 Suppl): S201-S205.
6. Ekman EF, Pope T, Martin DF, Curl WW (1994) Magnetic Resonance Imaging of Iliotibial Band Syndrome. *Am J Sports Med* 22(6): 851-854.
7. Fairclough J, Hayashi K, Toumi H, Lyons K, Bydder G, et al. (2006) The functional anatomy of the iliotibial band during flexion and extension of the knee: implications for understanding iliotibial band syndrome. *J Anat* 208(3): 309-316.
8. Fairclough J, Hayashi K, Toumi H, Lyons K, Bydder G, et al. (2007) Is iliotibial band syndrome really a friction syndrome?. *J Sci Med* 10(2): 74-76.
9. Baranyay F, Wang Y, Wluka A, English D, Giles G, et al. (2007) Association of Bone Marrow Lesions with Knee Structures and Risk Factors for Bone Marrow Lesions in the Knees of Clinically Healthy, Community-Based Adults. *Semin Arthritis Rheum* 37(2): 112-118.
10. Burgkart R, Glaser C, Hyhlik-Dürr A, Englmeier KH, Reiser M (2001) Magnetic resonance imaging-based assessment of cartilage loss in severe osteoarthritis: Accuracy, precision, and diagnostic value. *Arthritis Rheum* 44(9): 2072-2077.
11. Bolen J, Helmick CG, Sacks JJ, Langmaid G (2002) Prevalence of Self-Reported Arthritis or Chronic Joint Symptoms among Adults-United States, 2001. *JAMA* 288(24): 3103-3104.
12. Vasilevska V, Stäbler A, Samardziski M, Ulrike Szeimies (2012) Knee Osteoarthritis and Associated Periarticular Conditions: Iliotibial Band Friction and Baker Cyst. *INTECH Open science* 253-264.