Thrombolytic Therapy in Acute Myocardial Infarction

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Introduction

Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide. The heart requires its own constant supply of oxygen and nutrients, like any muscle in the body. It receives oxygenated blood through left and right coronary arteries. Most cases of acute myocardial infarction are caused by coronary artery plaque rupture with subsequent thrombus formation.1

Infarction may be
• Transmural
• Nontransmural

Transmural infarcts involve the whole thickness of myocardium from epicardium to endocardium and are usually characterized by abnormal Q waves on ECG. Nontransmural or subendocardial infarcts do not extend through the ventricular wall and cause only ST-segment and T-wave abnormalities. Subendocardial infarcts usually involve the inner one third of myocardium, where wall tension is highest and myocardial blood flow is most vulnerable to circulatory changes.2,3

Non–ST-segment elevation myocardial infarction (NSTEMI, subendocardial MI) is myocardial necrosis (evidenced by cardiac markers in blood; troponin I or troponin T and CK will be elevated) without acute ST-segment elevation. ECG changes such as ST-segment depression, T-wave inversion, or both may be present.2,3

ST-segment elevation myocardial infarction (STEMI, transmural MI) is myocardial necrosis with ECG changes showing ST-segment elevation that is not quickly reversed by Nitroglycerin. Cardiac markers, troponin I or troponin T, and CK are elevated.2,3

When thrombosis leads to total occlusion of blood flow, acute ST-elevation myocardial infarction (STEMI) is often the clinical outcome. Prompt reestablishment of blood flow in the culprit artery is the most important goal in the management of STEMI irrespective of the strategy used. Primary percutaneous coronary intervention (PCI) is the treatment of choice and should be performed within 120 minutes from the first medical contact. When it is not feasible, thrombolysis should be administered.2,3

Usually, the first symptom of infarction is deep, substernal, visceral pain described as aching or pressure, often radiating to the back, jaw, left arm, right arm, shoulders, or all of these areas. The pain is similar to angina pectoris but is usually more severe and long-lasting; more often accompanied by dyspnea, diaphoresis, nausea, and vomiting; and relieved little or only temporarily by rest or Nitroglycerin. About 20% of acute MIs are silent, more commonly in patients with diabetes. Patients often interpret their discomfort as indigestion, particularly because spontaneous relief may be falsely attributed to belching or antacid consumption.2,3

Drug Treatment of Acute Myocardial Infarction2,5,6,7,8,9

All patients should be given Antiplatelet drugs, Anticoagulants, Statins and if chest pain is present, Antianginal drugs. Patients with acute STEMI should receive coronary reperfusion therapy with either PCI or fibrinolysis.

Patients with AMI should be given the following:
• Antiplatelet drugs: Aspirin, Clopidogrel or both (Prasugrel or Ticagrelor are alternatives to Clopidogrel)
• Anticoagulants: Heparin (unfractionated or low molecular weight heparin)
• Glycoprotein IIb/IIIa inhibitor for some high risk patients
• Antianginal therapy usually Nitroglycerin
• Beta-blocker
• ACE inhibitor
• Statin

All patients are given Aspirin 160 to 325 mg (not enteric-coated), at presentation. Chewing the first dose before swallowing quickens absorption. Aspirin reduces short-term and long-term mortality risk. In patients undergoing PCI, a loading dose of Clopidogrel (300 to 600 mg po once), Prasugrel (60 mg po once), or Ticagrelor (180 mg po once) improves outcomes, particularly when administered...