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Gene expression biomarkers in the peripheral blood for postoperative pain prediction prior to knee or hip arthroplasty

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We assessed the importance of clinical indices, pain-related protease and proinflammatory cytokine gene expressions in the peripheral blood for prediction of postoperative pain (POP) development in patients with end-stage knee or hip osteoarthritis (OA) prior to arthroplasty. We examined peripheral blood of 31 hip and 50 knee OA patients undergoing joint replacement surgery and 26 healthy volunteers. Patients were tested before and 6 months after surgery. Pain was assessed prior to surgery using VAS index and neuropathic pain questionnaires DN4 and PainDETECT. Functional activity was evaluated by WOMAC. After surgery pain indices according to VAS of 30% and higher were considered. Total RNA isolated from whole blood was used in expression studies for cathepsin S, interleukin (IL)-1β, tumor necrosis factor (TNF)α, and cyclooxygenase (COX)2 genes using quantitative real-time RT-PCR. After 6 months' post-surgery pain complaints were obtained from 38.7% patients with hip OA and 34% patients with knee OA. Prior to surgery expression of cathepsin S, IL- 1β, TNFα, and COX2 genes was significantly upregulated in all examined subsets of OA patients compared with healthy controls.

Biography

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Leading Scientist, Principle Investigator, Immunology & Molecular Biology Department, Oct.2006-present. Research in genetics, cellular, and molecular physiology of osteoarthritis, rheumatoid arthritis, and osteoporosis. The importance of metabolic signalling pathways for the disease progression, inflammation, pain perception, joint destruction, and the response to therapy has been investigated in osteoarthritic, rheumatoid arthritis, and osteoporotic patients… McGILL UNIVERSITY, MONTREAL, CANADA Shriners Hospitals for Children, Joint Diseases Laboratory, Research Associate, Nov.1996-Sept.2006. Research in molecular cell biology, connective tissue physiology and bone development: The mechanisms of the onset of cartilage degradation in osteoarthritis (OA) at gene expression and protein level.