Background: OA is now widely recognized as a complex, multifactorial disease of the whole joint rather than solely a loss of articular cartilage. There is much evidence that at least in some forms of OA, morphological changes at the subchondral plate may precede cartilage damage. The aim of this study was to investigate the microanatomy of the bone-cartilage interface in ageing and disease in order to identify the earliest pathological changes in joint destruction.

Methods: We investigated microstructure in a series of weight bearing joints obtained at arthroplasty from patients with joint diseases, or from bequeathed cadavers. These were analysed by micro-dissection and a spectrum of contemporary imaging techniques including micro computed tomography (Micro CT), magnetic resonance imaging (MRI) of unembedded tissue and backscattered scanning electron microscopy (SEM) of PMMA embedded blocks with the recently introduced method of iodine block surface staining to reveal soft tissue histomorphology.

Results: In all the samples examined, there was evidence of microanatomical cracks in the subchondral plate, some of which were full thickness through the bone and calcified cartilage. Microscopy revealed that these were not formed by osteoclastic action. Using SEM we detected evidence that a mineralizable matrix was extruded through these microcracks into the hyaline articular cartilage (HAC). This matrix subsequently became calcified, with much higher mineral content than bone or calcified cartilage, and formed protrusions detectable by high resolution micro CT. These protrusions arose from the subchondral plate and extended into the HAC but generally not as far as the articular surface. On MRI, the hypermineralized protrusions were present as lesions of low signal yield, silhouetted against the signal-rich HAC. Optimum identification was made using DESS sequences. Within this cohort, the highest number of protrusions, 21, was found in the sample from the oldest donor (86 years) and the least, 8, was found in the youngest (30 years).

Conclusion: Our investigations reveal that hypermineralized protrusions from the subchondral plate are present in human weight bearing joints and that their incidence appears to increase with age. These protrusions have the potential to disintegrate and give rise to abrasive cutting and grinding particles within HAC and thus to promote its mechanical destruction. We discovered them because we used techniques not usually applied to investigate human joint pathology. In particular, we did not use decalcification, which effectively destroys the evidence.

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