

Ischemia Reperfusion Injury and its Effect on the Myocardium

Anthony Mathenge

Otterbein University

Abstract

Acute myocardial infarction is a leading cause of morbidity and mortality in the world. Reperfusion strategies are the current standard therapy for acute myocardial infarction (Neri, 2017). Most clinicians assume that ischemic injury terminates with return of spontaneous circulation (ROSC). However, according to Ibáñez 2015, damage inflicted on the myocardium during acute myocardial infarction is the result of two processes: ischemia and subsequent reperfusion (Ibáñez, 2015). The purpose of the poster is to inform clinicians regarding the process of ischemic injury, before and after reperfusion. Mitochondrial permeability transition pore (MPTP) is a large mega channel that opens due to increased Ca^{++} , inorganic phosphate, or Reactive Oxygen Species (ROS), all of which are present in reperfusion. Opening of MPTP results in mitochondrial swelling and ultimately lead to rupture of the outer membrane. (Ibáñez, 2015). Reperfusion also triggers a complex inflammatory reaction accompanied by cytokine release and inflammatory leukocyte infiltration. This contributes to edema, phagocytosis, proteolysis, apoptosis, and collagen deposition (Jiaqi, 2016). Pharmacological interventions have proven to be effective in reducing reperfusion injury: Cyclosporine-A inhibits opening of MPTP, Metoprolol influences circulating neutrophils and platelets, and Glycoprotein IIb/IIIa inhibitors reduce thrombosis (Ibáñez, 2015). The “lethal reperfusion injury”, is currently accepted to significantly contribute to the final infarct size, and of late has become the topic of intensive research. There has been an increasing interest from basic and clinical researchers in identifying novel therapeutic strategies to attenuate lethal reperfusion injury and opportunities to translate these strategies into improved patient care (Duicu, 2013).

References

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