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Innate Immunity and Ischemia / Reperfusion Injury

Abstract

Ischemia reperfusion injury is a major complication in many clinical entities. In general, it represents an acute inflammatory response following an ischemic event and subsequent restoration of blood flow. It is primarily responsible for the severity of myocardial infarction, cerebral ischemic events, intestinal ischemia, and many aspects of vascular surgery, trauma, and transplantation. During ischemia, hypoxic cells undergo specific changes in enzyme activities, mitochondrial function, cytoskeletal structure, membrane transport, and antioxidant defenses. A current view is that injury results from both intrinsic and extrinsic pathways following initial ischemia and reperfusion. In the intrinsic pathway ischemia and subsequent reperfusion lead to a number of intracellular changes including mitochondrial dysfunction and production of reactive oxygen species with downstream cellular injury that if left unchecked leads to cell death. However, ischemic cells are also susceptible to a second phase of injury, i.e. the extrinsic pathway, which is mediated by natural IgM and complement. Accordingly, Alterations in cell morphology are recognized by the innate immune system resulting in an acute inflammatory response.