Marrow lesions, pain and osteoarthritis

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Osteoarthritis (OA) is a condition affecting the whole joint and is the most prevalent arthritis worldwide. A number of pathological changes are recognised in OA, including deterioration of cartilage integrity, subchondral bone changes and synovitis. Although advances have been made in our understanding of OA pathophysiology, there are no current treatments that halt the progression of the disease. Treatments are largely based upon physical therapies to improve function, anti-inflammatory agents for pain and joint replacement surgery for late stage disease in larger weight bearing joints. There is an urgent need to better understand OA pathophysiology, which could help in the development of new treatments. Historically, evidence of OA structural damage was established using plain radiography of affected joints. In recent years, more advanced imaging techniques including magnetic resonance imaging (MRI) have led to an improved understanding of changes at the bone-cartilage interface in OA, with recognition that loss of integrity at the cartilage-bone junction and development of bone marrow lesions (BMLs) in the subchondral bone is associated with OA pain from large epidemiological studies. One of the next big challenges in OA BML research has been to identify the structural characteristics, gene and protein expression in OA BMLs. Gene analyses of BMLs have demonstrated that they are highly metabolically-active structures, demonstrating evidence of angiogenesis, new bone/cartilage formation and expression of neurotrophic factors. Findings from gene and protein studies of BMLs will be discussed in this talk. The gene signature of BMLs may assist in identification of new molecular targets in OA pathophysiology and treatment.