

ENTHESOPATHIES AND ENTHESITIS IN HUMANS



India's vision of a healthier globe emanates from the philosophy of Vasudhaiva kutumbakam which translates into One Earth, One Family, One Future. So, as part of India's G20 presidency, policymakers from member countries and experts from the medical fraternity shall unfold the foundation and framework of Global Health Architecture (GHA) at a series of Health Working Group meetings, starting this month. GHA envisages events equipping countries to face the next health emergency with robust healthcare systems."

- Dr. Mansukh Mandaviya (Honourable Health Minister said of India at the G20 Summit)

In the twentieth century the term "**Enthesis**", was introduced to refer to the sites where tendons, ligaments, fasciae, and joint capsules attach to bones ensuring a reduction in mechanical stress on the border of tissues with various strengths and elasticity. They are of two types: fibrous and fibrocartilaginous.

'**Enthesitis**' is the term used to describe inflammation at the entheses, crucially with or without swelling as explained by McGonagle & Benjamin, 2009 [2].

'**Enthesopathy**', is defined as the pathologies that affect the entheses, as described by Sudot-Szopińska & I. Kwiatkowska, 2015 [3].

'**Tendinopathy**' is a degeneration of the collagen protein that forms the tendon, whereas '**Tendonitis**' is just inflammation of any tendon of the body.

'**Tenosynovitis**' is a condition in which there is inflammation of a tendon along with its synovial sheath.

'**Enthesis Organ Complex**' a term coined by Benjamin et al (2004), is now the preferred reference for entheses-related pathology, incorporating more of the surrounding structures in the disease process like the bursa, fat pad, adjacent trabecular bone and perhaps even deep fascia [1].

1.) Plantar Fasciitis (PF):

CLINICAL PRESENTATION:

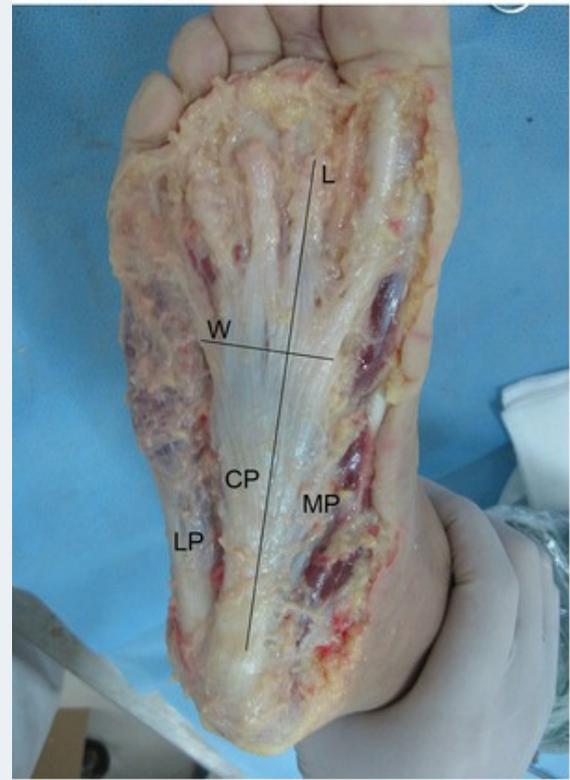
It presents as acute or chronic heel pain in both young, active, and also in older, more sedentary individuals. This is common especially in persons wearing high heels or using heavy, hard (rough and tough) footwear for longer periods and persons walking on uneven surfaces.

UNDERLYING PATHOPHYSIOLOGY:

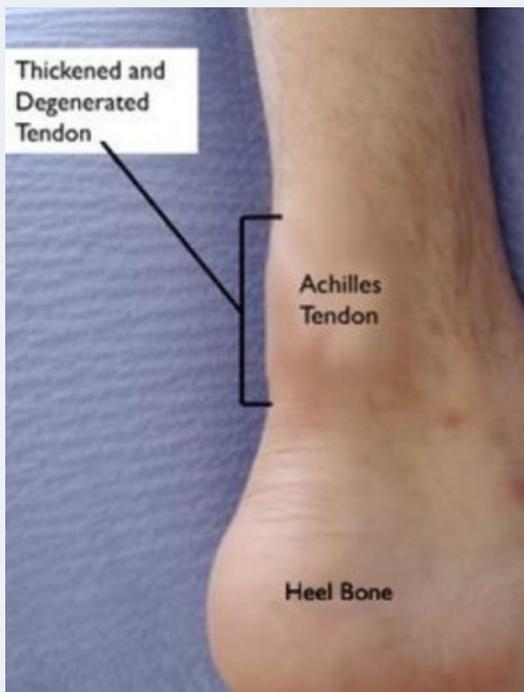
Classically, PF was thought to occur from a mechanical injury in which excessive tensile strain within the plantar fascia produces microscopic tears leading to chronic inflammation. However, the current understanding is that PF occurs through a degenerative rather than an inflammatory process, a “fasciosis,” rather than a fasciitis [4].

ANATOMICAL BASIS:

PF is a thick band of fibrous connective tissue originating from the medial tubercle of the calcaneus and inserted into the bases of the proximal phalanges. Histologically, the plantar fascia bears resemblance to both tendon and ligament with a relatively inelastic extracellular matrix composed of collagen fibers in a wavy or crimped pattern, produced by elongated fibrocytes embedded in longitudinal rows [4].



Courtesy: Plantar fascia. (2022, December 19).
In Wikipedia. https://en.wikipedia.org/wiki/Plantar_fascia



Courtesy: Stover Physical therapy Blog: Heel drops to tolerance for midportion Achilles tendinosis. 06/15/2015

2.) Achilles Tendonitis

CLINICAL PRESENTATION:

The patient presents with localized pain and swelling of the Achilles tendon and loss of function.

UNDERLYING PATHOPHYSIOLOGY:

The underlying cause is either degeneration or failed healing due to continuous overload without appropriate recovery. It can be at insertional, mid-portion, and proximal Musculo-tendinous sites.

ANATOMICAL BASIS:

A sheath-like structure comprised of a single layer of cells surround the tendon; called the ‘paratenon’. The paratenon supplies a significant portion of the blood supply to the tendon. Studies have shown an area of hypovascularity 2 to 6 cm proximal to calcaneal insertion, a common injury area. Histologically, localized or diffuse increases in the thickness of collagen fibers, loss of normal collagen architecture, an increased amount of proteoglycans, and general breakdown of tissue organization is observed [5]

3.) Adhesive Capsulitis:

CLINICAL PRESENTATION:

The patient presents with a painful and limited range of motion at the Glenohumeral joint with or without previous history of trauma [6].

UNDERLYING PATHOPHYSIOLOGY:

Initially, it was thought to be a fibrotic disorder, as histology of affected specimens primarily showed fibroblasts mixed with type I and type III collagen. Recent research indicates lowered expression of MMP-1 and MMP-2 in patients with adhesive capsulitis; at the same time, expression of tissue inhibitors of metalloproteinases (TIMPs) such as TIMP-1 and TIMP-2 is elevated. Hence, adhesive capsulitis is the result of an imbalance between extracellular matrix tissue degradation, remodeling, and regeneration [6].

ANATOMICAL BASIS:

Inflammation causes loss of the synovial layer of the capsule; reduced capsular volume; contracture of Glenohumeral capsule; adhesion of axillary fold to itself and anatomical neck of humerus; thickened and fibrotic rotator interval; contracted coraco-humeral ligament (CHL). Fibroblasts with the ability to transform into smooth muscle phenotype (myofibroblasts) lead to contracture.



Courtesy: Singapore Sports and Orthopaedic Clinic / Brain & Nerve Specialist. Manipulation under Anaesthesia (MUA) for Frozen Shoulder.

4.) Bicipital tendonitis:

CLINICAL PRESENTATION:

The patient presents with mild anterior shoulder pain with a normal range of motion and no trauma history with tenderness and weakness in the tendon of the long head of the biceps brachii. The pain radiates to the anterior aspect of the arm.

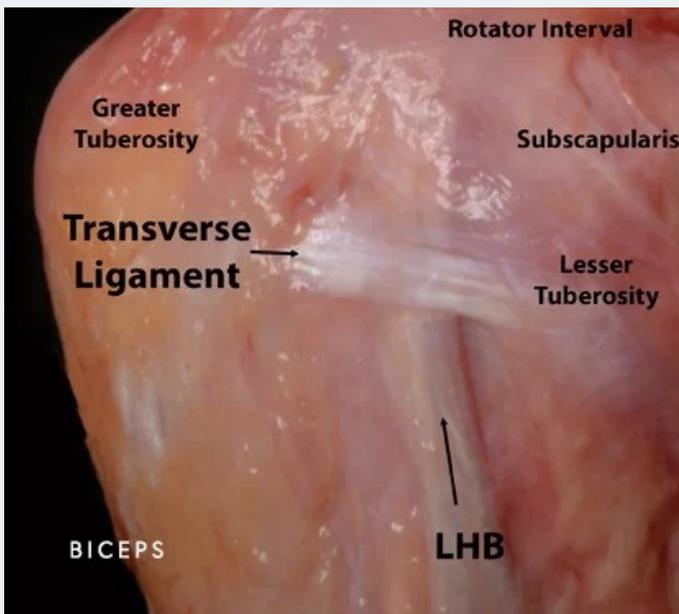
UNDERLYING PATHOPHYSIOLOGY:

The condition begins with the early stages of tenosynovitis and inflammation secondary to repetitive traction, friction, and shoulder rotation. The tendon increases in diameter secondary to swelling and associated haemorrhage, further compromising the tendon as it becomes mechanically irritated in its confined space.

In the later stages of tendonitis, long head of biceps (LHB) sheath thickening, fibrosis, and vascular compromise ensue. The LHB tendon undergoes degenerative changes, and associated scarring, fibrosis, and adhesions eventually compromise LHB tendon mobility [7].

ANATOMICAL BASIS:

Primary biceps tendinitis occurs in 5% of cases, while 95% are accompanied by rotator cuff tears or labral tears. The sheath of the biceps tendon is a direct extension of the synovial lining of the glenohumeral joint. Thus, concomitant or pre-existing rotator cuff pathology can directly compromise the LHB tendon itself.



Courtesy: Dr. Jorge Chahla. Biceps Tendonitis Chicago.IL
What causes biceps tendonitis?

5.) Lateral epicondylitis

(Tennis Elbow):

CLINICAL PRESENTATION:

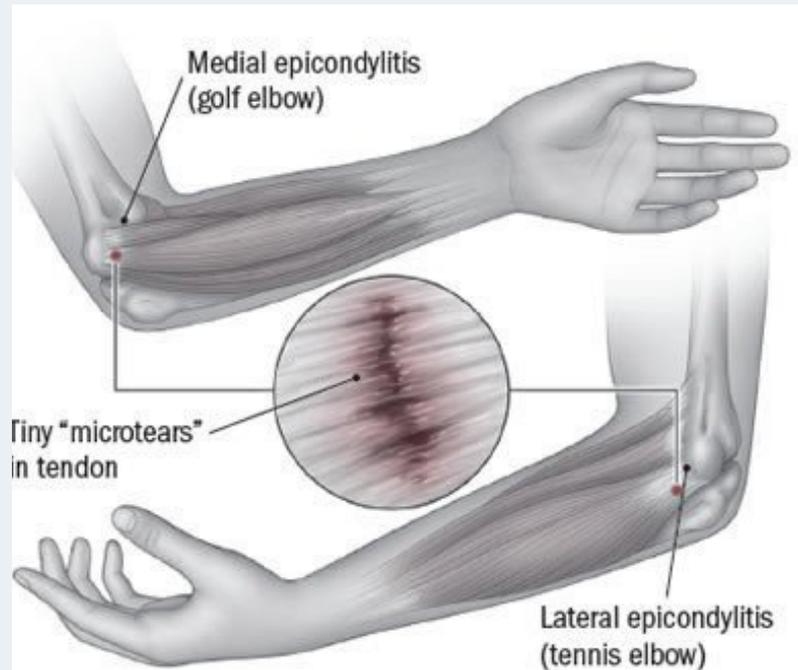
The patient presents with pain located just anterior to, or in, the bony surface of the upper half of the lateral epicondyle, usually radiating in line with the common extensor mass. The pain can vary from intermittent and low-grade pain to continuous and severe pain which may cause sleep disturbance.

UNDERLYING PATHOPHYSIOLOGY:

Repetitive overuse/stress lead to repetitive stretching of the tendons and muscles resulting in multiple micro tears leading to degenerative changes within the tendon known as tendinosis.

ANATOMICAL BASIS:

Extensor carpi radialis brevis (ECRB) is the most commonly affected muscle. Histological changes such as Angio-fibroblastic hyperplasia (a manifestation of granulation tissue that disturbs correct collagen synthesis) can also be seen. Histopathological studies of ECRB in patients with long-standing lateral epicondylitis have shown necrosis as well as signs of fiber regeneration.

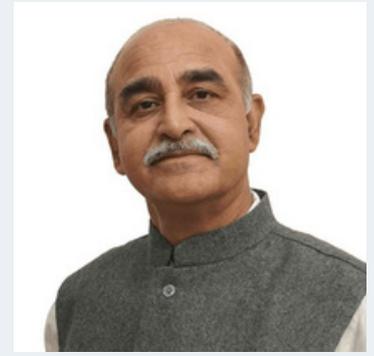


Courtesy: John Hopkins Medicine Home: Health: Lateral epicondylitis (Tennis elbow)

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MESSAGE FROM EXECUTIVE DIRECTOR
PROF.DR. (COL.) CDS KATOCH, AIIMS RAJKOT



I heartily congratulate the Department of Anatomy for bringing this informative newsletter on 'Enthesopathies and Enthesitis in Humans'

My best wishes to the entire team.

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