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TREATMENT OF ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION

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Abstract: Myocardial infarction (MI) is a term used for an event of heart attack which is due to formation of plaques in the interior walls of the arteries resulting in reduced blood flow to the heart and injuring heart muscles because of lack of oxygen supply[14]. Acute myocardial infarction has traditionally been divided into ST elevation or non-ST elevation myocardial infarction; STEMI results from complete and prolonged occlusion of an epicardial coronary blood vessel and is defined based on ECG criteria while NSTEMI usually results from severe coronary artery narrowing, transient occlusion, or microembolization of thrombus and/or atheromatous material, however, therapies are similar between the two, and the overall management of acute myocardial infarction can be reviewed for simplicity. Acute myocardial infarction remains a leading cause of morbidity and mortality worldwide, despite substantial improvements in prognosis over the past decade. The progress is a result of several major trends, including improvements in risk stratification, more widespread use of an invasive strategy, implementation of care delivery systems prioritising immediate revascularisation through percutaneous coronary intervention (or fibrinolysis), advances in anti-platelet agents and anticoagulants, and greater use of secondary prevention strategies such as statins.[15] Treating a STEMI is time-sensitive. That means the faster the treatment, the better the chances for a favourable outcome.

IndexTerms -Myocardial infarction, STEMI, NSTEMI, ischemia,cardiac troponin.

INTRODUCTION:

Myocardial infarction (MI) is a syndrome where myocardial cells die due to imbalances between myocardial oxygen supply and demand.[1] In 2007, the Task Force for the Universal Definition of Myocardial Infarction simultaneously published an international consensus document in the Journal of the American College of Cardiology, the European Heart Journal, and Circulation. The 2007 document was an updated revision of the original document from this group that had first been published in 2000. In this second communication, the task force defined 5 subtypes of myocardial infarction (MI), which were retained in the 2012 revision.[2]

Type 1

Type 1 MI is due to acute coronary atherothrombotic myocardial injury with either plaque rupture or erosion and, often, associated thrombosis. Most patients with ST-segment elevation MI (STEMI) and many with non-ST-segment elevation MI (NSTEMI) fit into this category. [4]

Type 2

Type 2 MI includes patients with evidence of acute myocardial ischemia who do not have acute coronary atherothrombotic injury but instead have oxygen supply-demand imbalance from other reasons. This occurs most often due to the presence of coronary artery disease, which limits increases in coronary perfusion in cases of severe anaemia, significant arrhythmias and other stressors. However, the presence of fixed coronary obstruction is not obligatory, including primary coronary causes such as vasospasm, coronary embolus, and coronary artery dissection.[4]

Type 3

Type 3 MI continues the concept that there may be an occasional patient who has characteristic symptoms of myocardial ischemia but whose cTn values have not become elevated because the patient succumbs before values are measured or who is stricken by sudden death with evidence of MI by autopsy.[4,12]

Types 4 and 5

Types 4 and 5 MIs are related to coronary procedural events.[4]

Pathophysiology of ST-segment Elevation Myocardial Infarction

For an acute thrombotic coronary event to cause ST-segment elevation on a surface ECG, there needs to be a complete and persistent occlusion of blood flow. Coronary atherosclerosis and presence of high risk thin cap fibroatheroma (TCFA) can result in sudden onset plaque rupture. This results in changes in vascular endothelium resulting in cascade of platelet adhesion , activation and aggregation resulting in thrombosis formation.[5] When thrombus completely occludes the artery and causes infarction of tissue distal to the thrombus. The complete obstruction of a coronary artery, results in the death of heart muscle tissue, which is referred to as STEMI, the worst form of ACS.

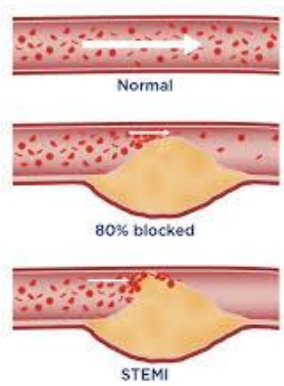

Fig.1 occlusion of artery during stemi

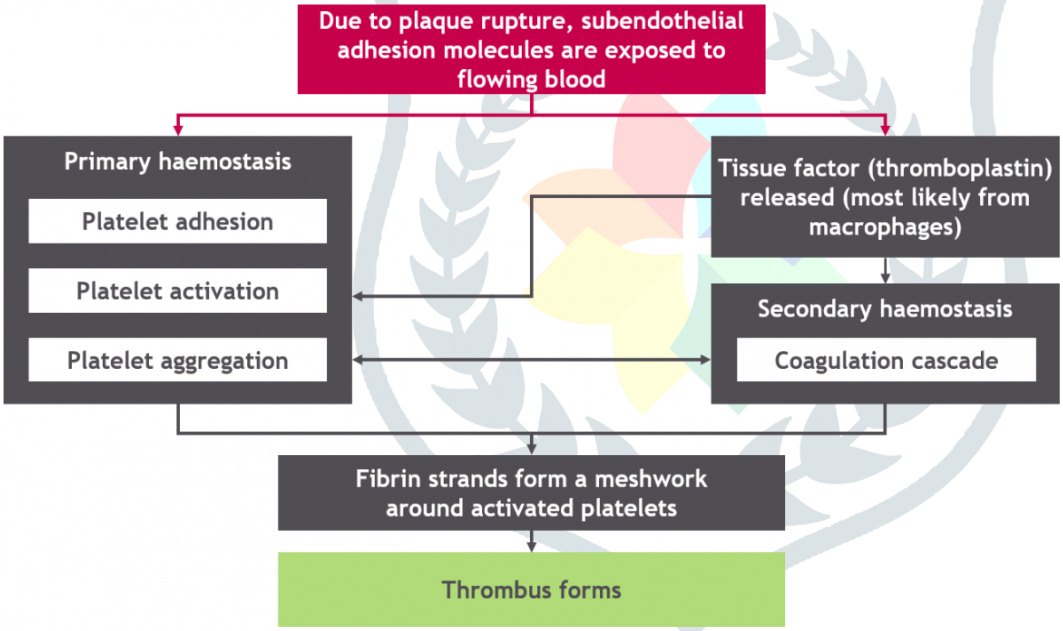

Fig.2 pathophysiology of stemi[16]

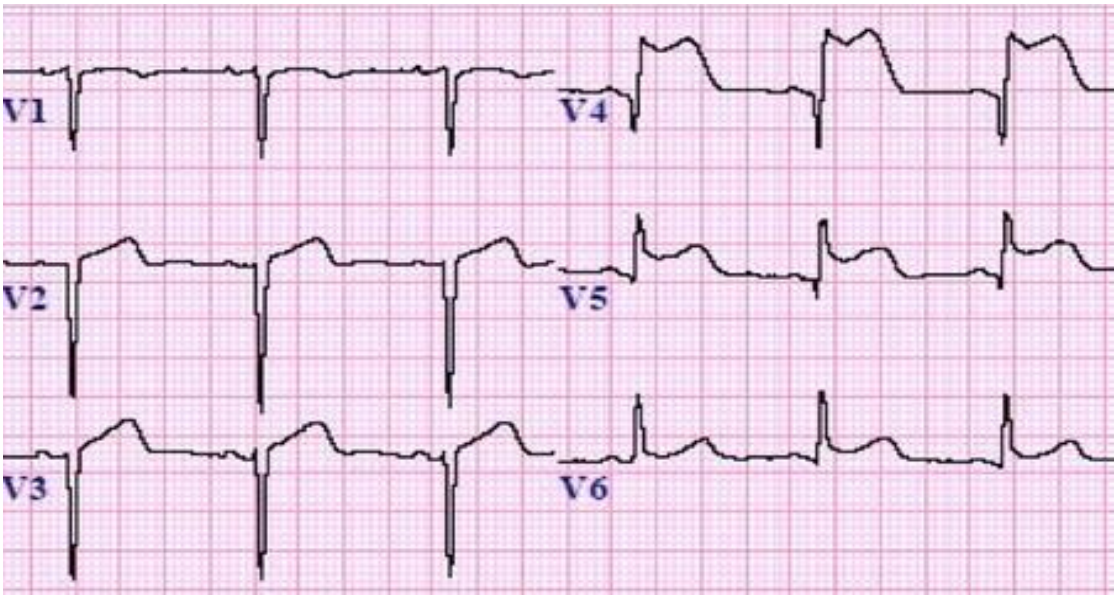

Fig.3 ecg of stemi[6]

Clinical Presentation

Patients who are experiencing an acute STEMI are classically described as presenting with pressure or crushing chest pain associated with shortness of breath, nausea/vomiting or diaphoresis. The chest pain might be described as radiating into the patient's neck, left arm or jaw. Often times, they will appear visibly uncomfortable and may have a fist clutching the centre of their chest. This classic finding of angina is known as the Levine sign.[9]

Diagnosis of STEMI

The diagnosis is straightforward using the Electrocardiogram(ECG). Pre-hospital personnel has proven to be highly capable of recognising STEMI using 12 lead ECG.Moreover patient who utilise the EMS may have better outcomes, since several evidence based therapies(including reperfusion) may given in Pre-hospital setting.Obviously measurement of cardiac troponins is not necessary to establish diagnosis of acute STEMI; the diagnosis is based on clinical presentation(notably chest pain)and ST elevation on ECG.Nevertheless cardiac troponins are always analysed once the situation allows.[11,8]

Treatment of ST-segment elevation myocardial infarction

Initial medical therapy during STEMI consists of oxygen administration, anti-platelet therapy (aspirin, thienopyridines and glycoprotein IIb/IIIa inhibitors), anticoagulation, anginal pain relief with nitrates and morphine, and beta-blockade.[10]
Emergency reperfusion of ischemic myocardium that is in the process of becoming infarcted is the most important advance in the treatment of STEMI over the past three decades and is the primary therapeutic goal.Coronary reperfusion is accomplished by means of primary Percutaneous Coronary Intervention(PCI) (angioplasty and stenting) or intravenous fibrinolytic therapy. Prompt PCI (with a performance goal of ≤90 minutes from the first medical contact) is the preferred approach at PCI-capable hospitals for STEMI with onset of symptoms within the previous 12 hours and for STEMI with cardiogenic shock, regardless of the timing . The advantages of primary PCI over fibrinolysis include lower rates of early death, reinfarction, and intracranial hemorrhage. However, when PCI is delayed by more than 120 minutes, fibrinolytic therapy should be given if it is not contraindicated , followed by routine consideration of transfer in the following 3 to 24 hours to a PCI-capable facility .With broad application of reperfusion therapy for STEMI, 30-day mortality rates have progressively declined from more than 20% to less than 5%.[3]
Medical therapy upon hospital discharge may include ACE inhibitors, angiotensin receptor blockers, aldosterone antagonists and HMG CoA reductase inhibitors.[10]

Aspirin: Aspirin should be chewed at a dose of 162 to 325 mg immediately once STEMI is diagnosed unless a contraindication exists. Lifelong therapy using 75-162 mg daily should follow upon hospital discharge.[10]
Thienopyridines: P2Y212 receptor antagonists (clopidogrel, prasugrel, ticagrelor, and ticlopidine) are indicated in all STEMI cases unless surgery is needed. Clopidogrel can also be used as an adjunct to fibrinolytic therapy in patients intolerant to aspirin. If coronary artery bypass grafting is required, these agents should not be used. These agents must be discontinued for 5-7 days prior to CABG(coronary artery bypass graft) unless urgent and the bleeding risk is less than the benefit of revascularization. Regardless of the type of stent used during PCI, thienopyridines are preferred to be continued for 12 months if possible. Prasugrel is not recommended in a patient with a prior history of stroke or transient ischaemic attack (TIA). Ticlopidine is rarely used due to the risk of thrombocytopenia and TTP (thrombotic thrombocytopenic purpura).[3,10]
Glycoprotein IIb/IIIa inhibitors: These drugs include abciximab, eptifibatide, and tirofiban. They very strongly inhibit platelet function by blocking the binding of fibrinogen to the activated glycoprotein IIb/IIIa receptor complex. Any of these agents may be used in addition to aspirin, a thienopyridine and anticoagulation (except with bivalirudin) at the time of PCI in high risk patients with STEMI. Using glycoprotein IIb/IIIa inhibitors prior to PCI does not have strong data to support its use at the present time.[3,10]
Anticoagulation: Full anticoagulation should be started in all STEMI patients unless a contraindication exists. Either unfractionated heparin, low molecular weight heparin (enoxaparin or fondaparinux) or bivalirudin can be used. Unfractionated heparin for 48 hours total and low molecular weight heparin for 8 days or until hospital discharge.

Nitrates: Nitrates are helpful to treat angina symptoms, hypertension and heart failure during STEMI, however no clinical data exists to show a mortality benefit and thus their use is individualized. The use of nitrates should not preclude using drugs that do show a mortality benefit.[3,10]

Sublingual nitroglycerine tablets administered every 3-5 minutes with a maximum dose of three tablets can be given to relieve angina. Should angina persist, then intravenous nitroglycerine can be considered. Hypotension or right ventricular involvement is a contraindication to their use. Phosphodiesterase inhibitors (sildenafil, vardenafil, tadalafil) used to treat erectile dysfunction enhance nitric oxide production and can cause potentially fatal hypotension when used in combination of nitrates. These two drugs should not be used together within 24 hours (sildenafil) or 48 hours (vardenafil, tadalafil) due to this interaction.[10]

Morphine: Morphine is effective to relieve anginal chest pains and the sensation of dyspnea when pulmonary edema is present. There are also some beneficial hemodynamic effects including arterial vasodilation.[10]

Beta-blockers: While there is little data in regards to the efficacy of beta-blockers during UA/NSTEMI, there is an abundance during STEMI. Guidelines from the American Heart Association recommend early intravenous beta-blockers when no contraindication exists and there is angina, hypertension or tachycardia not related to heart failure. Otherwise, oral beta-blocker therapy is given in the acute setting. It is important NOT to give beta-blockers if there are signs of cardiogenic shock such as hypotension or pulmonary edema on chest x-ray. Long-term therapy (lifetime) has been shown to reduce MI incidence and improve mortality. Also, if left ventricular systolic dysfunction remains after a STEMI, beta-blockers are important for chronic systolic heart failure.[10]

ACE inhibitors/Angiotensin receptor blockers: Either an ACE inhibitor or angiotensin receptor blocker should be given to all STEMI patients upon hospital discharge. Caution must be used in the acute setting in order to avoid hypotension which can worsen myocardial ischemia. Guidelines give the use of these drugs a class I indication when there is left ventricular systolic dysfunction or if the patient is diabetic. When left ventricular function returns to normal and the patient is not diabetic, the benefits are less clear. Usually ARBs are only given if ACE inhibitors are not tolerate due to cough or other side-effects.[10]

Aldosterone antagonists: The aldosterone antagonist eplerenone was evaluated in the EPHESUS trial leading to the recommendation for their with an ACE inhibitor prior to hospital discharge after UA/NSTEMI if there is left ventricular systolic dysfunction (EF < 40%) and either diabetes or symptomatic heart failure present and no contraindication (serum creatinine > 2.5 and or potassium > 5.0). A class effect is likely present and thus spironolactone is frequently used instead of eplerenone due to cost concerns, although there is no direct data to support this practice.[10]

HMG-CoA reductase inhibitors: Every patient with STEMI should receive therapy with a statin. The 2013 ACC/AHA cholesterol guidelines recommend high intensity statin therapy (defined as LDL reduction > 50%) in those age < 75 and moderate intensity (defined as 30-50% reduction of LDL) in those > 75 years old. No specific target LDL are recommend in these guidelines, simply a reduction of LDL levels from baseline. The MIRACLE and PROVE-IT TIMI 22 trial used atorvastatin 80 mg PO daily with good results. Statin therapy should be lifetime after a person has an acute coronary syndrome unless a contraindication exists.[10]

Calcium Channel Blockers: The non-dihydropyridine calcium channel blockers diltiazem and verapamil can be used when there is a contraindication to beta-blockers (such as asthma) and there is no heart failure or significant left ventricular systolic dysfunction present. Sublingual nifedipine is contraindicated due to a reflexive increase in the sympathetic nervous system which can be harmful.[10]

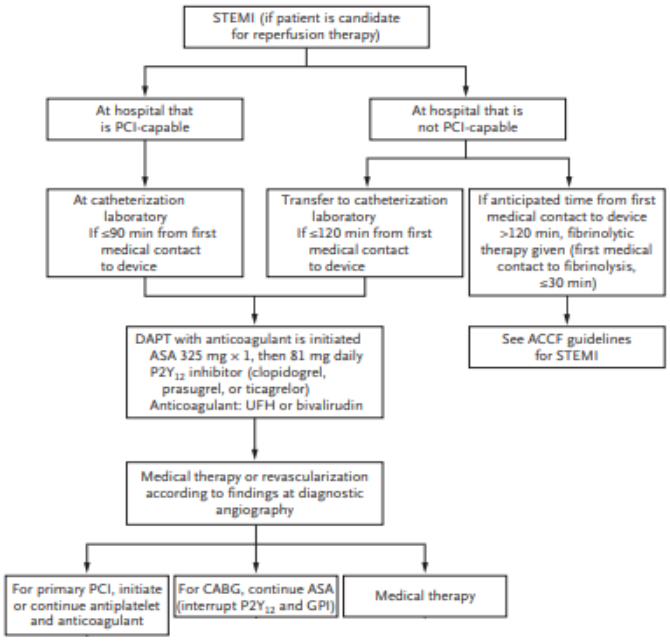

Fig.4 Flowchart

CONCLUSION:

The overall prevalence of STEMI among younger patients was 12.8%. There was male dominance (96.8%). Smoking (37.6%) was observed to be the most common risk factor for young STEMI, followed by diabetes mellitus (16.8%) and hypertension (16%). The most commonly used management strategy was mechanical revascularization (43.2%), followed by thrombolysis (28.8%) and medical management (28%).The young MI patients are unique in having male dominance, better outcome, more of single-vessel disease with significant number of normal coronaries, better response to mechanical as well as pharmacological revascularization.[13]

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