Winter 2016

ORTHOPAEDIC EVIDENCE

NEWSLETTER

In This Issue

Tendinopathy: Is Imaging Telling Us the Entire Story?

November 2015 | volume 45 | number 11 | Journal of Orthopaedic & Sports Physical Therapy

Tendinopathy is the clinical condition that describes pain and dysfunction of the tendon, which is independent of pathology within the tendon.

Degenerative changes in structure observed histologically or on imaging, independent of clinical symptoms, are termed **tendinosis**. The primary change being increases in cell numbers that exhibit an altered, more metabolically active phenotype.

Ultrasound and MRI are the preferred imaging modalities to detect tendinosis.

Ultrasound is somewhat user dependent, as slight changes in the ultrasound transducer tilt generate imaging artifacts that are similar to those seen with tendon pathology. It describes tendinosis changes as increases in tendon dimensions and heterogeneous or diffuse changes in echogenicity. Multiple reflections and shadowing are generated by fibrillar disorganization and lack of parallel-aligned fibres, which are represented by an area of hypoechogenicity.

MRI has excellent soft tissue contrast detail and multiplanar imaging capabilities with excellent reproducibility but is costly and of limited availability. The alteration in fibrillar alignment and increased water content result in an increase in intratendinous signal.

But there has always been poor correlation with the presence of pain and pain severity. Because tendon pain is not solely driven by local tissue changes, there is likely to be an interaction between the local tissue and the peripheral and central nervous system.

Therefore, if structural disorganization seen on imaging is not necessarily responsible for symptoms, imaging should be considered as part of the *risk factor profile for tendinopathy,* similar to that of load, anthropometric factors, and genetics, rather than solely as a diagnostic feature. As such, the local tissue structure seen via imaging may be considered a risk factor for development of symptoms.

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Other study findings also suggest that improvements in the tendon are not necessary to facilitate clinical improvement after an eccentric exercise program.
While improvement in, or normalization of, tendon structure is a positive result, it is not necessary for improvement in pain and function, suggesting that the pathological tendon might have adapted to become, and remain, load tolerant.

It appears that the pathological tendon maintains sufficient amounts of aligned fibrillar structure by increasing tendon dimensions in parallel with the mean cross-sectional area of disorganization (ie the more disorganization, the bigger the tendon).
The increase in tendon dimensions may be a mechanism by which the pathological tendon maintains sufficient mean cross-sectional area of aligned fibrillar structure to still tolerate load.
Interventions such as eccentric exercise may not be efficacious in remodelling the area of pathology; rather, these loading protocols may cause adaptation and increase the loading capacity of the surrounding aligned fibrillar structure.
Stability in tendon structure accompanied by improvements in pain and function can then be considered a positive outcome.

Imaging allows for the visualization of structure; it does not represent the entire clinical picture and should not be used as the sole diagnostic criterion in determining whether the clinical presentation is generated by the tendon.

It can be useful in differential diagnosis.
It needs to be placed in the context of the overall clinical picture.

2 new ultrasound techniques may be of further value:

Ultrasound Tissue Characterization captures contiguous transverse ultrasound images over the length of the tendon and semi-quantifies the stability of the echo texture over the length of the tendon. The ability to capture a 3-dimensional ultrasound image of the tendon, which standardizes parameters that affect the repeatability of conventional ultrasound (ie transducer tilt angle, depth, and gain settings), and semi-quantification of tendon structure attempt to address the limitations of conventional ultrasound imaging.

Sonoelastography evaluates the mechanical properties of tissues. It is based on the principle that tissue displacement in response to external compression produces strain within the tissue, with *strain* defined as the change in length. Adding ultrasound elastography to conventional ultrasound may improve the association with clinical symptoms.

There is overwhelming evidence that structural disorganization predates the development of symptoms and tendon rupture. These new imaging modalities may help to define loading parameters that result in structural disorganization and to identify the point at which tendon load exceeds the tendon capacity.

The development of new imaging techniques that utilize more quantifiable parameters, such as UTC or sonoelastography, will hopefully enhance our ability to diagnose, predict the development of symptoms, and monitor the efficacy of treatments.

Comment:

This is an excellent clinical commentary that contains substantially more detail than presented here. It is an excellent resource for any clinician involved in the diagnosis, ordering of imaging and treatment of tendinopathy. It can be found in the JOSPT or contact one of our clinics to obtain a copy of this useful paper.