

Enthesopathy – a personal perspective on its manifestations, implications and treatment

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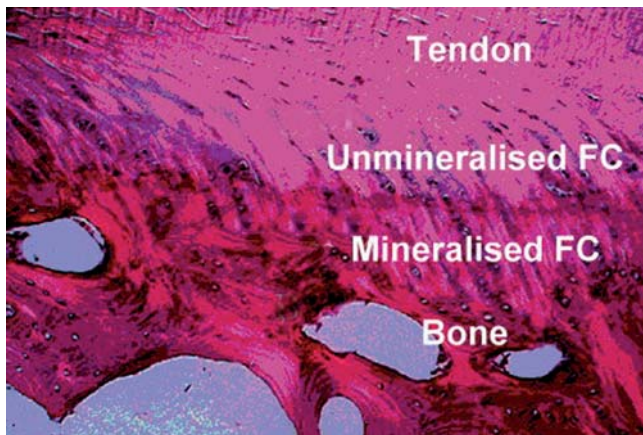


Fig. 1: Histopathology slice of an enthesis showing the 4 layers of the fibrocartilage (FC) enthesis. Modified from reference 6.

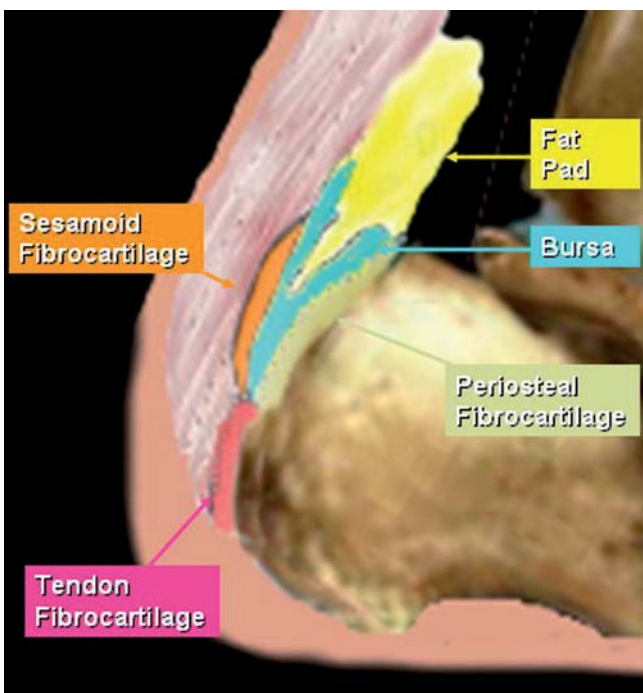


Fig. 3: The major components of the enthesal organ of a schematic Achilles tendon insertion.

Introduction

The enthesis is the insertion of the tendon onto the bone. Over 10 years ago, it was noted that the term tendonitis is a misnomer and attempts are still ongoing to have it replaced with terms like tendinopathy¹. This should also extend to the terms epicondylitis and fasciitis, where epicondyle “tendinopathy”² and “fasciosis” or “fasciopathy”, respectively may be more appropriate³. In the last 10 years the rheumatological literature has prompted a review of our concepts

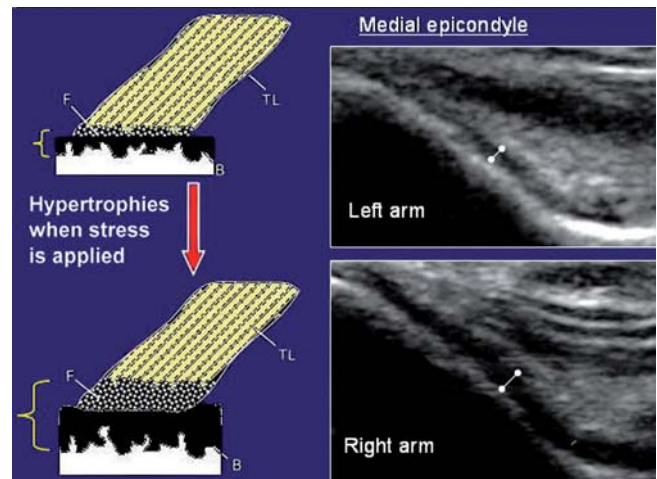


Fig. 2: There is physiological thickening of the fibrocartilage with stress. The ultrasound images are from a right handed manual labourer showing the hypertrophied enthesal fibrocartilage on the right. Modified in part from reference 4.

regarding tendon insertion disease. The problem in fact is one of an enthesopathy rather than isolated tendinopathy⁴⁻⁷.

Anatomically there are four distinct zones, namely the:

- 1 Tendon substance
- 2 Enthesal fibrocartilage
- 3 Mineralised fibrocartilage
- 4 Bone substance.

On ultrasound imaging, the normal enthesal fibrocartilage is a dark line adjacent to the bone.

Functionally there are other components adjacent to the enthesis proper which also share the stress forces and are termed the “enthesis organ”^{9,10}. These include the periosteal fibrocartilage, the sesamoid fibrocartilage, the fat pad and bursa¹¹ (Fig. 3). The synovial enthesal complex is a concept that the adjacent bursa or joint lining share stress forces, especially compressive forces and are an integral part of the enthesis organ^{12,13}.

As the stress forces of the enthesis are shared across all these structures, failure and resultant pathology is also shared across all these structures¹⁴. It applies to the distal Achilles tendon, the supraspinatus tendon, the medial and lateral epicondylar tendons, the plantar fascia, the gluteal tendons, the patellar tendon and the adductors and hamstrings among others.

The normal enthesis on ultrasound shows three of the four layers. There are healthy tendon fibres, a thin dark line representing the fibrocartilage enthesis and a sharply defined intact bone interface representing the mineralised fibrocartilage. The spectrum of findings that may be present in an abnormal enthesis on ultrasound include: enthesophyte,

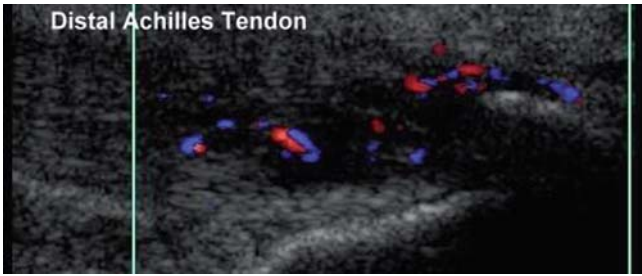


Fig. 4: A colour Doppler image showing the presence of blood flow at the distal achilles tendon.

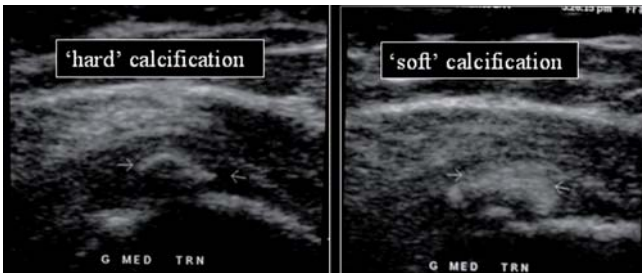


Fig. 5: Hard and soft calcification in gluteus medius tendons.

bone erosions, disrupted fibrocartilage, tendinopathy, tendon clefts, neovascularity, bursitis, synovitis and calcific tendinopathy. The latter is distinct to the more usual form seen in the mid tendon substance. This occurs immediately adjacent to the enthesis or at the “periosteal fibrocartilage” portion of the tendon.

Also, there are others which are site specific. Of these, some reflect enthesis failure and some are symptomatic. The symptomatic components are particularly important to identify as tailored treatments are aimed at these. The major symptomatic components appear to be the:

- 1 Tendinopathy
- 2 Calcific tendinopathy
- 3 Synovitis
- 4 Bone erosions.

Most treatments are based on symptoms only. The best treatment is to address which changes are present and treat those, i.e. single entity pathology can be treated with monotherapy, but multiple pathology requires polytherapy. Single treatments like blood injection (which address the tendinopathy) would of course be successful in those patients where the major pathology is isolated to the tendinopathy component.

Treatments

1 Tendinopathy

This is due to failed healing or “dysrepair”, a phrase coined by Cook and Purdham¹⁵. The pathology and treatment approach to tendinopathy has recently been well discussed¹⁶⁻¹⁸. The cornerstone of therapy is to remove any biomechanical predispositions and treat with physiotherapy. Injection techniques may be useful if this fails. With the tendinopathy, I start by assessing neovascularity in the tendon and may use polidocanol injection for this¹⁹ (Fig. 4). Like in tendons elsewhere, pain and tenderness at the enthesis is associated with increased vascularity on colour imaging²⁰; the pain is probably due to accompanying nerve ingrowth²¹. No vascularisation is demonstrated on ultrasound in the normal heel enthesis, despite using contrast injection²². I

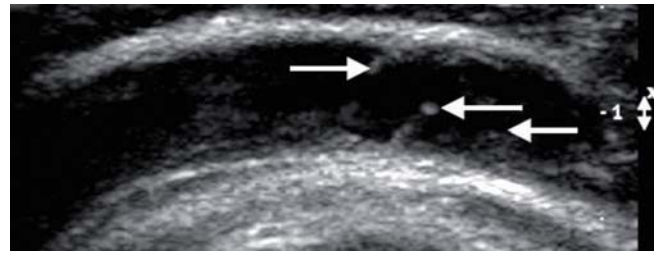


Fig. 6: Shows a subdeltoid bursa post hydrodilatation with disrupted fibrin strands now apparent and marked by arrows.

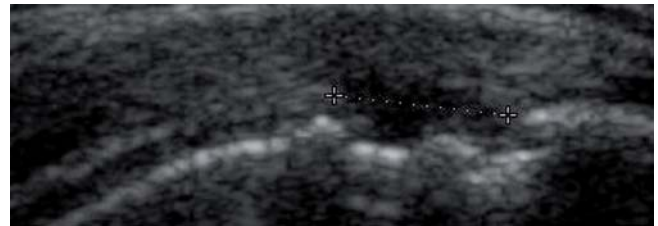


Fig. 7: There are bone erosions on the humeral head at the site of the supraspinatus tendon insertion.

have also used blood, glucose and dry needling²³⁻²⁶. Steroid around the tendon is very effective in providing relief of pain for some time²⁷, which allows patients with limiting pain to better complete physiotherapy. There may be benefit from other agents like platelet rich plasma²⁸. I find tendon clefts or tears are commonly noted with injection of substances into the tendon. They are probably not painful in themselves but are simply a reflection of the progression of the tendinopathy component.

2 Calcific tendinopathy

This can be “hard” or “soft”. It may be an incidental finding or exquisitely painful and is addressed with dry needling and attempted aspiration/irrigation and celestone^{29,30} (Fig. 5).

3 Synovitis

This can involve the adjacent bursa, or a joint. It can be an acute inflammatory synovitis or a chronic process with thickening of the bursa and internal fibrin bands that cause internal impingement. Acute inflammation responds to celestone injection while I do a hydrodilatation for the chronic bursa with bands. I use ice cold normal saline with celestone and local anaesthetic (Fig. 6).

4 The bone component

The bone component to the pain is difficult to address. Erosions are common³¹. Bone marrow changes are seen on pathology³² and bone oedema is sometimes seen on MRI². The erosions may have new vessels in them and are tender if you needle them. I suspect this is a cause of treatment failure and the constant nocturnal pain that some patients experience. There is a recent study suggesting we may be able to treat this component by drilling into the bone but I have not gone to this step yet³³ (Fig. 7).

As mentioned there are some site specific findings and these are discussed briefly.

Supraspinatus tendon enthesopathy

Compression under the acromion and friction of the supraspinatus tendon on the greater tuberosity during abduction are probably the major contributing factors. The tendon,

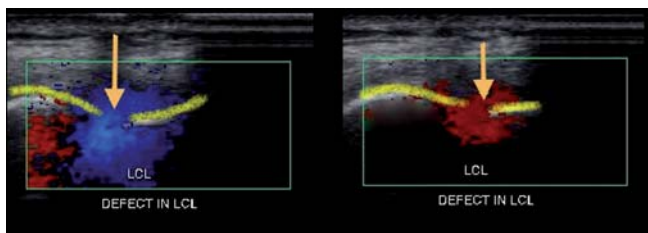


Fig. 8: Color flow images of a disrupted lateral collateral ligament at the elbow. With pronation and supination there is to and fro movement of fluid through the defect in the ligament (ligament shown schematically in yellow).

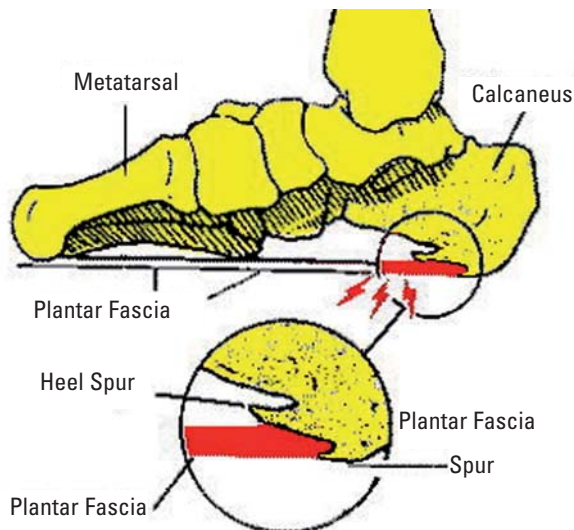


Fig. 9: Shows the origin of the common inferior calcaneal spurs in relation to the plantar fascia.

subdeltoid bursa and the synovial recess under the tendon are all involved. There is often an abnormal tendon with an abnormal bursa. These are not competing diagnoses, but reflect the multipathology of the enthesis organ. It is common to find a thickened or multilayered bursa which bunches with abduction. At least one study³⁴ has noted the association between bone erosions and tendon tears but failed to realise that the erosions were due to the enthesopathy which in turn results in the tendon softening and subsequent tendon tears. Most treatment is based around relieving the patient's pain. There is a preoccupation with finding small tears to account for the patient's pain, but I suspect most small tears probably do not cause pain. It is known that looking at the contralateral pain-free shoulder often reveals a larger tear that the patient was not aware of.

The real problem is that patients have painful enthesopathy and the small tears are merely the ultrasonographic evidence of that enthesopathy.

When tears are treated surgically, the surgeon does not just sew up the tear. They also created a bone groove and reattach the tendon, effectively doing an enthesis repair and or reconstruction. So perhaps for years they have been applying the correct treatment to the wrong diagnosis. Complete tendon tears cause loss of function and usually require surgical treatment for this. If the problem was simply coming from the tendon, a complete tear should remove the pain. If it is still painful, it suggests an underlying painful enthesopathy.

Lateral epicondylar enthesopathy

The attachment of the common extensor organ includes its adherence to the lateral collateral ligament and this is part of

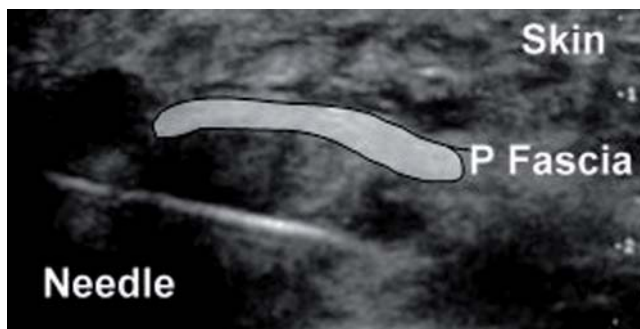


Fig. 10: A transverse image showing needle placement deep the plantar fascia from a medial approach. The plantar fascia is shown in cross section and shaded grey.

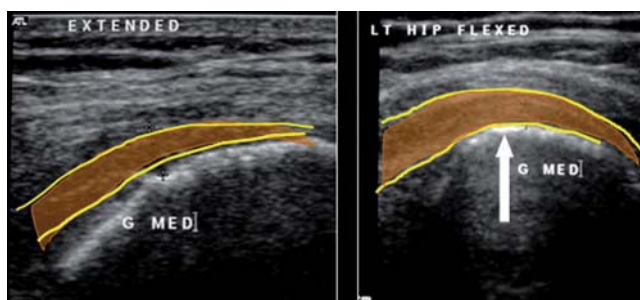


Fig. 11: The distal gluteus medius tendon in extension and flexion showing the altered angle of insertion and compression that occurs over the greater trochanter.

the enthesis organ of the lateral epicondyle³⁵. Inflammation and secondary tears in the lateral collateral ligament occur. I suspect the tear in the lateral collateral ligament allows the synovial fringe to prolapse into the joint and contributes to a “fringopathy” in some patients. Like most tendons, proper assessment is a dynamic test and the tear is best seen with stressing the ligament by flexion/extension and pronation/supination. A tear can also be proven with injecting gas into the joint and watching it appear superficial to the ligament, or when there is a small amount of fluid in the joint, pronating and supinating and a colour flash will show the fluid moving through the defect (Fig. 8). I find a small amount of fluid arising from the radiohumeral joint common when there is a tear.

Plantar calcaneal enthesopathy

Enthesophytes (spurs) are common but as in other sites rarely cause pain. Spurs always occur at the distal tendon attachment. The plantar fascial spur occurs at the posterior inferior calcaneus and the common spur at the medial tuberosity is from the flexor digitorum attachment (Fig. 9). This shows that stress forces at the calcaneus occur deeper to the fascia itself. In up to 70% of cases there is also deeper pathology related to the flexor digitorum attachment and long plantar ligament attachment³⁶. I have seen patients with a normal plantar fascia and significant painful enthesopathy deeper to it. Perifasciitis may occur and irritate Baxter's Nerve (medial calcaneal branch) under the proximal fascia³⁷. If celestone is being injected it commonly needs to be applied deep to the fascia as well as superficial to it. I use a medial approach as it is less painful and allows access to the superficial and deep structures more readily (Fig. 10).

Trochanteric and gluteal tendon enthesopathy

Like the other tendons this is a dynamic assessment,

pathology is most often seen in the gluteus medius tendon +/- the submaximus bursa. Like at other sites, these are not competing diagnoses. Extra stress changes due to compression occur at the gluteus medius tendon. With flexion of the hip compression occurs at the distal gluteus medius tendon and overlying submaximus bursa (Fig. 11). This is exacerbated in overweight patients and females where the angle of the pelvis is greater. The cornerstone of treatment is with physiotherapy but celestone injection can effectively relieve pain in the short term and allow patients to better do their physiotherapy. The bursa is commonly thickened and multilayered and responds better to hydrodilataion – as I have described above – than simple celestone injection.

There are also inflammatory enthesopathies which may occur at multiple sites. They can be demonstrated with ultrasound in spondyloarthropathy and psoriatic arthritis³⁸⁻⁴¹. The same spectrum of features is present in large entheses like the adductors of the legs or very small entheses like the fingers. Many are abnormal on ultrasound but typically asymptomatic³⁸.

Watcha see and watcha do

Reporting any enthesopathy should take a systematic approach. I describe the whole spectrum of positive findings, and then comment particularly on those features which are clinically relevant and likely to be painful. If relevant I will comment on interval change. I am fortunate in that I am usually asked to “ultrasound and inject as appropriate”. Simply, what I do depends on what I find. By tailoring the treatment of each patient to what particular pathology is present we can better define the actual underlying pathology and better achieve relief of symptoms.

Conclusion

Thanks to work being in the rheumatological literature we have been able to advance our understanding of tendon insertion disease greatly. The enthesis is an increasingly recognised important structure in the cause of pain and should form the basis of the musculoskeletal examination. Sonologists and sonographers should be acquainted with the normal anatomy and pathology of the enthesis to be able to apply the concepts described in this article.

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