



Post-Traumatic Osteoarthritis in Musculoskeletal Medicine

Peter Min*, Philip Ghor

Department of Medical Sciences, St. Joseph Medical Center, Stockton, California, USA

DESCRIPTION

Post-Traumatic Osteoarthritis (PTOA) is a major unsolved problem in modern musculoskeletal medicine. It afflicts over 75 million individuals worldwide. It develops as a result of traumatic joint injury, usually involving Anterior Cruciate Ligament (ACL) or meniscal tears, and the incidence of these injuries is increasing over time. As PTOA progresses, fibrillation and loss of cartilage volume is accompanied by hyperplasia and fibrosis of the synovial membrane and joint capsule, subchondral bone sclerosis, and formation of bone osteophytes and cysts. Unfortunately, surgical repair does not reduce the risk of PTOA progression, and persons who suffer from advanced PTOA are often too young to be suitable candidates for joint replacement. The etiopathogenesis of PTOA has not been elucidated and there is no known cure.

This acute overload can itself cause matrix alterations, cell death and subchondral bone damage. In addition, multiple studies report that inflammatory cytokines along with cartilage and bone markers are increased in patient synovial fluids as early as 24-48 hours after knee injury. Levels of IL-6, IL-8, TNF- α , IL-1 β , IFN γ and others can remain elevated up to 5 years after injury, are associated with proteolysis of aggrecan, type II collagen and additional cartilage matrix molecules along with accompanying bone changes leading to fully-developed PTOA.

In vivo animal models of OA/PTOA and *in vitro* studies of isolated cartilage and bone have revealed important insights into degradative processes relevant to PTOA progression. Recently, more advanced *in vitro* coculture models have provided further approaches to achieve mechanistic understanding and platforms for drug discovery and drug delivery. This is particularly useful for the earliest stages of PTOA, as clinical studies in humans typically focus on more advanced OA. As PTOA is a disease of the total joint, including cartilage, bone, synovium, ligaments and menisci, *in vitro* models focusing on single isolated tissues

fail to incorporate cross-talk between tissues, which is critical to the pathophysiology of disease and evaluation of potential therapeutic targets. Experiments utilizing cell monolayers or 3D cultures of cells in scaffolds are often unable to recapitulate the native biology of fully developed extracellular matrix-rich musculoskeletal tissues.

However, given the combined effects of inflammation and mechanical trauma on the osteochondral unit in human PTOA pathogenesis, it would be most revealing to include joint capsule synovium tissue in an explant coculture system. Due to the challenge of obtaining nearly normal human knee tissues, most previous human co-culture studies have utilized tissues from knee arthroplasties. While such end-stage OA tissues may provide valuable insights, tissues from donors having no OA disease history might best enable study of cellular and mechanistic events at the earliest stages of PTOA development, to establish a foundation for discovery of biomarkers and therapeutic targets.

Therefore, the overall goal was to develop a human Cartilage-Bone-Synovium (CBS) coculture system incorporating Impact Mechanical Injury (INJ) to model early events in PTOA initiation and in an inflammatory environment. Our specific aims were to establish and characterize the CBS model involving coculture of human knee osteochondral plugs with joint capsule synovium explants harvested from the same knees, and to incorporate a single injurious impact compression of the osteochondral plugs in a manner that mimics osteochondral lesion caused by acute knee injuries, and to use this CBS + INJ system to study early PTOA-like initiation and progression, focusing on changes in cell viability, matrix composition and degradation, metabolomic readouts of individual cartilage, bone, and synovial tissues, and metabolomic changes associated with coculture of near-normal human osteochondral tissues with inflammatory synovium.

Correspondence to: Peter Min, Department of Medical Sciences, St. Joseph Medical Center, Stockton, California, USA, E-mail: Peter7@gmail.com

Received: 04-Apr-2022, Manuscript No. JCMS-22-16986; **Editor assigned:** 07-Apr-2022, Pre QC No. JCMS-22-16986 (PQ); **Reviewed:** 25-Apr-2022, QC No. JCMS-22-16986; **Revised:** 02-May-2022, Manuscript No. JCMS-22-16986 (R); **Published:** 09-May-2022, DOI: 10.35248/2593-9947.22.6.186.

Citation: Min P, Ghor P (2022) Post-Traumatic Osteoarthritis in Musculoskeletal Medicine. J Clin Med. 6:186.

Copyright: © 2022 Min P, et al. This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.