Traditional & Emerging Medical Treatment for STEMI

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The management of acute myocardial infarction continues to undergo major changes. Because of the great number of trials on new treatments performed in recent years, the ESC and AHA/ACC upgraded the previous guidelines. For patients with the clinical presentation of STEMI within 12 h of symptom onset and with persistent ST-segment elevation or new or presumed new LBBB, early mechanical (PCI) or pharmacological reperfusion should be performed as early as possible to save the myocardium. Because PCI leads to endothelial denudation, exposure of the subendothelial matrix triggers a complex cascade that, in the absence of adequate antiplatelet therapy, may culminate in thrombus formation in the epicardial vessels and thromboembolism of the coronary microvasculature. Therefore, anticoagulant and antiplatelet drugs are the main cornerstones of therapy in the treatment of STEMI. Following a STEMI, resultant myocardial infarct has been strongly linked with the development of adverse LV remodelling and heart failure. Aangiotensin converting enzyme inhibitors and statins are very effective for the prevention of secondary events after myocardial infarction. .

More recently, a number of attempts to reduce MI size in patients presenting is ongoing. Preliminary clinical studies have explored the value of myocardial pre- and post-conditioning to improve myocardial salvage. However, the results are conflicting. Given the preliminary nature of these findings and the small size of the trials, confirmation of a clinical benefit of myocardial pre- and post-conditioning by ongoing randomized trials is warranted before these procedures can be recommended in routine clinical practice.

New developing and emerging fields in the management of STEMI patients in the acute phase are represented by the metabolic approach, the treatment of the reperfusion injury and the stem cell therapy. The goal of ongoing research on the pharmacological approach to STEMI patients is a prevention of reperfusion injury. Recent trials with STEMI have also failed to meet their primary end point of cardioprotection—these have included studies investigating therapeutic hypothermia, targeting mitochondrial function, and modulation of nitric oxide signalling as adjuncts to myocardial reperfusion. Clinical cardioprotection research remains a challenge—mortality rates following a PPCI-treated STEMI are in decline, which makes demonstrating a further reduction in MI size and improved clinical outcomes increasingly more difficult. However, Initial data suggest that using a combination of therapies to target myocardial reperfusion injury may be more beneficial than using a single therapy approach, and using this approach may result in improved clinical outcomes in this patient group.