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Myocardial Infarction and Protection

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Cardioprotection has been defined as a therapy that limits the infarct size during an acute myocardial infarction that may lead to better clinical outcomes [1]. Various interventions have been investigated in the past to reduce myocardial infarct size with variable success. The best strategy till date to limit infarct size has been the early restoration of coronary blood flow, with percutaneous coronary intervention-where available. Stenting following invasive reperfusion, especially in the early hours after an infraction has been found superior to all other available modalities of therapy. Attempt to decrease time from onset of symptoms to start of reperfusion and maintaining vessel patency has been proven to yield the best outcomes. Future studies powered to better assess clinical outcomes are needed for adjunctive therapy with stem cells and hypothermia.

Assessment of infarct size following an acute myocardial infarction: Infarct sizes were traditionally assessed with electrocardiographic changes and blood biomarkers including cardiac enzymes. However with the recent advances in cardiac imaging modalities, nuclear scans, echocardiography, as well as computed tomography and magnetic resonance imaging have become established modalities for assessment of infarct size [2,3].

Early Reperfusion

Of the different modalities of re-establishing coronary blood flow after an acute myocardial infarction resulting from occlusion of a coronary vessel, angioplasty with stenting has been shown to be the best in terms of improved mortality and limiting infarct size [4,5]. Certain adjunctive pharmacotherapies have improved on the success of early reperfusion with angioplasty and stenting-most notable of them have been aspirin, clopidogrel, low molecular weight heparin, and glycoprotein IIb/IIIa platelet inhibitors [6-9]. Other agents (as noted below) have reduced infarct size irrespective of invasive management for myocardial infarction.

Pharmacotherapy for Cardioprotection

Beta-blockers administered early and through intravenous route have been consistently shown to improve mortality [10] and reduce infarction size [11]. The early intravenous administration of beta-blockers has recently fallen out of favor due to unfavorable results of the COMMIT-CCS2 trial-however; poor patient selection may have been contributory [12,13].

Glucose-insulin-potassium infusion has been purported to reduce mortality and reduce infarct size, especially for diabetic patients-however recent trials have thrown this hypothesis into serious question [14,15].

Adenosine or adenosine receptor agonists have been shown to reduce the extent of myocardial damage from AMI in some experimental studies [16] however a definitive outcomes trial is still awaited.

Therapeutic hypothermia has been shown to reduce infarct size in clinical studies [17]. However whether that reduction in infarct size translates into clinically meaning reductions of hard endpoints remains investigational.

Stem cell therapy for myocardial infarction has been shown to consistently reduce infarct size and improve systolic function-however, most trials of stem cell therapy in myocardial infarction have been small studies-hence this promising therapeutic modality too awaits a conclusive trial with adequate sample size and power [18].

Intra-aortic balloon pump, once believed to reduce infarct size has fallen out of favor recently [19].

Future Directions

Many of the newer pharmacologic agents that show promise in smaller studies need validation in adequately powered randomized trials. Stem cell therapy also appears extremely promising, however may need fine tuning before being incorporated into clinical practice.

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