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Ischemia Reperfusion Injury and its Effect on the Myocardium

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Introduction

- 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).
- Ischemia: the occlusion of blood flow to the tissue can be detrimental to the heart due to its high energy demand (Zhou, 2015).
- Reperfusion injury: Reperfusion strategies may result in capillary destruction and hemorrhage.

Effects of Reperfusion Injury

The manifestations indicated below start occurring during myocardial ischemia, but are accelerated with reperfusion.

- Edema, swollen or ruptured mitochondria
- Impaired coronary vasomotion
- Ventricular arrhythmias
- Myocardial stunning
- Leukocyte adherence and extravasation
- Intravascular platelet and erythrocyte aggregates
- Capillary destruction and hemorrhage
- (Heusch, 2017).

Pathophysiology

Myocardial ischemia-reperfusion injury occurs when restoration of blood to ischemic heart reduces cardiac function and causes acceleration of myocardial injury through (Zhou, 2018):
- Reactive oxygen species (ROS): small amounts of ROS offer cardioprotection. However, excessive production of ROS during reperfusion causes injury (Neri, 2017).
- Platelets: thrombocytes are equipped with NADPH oxidase, an enzyme located at a cell membrane catalyzing the formation of superoxide therefore releasing ROS. These ROS are able to induce a reperfusion injury (Seligmann, 2013).
- Inflammation: reperfusion triggers a complex inflammatory reaction accompanied by cytokine release and inflammatory leukocyte infiltration into the endangered myocardial region. This contributes to edema, phagocytosis, proteolysis, apoptosis, and collagen deposition (Jiaq, 2016).
- Cytosolic Ca++ overload: during ischemia, acidosis from anaerobic glycolysis increases influx of Na+ through Na+/H+ exchange. ICF Na+ accumulation is due to inhibition of Na+/K+ ATPase due to lack of ATP. Subsequent exchange of Na+ for Ca++ by reverse mode operation of sarcolemmal Na+/Ca++ exchanger induces ICa++ overload. Upon reperfusion, the rapid normalization of pH and reenergization in the context of elevated cytosolic Ca++ induces oscillatory release and reuptake of Ca++ into the sarcoplasmic reticulum, causing excessive myocardial hypercontraction (Ibáñez, 2015).
- Edema: the high cytosolic concentrations of Na+ and Ca++ result in ICF edema when ECM osmolality is rapidly normalized by reperfusion (Ibáñez, 2015).
- Mitochondrial permeability transition pore (MPTP): a large channel that opens due to increased Ca++, inorganic phosphate, or ROS, all of which are present in reperfusion. Opening results in mitochondrial swelling and ultimately lead to rupture of the outer membrane. (Ibáñez, 2015).

Implications for Nursing Care

Pharmacologic interventions to reduce reperfusion injury:
- Cyclosporine-A: inhibiting the opening of the MPTP.
- Metoprolol: contrary to the classical theory of reduced Na+/Ca++ exchange-induced ICF Ca++ overload, upon reperfusion, the rapid normalization of pH and reenergization in the context of elevated cytosolic Ca++ induces oscillatory release and reuptake of Ca++ into the sarcoplasmic reticulum, causing excessive myocardial hypercontraction (Ibáñez, 2015).
- Glucagon like peptide-1 (GLP1) analogs: use of glucose to protect cardiomyocytes from energy depletion.
- ABC/CDI/XMR: Glycoprotein Ib/IIIa inhibitors were developed for the reduction of thrombotic events due to their potent effect on platelets and platelet leukocyte aggregates implicated in reperfusion injury (Ibáñez, 2015).

Conclusion

The “lethal reperfusion injury”, currently accepted to significantly contribute to the final infarct size, and lately 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 (Ducu, 2013).

References