Enthesopathy: Clinical recognition and significance

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ABSTRACT

Enthesopathy is a common clinical finding denoting pathology at the ‘entheses’, i.e. attachment sites of muscles, tendons, joint capsules, ligaments and fascia to the bone. Inflammatory enthesopathy or enthesitis is a sine qua non of seronegative spondyloarthropathies (SSA). It can also be occupational, metabolic, drug induced, infective or degenerative. Bursitis closely mimics enthesis. Ultrasound with high frequency transducers is a simple, cost-effective and feasible test to detect enthesopathy which is amenable to treatment with local steroid injections, physiotherapy and non-steroidal anti-inflammatory drugs, in addition to treatment of the primary disease. Unrecognized and untreated, it can lead to considerable morbidity.

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WHAT IS ENTHESOPATHY?

The word ‘entheses’ refers to the site of attachment of muscles, tendons, joint capsules, ligaments and fascia to the bone, e.g. Achilles tendon, plantar fascia, adductor tubercle, greater trochanter, intercostal, sternocostal, costo-vertebral and several such sites. Only the attachment sites of these soft tissue structures closest to the bone (and not the tendon, ligament, muscle, fascia or bone to which these structures are attached) are known as entheses.

Pathology of the entheses or enthesopathy can have a varied aetiology. Among them, inflammatory enthesopathy or enthesitis is widely mentioned in the medical literature. For example, an Achilles tendinitis close to the calcaneum bone at the insertion site is enthesitis, but any inflammation proximal to it would be called tendinitis. Hence the synonym insertional tendinitis for enthesitis. On the other hand, a tennis elbow without the involvement of adjacent soft tissue is just an epicondylitis which is a pathology of the bone and periosteum and hence strictly not an enthesitis.

THE IMPORTANCE OF ENTHESOPATHY

It is the sine qua non of seronegative spondyloarthropathies (SSA), the commonest inflammatory arthropathies. Approximately 2% of the population has SSAs. These include reactive arthritis, undifferentiated SSA, enteropathic or inflammatory bowel disease associated with SSA and ankylosing spondylitis. Unfamiliarity with the entheses sites and enthesitis may lead a clinician to disregard the patient’s symptoms, and consequently to an erroneous diagnosis and treatment. Although ultrasound imaging is helpful in making the correct diagnosis, clinico-radiological correlation is needed.

TYPES OF ENTHESOPATHY

They are usually inflammatory (enthesitis), but can be traumatic or occupational, such as work-related musculoskeletal disorders. In the long run these can cause formation of spurs at entheses sites with associated morbidity. Metabolic diseases and drugs such as isotretinoin can also cause enthesopathy. Pachydermoperiostitis is now thought to be due to generalized enthesopathy by some. Gout can also occasionally cause enthesopathy.

Enthesitis is one of the diagnostic criteria of the European Spondyloarthropathy Study Group (Table 1).

In children, there is a subset of juvenile arthritis known as the SEA (seronegative enthesopathy with arthropathy) syndrome in which enthesitis is the major manifestation. It can also be the sole manifestation of a subset of psoriatic arthropathy and has been described in infectious diseases such as leprosy.

HOW DOES ENTHESOPATHY MANIFEST?

A patient with enthesopathy may be absolutely asymptomatic while pain at the mentioned entheses site may be the only feature in most symptomatic patients. Only very rarely, local swelling and tenderness may occur.

DOCUMENTING ENTHESITIS

‘Seeing is believing’ and in the absence of any clinical sign, imaging is the only method of detecting enthesitis. Plain X-rays can show only advanced irreversible changes such as a spur. Although some consider MRI or technetium bone scan as gold standards for enthesopathy, ultrasonography with high frequency transducers (≥10 MHz) is fast becoming the method of choice for detecting enthesopathy. Enthesopathy changes seen in ultrasonography include thickening of the tendon, peritendinous fluid collection, nodule formation, calcification and bony irregularity at the site of insertion. Moreover, ultrasound can delineate the degree of enthesopathy, its inflammatory component and can also differentiate it from bursitis and normal tendinitis (Figs 1-3). In

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<th>Table 1. European Spondyloarthropathy Study Group (ESSG) criteria</th>
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<td>A. Inflammatory backache</td>
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<td>OR</td>
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<td>Asymmetric oligoarthritis, predominantly of the lower limbs</td>
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<td>AND one or more of the following:</td>
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<td>B. Enthesitis</td>
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<td>Alternate buttock pain</td>
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<td>Diarrhoea, dysentery, urethritis or cervicitis</td>
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<td>Inflammatory bowel disease</td>
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<td>Psoriasis</td>
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<td>Family history</td>
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<td>Sacroiliitis</td>
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DANDA et al.: ENTHESOPATHY

FIG 1. Ultrasound sagittal view of right tendoachilles entheses showing normal fibrillary architecture of the tendon (white arrow head) and smooth surface of the calcaneum (white arrow).

FIG 2. Ultrasound sagittal view of tendoachilles entheses showing thickened tendon (black arrow head), irregularity of the calcaneal surface (white arrow) with periarticular fluid collection (white arrow head).

Ankylosing spondylitis is the result of the syndesmophytes that ultimately lead to a bamboo spine.

TREATMENT
Presently non-steroidal anti-inflammatory drugs, local steroid injections and physical therapy are the mainstay of treatment. However, targeted anticytokine therapy may become a reality in the future, preventing or at least reducing morbidity. Although the overall treatment of spondyloarthropathies with drugs such as sulphasalazine and/or low-dose methotrexate does help, this symptom is one of the last to remit in patients with spondyloarthropathies.

CONCLUSION
Failure to recognize a common and treatable clinical condition such as enthesopathy can lead to persistent morbidity and incorrect diagnosis. This may range from overdiagnosis of fibromyalgia and depression to under-recognition of spondyloarthropathies. Enthesopathy can be detected by a high degree of clinical suspicion combined with ultrasonography which is simple, inexpensive and feasible at primary care centres.

REFERENCES

a pilot study of 94 patients with spondyloarthropathy and a clinical suggestion of tendoachilles enthesopathy, we found 44 patients (47%) had only bursitis; 36 (40%) had only enthesitis and 7 (8%) had both enthesitis and bursitis.18

DIFFERENTIAL DIAGNOSIS
Bursitis can both mimic or accompany enthesopathy in several arthropathies including spondyloarthopathies.5,7 In addition, symptoms of fibromyalgia, myofascial pain syndrome, neuralgia, arthralgia, osteoporosis and osteomalacia can also mimic enthesopathy. Other important considerations include various non-inflammatory enthesopathies such as fluorosis16 and other 'unspecified enthesopathies' such as overuse syndrome.9

WHY DOES ENTHESITIS OCCUR?
The pathogenesis of enthesitis is thought to be triggered by cytokines such as transforming growth factor β, tumour necrosis factor α, bone morphogenetic protein 6,1 vascular endothelial growth factor, etc. which are responsible for the proliferation of vascularity, inflammation and new bone formation.

Second Wellcome Trust Workshop on HIV/AIDS and TB
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The Workshop is supported by a Grant from the Wellcome Trust and sponsored by the South African Medical Research Council. All travel, accommodation and subsistence costs will be covered. Applicants must be currently and actively involved in laboratory-based research into HIV/AIDS or TB. Preference will be given to applications from Africa and from those involved in projects in Africa. The Workshop is aimed at pre-doctoral students and recent post-doctoral fellows and is limited to 30 participants.

Please send a copy of your CV including publications, a motivation for why you wish to participate in the Workshop, a brief account of your research as well as 2 sealed referees reports to:
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Closing date for application is 31 May 2001