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Characterization of programmed cell death (apoptosis) in knee articular cartilage of patients with osteoarthritis

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Osteoarthritis (OA) is classically defined as a progressive degenerative rather than an inflammatory disease, and is characterized by deterioration of joints including loss of articular cartilage and subchondral bone as well as osteocyte formation. OA affects about 8 million people in the United Kingdom and nearly 27 million people in the United States. OA, one of the most common skeletal disorders, characterized by joint cartilage degradation, is induced by accumulated mechanicals stress; however little is known about the underlying molecular mechanism. Thus the present study was conduced in order to analyze the molecular changes during the apoptotic cascade in knee articular cartilage of patients with OA. Articular cartilage specimens harvest from eight patients with knee OA and two control articular cartilage samples were obtained from autopsy cases with no history of joint disease, were assessed by histology (Hematoxlyn and Eosin), histochemistry (Masson's Trichromic and Alcian Blue), immunohistochemistry through TRAIL, DR5 and Caspase-3, TUNEL and Hoechst 33258 staining in fresh isolated chondrocytes. Histology results demonstrated the structural alterations in the articular knee cartilage with OA and histochemistry results demonstrated the presence of matrix calcification and a proteoglycans reduction, respectively.

Immunohistochemistry staining showed that structural alterations, matrix calcification and a proteoglycans reduction, coincided with an increase in apoptotic cells when compared to normal cartilage, however this cellular mechanism of death was demonstrated by TUNEL and Hoechst 33258 staining in fresh isolated chondrocytes. The apoptosis positive cells might be due to a protection mechanism after sublethal injury, in particular represented by an increased survival of chondrocytes that are able to participate in the repair process.

Key words

Histology, Histochemistry, Immunohistochemistry, TUNEL, Apoptosis, Osteoarthritis