Hand Enthesophytes and Knee Enthesopathy: Is Osteoarthritis Related to a Systemic Enthesopathy?

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To the Editor:

We read with interest the recent article by Gibson and colleagues 1 and the accompanying editorial 2 on the role of enthesopathy in osteoarthritis (OA). Gibson, et al suggest that OA is not a systemic enthesopathic disease, based on the finding that there is no increase in radiographically determined hand enthesophytes in subjects with bone marrow lesions of the knee. We contend that there is insufficient evidence from the study to make such a claim.

It is considered that isolated knee OA and knee bone marrow lesions are relatively more sensitive to mechanical and trauma-related stressors like obesity and injuries 3,4, while hand OA has a stronger genetic basis 5, a case of nurture (or lack of, in the case of the knee) versus nature (genetics).

Further, Gibson, et al looked only at enthesophytes at the mid-shaft of phalanges, therefore missing enthesophytes commonly seen at the distal insertions of collateral ligament in the hand. In addition, enthesopathy includes not only enthesophytes, but other changes due to enthesitis, like bone edema and soft-tissue changes such as structural abnormalities of the tendons, ligaments, and joint capsules. Therefore using only plain radiography of the hands used in the study is a less sensitive imaging technique, and would have been less sensitive at identifying small enthesophytes visible on MRI or even ultrasonography. As mentioned in the accompanying editorial 2, the uniplanar characteristic of the plain radiograph is unable to visualize possible enthesophytes at the tendon insertions, resulting in potentially lower counts of enthesophytes, in addition to completely overlooking early-stage enthesal abnormalities that are pretty ubiquitous in early hand disease 6. MRI of the hands is a more sensitive imaging method to study hand OA 5, and permits identification of enthesopathic processes, giving a much more accurate picture of the actual changes taking place.

Further, Gibson, et al attempted to help define generalized OA in relation to enthesopathy 1. Haugen 2 noted that generalized OA is a puzzle in that there was no consensus for the definition, and the study by Gibson, et al raised the question of whether there is a more systemic process or a more local biomechanical factor to explain the distribution of the enthesopathic changes. We would point out that the role of enthesopathy in OA remains difficult, but not a puzzle, with emerging literature that recognizes the heterogeneous expression of OA. For example, our work 7 considers systemic enthesopathy-related OA as a specific subcategory of disease, which was omitted by Gibson, et al and the editorial. There is evidence that some OA is enthesopathic-related but much of it is clearly arising elsewhere — especially for knee disease. When OA is classified according to anatomic changes, we can see that early knee OA includes not only enthesopathic-related disease but also other disease categories, including osteogenic, meniscogenic, and chondrogenic variants 2. This allowed a logical definition of OA based on structural changes in light of more recent knowledge, hence so-called idiopathic OA is no longer a phenomenon.

We therefore suggest that the study by Gibson, et al does not allow for conclusions about systemic enthesopathy in OA. In any event the evidence for such a claim may no longer be sustainable as enthesopathic-related OA represents a specific category of disease.

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