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Chondrocyte expression of apoptotic and pro-inflammatory factors in the development of post-traumatic arthritis in humans

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The development of post-traumatic arthritis following intra-articular fracture remains an important unsolved clinical problem. The possibility that extensive chondrocyte apoptosis occurs following intra-articular fracture, thus contributing to the development of post-traumatic arthritis, has received increasing attention [1]. It has been demonstrated the existence of a direct correlation between the rate of apoptosis and the severity of osteoarthritis [2]. Pharmacologic inhibitors of enzymes involved in apoptosis have been explored as potential therapeutic agents [3]. In the present study we aimed to deepen the characterization of apoptotic mediators, expressed by chondrocytes, involved in human post-traumatic arthritis following intra-articular fracture and the possible implication of pro-inflammatory receptors in arthritis. The expression of a panel of pro/anti apoptotic factors (Caspase-3, PARP-1, BCL2) and inflammation-related receptors (ChemR23) were analysed in chondrocytes from patients undergoing surgery for intra-articular calcaneal fractures. The factors were investigated by immunofluorescence coupled with confocal analysis and western blotting, followed by densitometric evaluation of chondrocyte cultures harvested from patients with intra-articular fractures compared with control ones. The results clearly demonstrated that a statistically significant difference exists in the expression of pro/anti apoptotic factors and ChemR23 between fractured and control patients. In conclusion our data suggest that increased chondrocyte death, occurring after cartilage injury together with inflammatory process, could play a pivotal role in the onset of arthritic disease.

References

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Key words
Articular fracture, post-traumatic arthritis, chondrocyte culture, apoptosis, inflammation.