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EDITORIAL

Clinical implications of reconsideration of enthesitis/enthesopathy/enthesial erosion, as tendon attachment-localized avulsions and stress fracture equivalents

Bruce M Rothschild

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Abstract

Recognizing the mechanical origin of enthesitis/enthesopathy and the avulsion-nature of what had previously been considered erosions, it seems inappropriate to attribute it to stresses related to a person's normal activities. Conversely, sudden or unconditioned repetitive stresses appears the more likely culprit. Studies of enthesial reaction have lacked standardization as to findings present among individuals who appear to be healthy. Clinical evaluation by palpation and manipulation may be as effective as application of radiologic techniques. Recognition of the mechanical nature of the disease, including individuals with inflammatory arthritis suggests prescription of mechanical solutions that reduce stresses across the involved enthesis.

Key Words: Enthesitis; Epicondylitis; Erosion; Avulsion; Clinical examination

Core Tip: Enthesitis, unrelated to inflammatory diseases, is not the result of, and its presence is not a measure of, routine activities. It results from the application of sudden or unconditioned repetitive stresses. Bone defects at tendon/ligament insertion sites are not the result of biological erosion, but rather of avulsions. To avoid overinterpretation of osseous reaction at entheses, their presence needs to be compared to findings among individuals who appear to be healthy. Recognition of clinically significant enthesitis may be as effectively identified by physical examination, with radiologic techniques possibly redundant. An important component of treatment is reduction of the mechanical stresses to which a given enthesis is exposed.

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INTRODUCTION

The term enthesopathy/enthesitis describes calcification or cortical disruption occurring at tendon, ligament and capsule insertion sites[1-4], traditionally attributed to mechanical stress-related tissue remodeling[5,6] or to aging, inflammation, metabolic or medication effects[7-9]. The normal stresses hypothesis is challenged by Zumwalt's observation that both the shape and size of enthesial attachments were indistinguishable between sheep subject to three months of treadmill exercising[9]. Studies by Rabey *et al*[10] further confirmed that observation. It was subsequently recognized that muscle activity may alter bone shape, but not that of the entheses. Alternatively, Balint *et al*[11] and Shaibani *et al*[12] documented that entheseal reaction actually represented injury/trauma-related structural damage. The latter apparently does not occur in the absence of attachment site overload[9], similar to what is observed in stress fractures. Indeed, so-called enthesial site erosions were revealed actually to represent tendon avulsions and thus are also mechanically-induced alterations[13,14]. Indeed, close examination of the images in Donners *et al*[15] reveals feather-like disruptive processes, characteristic of partial tendon avulsions. Actual erosions, occurring in patients with erosive arthritis of the spondyloarthropathy variety, are below radiologic resolution[15], precluding confusion with mechanically-derived avulsions.

CLINICAL IMPLICATIONS OF ENTHESOPATHY

Skeletal manifestations of enthesopathy, whether radiologically or macroscopically viewed, document past events, not necessarily on-going activities. This perception is partially based on De Lorenzis *et al*'s notation that pain and structural findings are dissociated in what they referred to as "enthesial disease"[16] and the observation by Bakirci *et al*[17] that indistinguishable enthesial reactions are also seen in asymptomatic individuals. As prevalence corelates with age[13], it is as likely that they simply reflect a lifetime of insults. This is not surprising, as normal entheses actually have few of the small non-myelinated C and large myelinated Aδ nociceptive nerve fibers recognizing painful stimuli[18]. This is in contrast with surrounding para- and endotenon and fat pads and the bone into which they actually insert[19], which may be the true source of "enthesopathy-derived" pain. These enthesial changes often become involved as periarticular fibrotic proliferation secondary to chronic joint disease[20]. Conceptually, such involvement could be primary, secondary or a combination of the two.

Thus, findings noted on physical examination of patients may be as pertinent as those identified radiologically. This seems analogous to the discrepancy between clinical and X-ray findings in dactylitis[14,21]. Indeed, even magnetic resonance imaging (MRI) identifies less than half of enthesopathy sites revealed on clinical examination[22-25] and even those don't seem to overlap[26]: Some radiologic findings are observed in asymptomatic individuals and symptomatic findings are sometimes found among individuals lacking radiologic evidence[1,22]. Obviously, X-ray, if not MRI findings relate to past "activity" and may have reflected a short duration event or even diminishing symptoms over time[27]. That being said, ultrasound[28], MRI[29,30] and positron emission tomography/computed tomography[31], but not necessarily X-ray studies[21,32] do have value, complementing clinical examination[13,15,21,28], permitting distinguishing among various varieties of inflammatory arthritis (*e.g.*, rheumatoid arthritis and spondyloarthropathy).

CLINICAL RECOGNITION OF ENTHESOPATHY

Several clinical examination techniques have been suggested for recognition/confirmation of enthesial sources of pain [33]. These can be divided into palpation and manipulation (Table 1). Palpation may seem the most obvious, but standards for its recognition have been controversial. Applying sufficient pressure on the area of interest to produce nailbed pallor in the palpating digits may elicit discomfort and it must be assured that it actually reproduces the patient's complaint[34]. Assessment of tendon thickening has also been suggested[33], but that is not straight-forward. Comparison with the contralateral side may help to confirm enthesopathy as the source of the patient's complaint, in the absence of bilateral or inflammation (*e.g.*, spondyloarthropathy) derived disease[35]. Comparison of insertion thickness with more proximal tendon thickness has also been suggested. However, biologic variation is significant, requiring consideration of "cutoff" values. Ultrasound attempts to identify appropriate cutoffs have been compromised by technical inadequacies and small sample sizes[36,37]. Even with such approaches, the only enthesis in which healthy individuals and those with spondyloarthropathy differed was at the triceps insertion[33]. Even swelling may not be informative, as swelling 5-10 cm proximal to the Achilles tendon insertion may be a normal finding[38] and Achilles and plantar calcaneal spurs are found in 25% of apparently normal individuals[39-41].

Table 1 Techniques for clinical recognition of enthesopathy, derived from Canoso *et al*[34]

Tendon	Maneuver
Supraspinatus at shoulder	Resisted arm abduction through internal and external rotation
Bicips brachii at proximal forearm	Resisted forearm supination
Extensor carpi radialis at wrist	Resisted wrist dorsiflexion
Pronator teres at wrist	Resisted wrist palmar flexion
Abdominal muscles at iliac crest	Contralateral bending
Gluteus medius at hip	Resisted thigh adduction from supine abducted, externally rotated hip (knee 30 degrees flexed) position
Quadriceps tendon at knee	Quadriceps contraction of flexed knee

WHAT ARE THE IMPLICATIONS FOR TREATMENT?

Analgesic medications, both oral and topical, generally do not provide adequate relief[42-46]. The challenge presented by the enthesopathy/epicondylitis referred to as tennis elbow is illustrated by the cacophony of modalities that have been suggested for therapeusis [*e.g.*, injections of corticosteroids anesthetics (*e.g.*, bupivacaine) botulinum toxin, autologous blood, platelet rich plasma or even stem cells[43,47-58], radiofrequency and shock wave treatments[59-61] and surgery (*e.g.*, tenotomy)[42,62-68], none with documented long-term clinical benefit[49]]. These are in addition to somewhat more effective modalities such as manipulation, therapeutic ultrasound, phono- and iontophoresis, dry needling acupuncture, and low level laser application[46,47,49,50,52,53,55,56,64,65,69-74], but none resulted in complete resolution of symptoms [69].

If most enthesopathy and all of the "osteolytic"/errosion variety are post-traumatic events and if inflammatory arthritis-related enthesopathy could be considered the musculoskeletal equivalent of the dermal Koebner phenomenon, perhaps a mechanical solution would be worth pursuing? Reducing the stresses across clinically-involved entheses may well reduce symptoms and allow healing. This approach has actually been applied[75] to the enthesial disorder referred to as tennis elbow or lateral epicondylitis[76,77]. Afterall, microscopic and ultrasound evaluation revealed mechanically-, not inflammatory-derived damage[49,78-81]. Circumferential use of commercially available bands (*e.g.*, two inches wide proved ineffective, as they only reduced effective muscle strength, without reducing attachment stresses and was less effective than immobilization of the elbow)[44]. The latter interferes with function (activities of daily living) and has the inherent risk of reflex dystrophy development. My personal approach has been to apply circumferential apply ¾-1" wide velcro bands just distal to the epicondyle with sufficient tension to reduce muscle traction on the elbow, but not tight enough to impair circulation[75]. This approach was associated with failure to complete resolve symptoms in less than 1% of afflicted individuals. Failure was limited to individuals who would or could not modify the activity[77,80,82,83] that was repeatedly stressing the epicondylar entheses. Similarly, extension exercises often relieve pain symptoms for individuals afflicted by the enthesiallay-related back pain of ankylosing spondylitis.

CONCLUSION

Thus, the solution for painful entheses may well be relief/reduction/elimination of stresses transmitted across it, whether by altering activities and/or the technique involved in those activities or use of external structures (*e.g.*, bands to modify stress distribution). Absence of sufficient effectiveness might suggest that the entheses involved may actually be inflammatory in nature (*e.g.*, those related to spondyloarthropathy) and require intervention aimed at reducing the metabolic components of the inflammatory reaction.

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