Acute Myocardial Infarction- The Changing Face

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Much water has flown under the bridge in the diagnosis and treatment of acute myocardial infarction of yore when the patient was put to bed for two or three weeks, there was hardly any treatment apart from pain killers and nitrates and the value of rehabilitation was something unheard of. This issue of the journal emphasises the recent advances in the diagnosis, treatment and rehabilitation.

ST Elevation Myocardial infarction (STEMI) is a major public health problem in both the developed and the developing countries in the world. The incidence in the developing countries is now similar to that in the developed countries.1

Diagnosis of myocardial infarction has two components. A pathological component which requires evidence of myocyte cell death as a consequence of prolonged ischaemia and a clinical diagnosis which requires an assessment of the history with evidence of infarction using electrocardiographic, biochemical and imaging modalities. Classically the criteria required the presence of at least two of the following three criteria to establish the diagnosis of myocardial infarction: Characteristic symptoms, electrocardiographic changes and typical rise and fall in the biochemical markers.2 Since the earlier publications many advances in the laboratory and electrocardiographic aspects of the definition have occurred. The assays for biochemical parameters have become more specific. Keeping this in mind a consensus document with regard to the revised definition of acute myocardial infarction was published jointly by the European society of cardiology and the American College of Cardiology.3

Revised Definition of Myocardial Infarction (MI)3

Criteria for acute, evolving or recent MI

Either one of the following criteria satisfies the diagnosis for an acute, evolving or recent MI

1. Typical rise and gradual fall (troponin) or more rapid rise and fall (CPK-MB) of biochemical markers of myocardial infarction with at least one of the following:
   a. Ischaemic symptoms
   b. Development of pathologic Q waves on the ECG reading
   c. ECG changes indicative of ischaemia (St segment elevation or depression)
   d. Coronary artery intervention(e.g. Coronary angioplasty)

2. Pathological findings of acute MI

Criteria for established MI

Either of the following criteria satisfies the diagnosis for established MI:

1. Development of new pathological Q waves on serial ECG readings. The patient may or may not remember previous symptoms. Biochemical markers of myocardial necrosis may have normalised, depending on the length of time that has passed since the infarct developed.

2. Pathological findings of a healed infarct or healing MI. In this issue Wander has drawn attention to a correct diagnosis after proper history taking. He has stressed on the difficulties in diagnosing infarction in the presence of Left Bundle Branch Block and on the newer modalities of diagnosis.

Prehospital Thrombolysis

STEMI is fatal in about one third of the patients, with 50% deaths occurring in the first hour from ventricular tachyarrhythmias. Time to thrombolysis remains a key modifiable determinant of mortality in STEMI.1,5 A prehospital treatment strategy when compared with in-hospital thrombolysis has been shown to reduce time to thrombolysis with around one hour and in-hospital mortality by 17% in a meta-analysis of randomized trials.6 A nationwide registry (>13000 patients) of real-life patients showed that prehospital diagnosis and treatment are associated with reduced time to thrombolysis by almost 1 h and reduced adjusted long-term mortality by 30%. Importantly, prehospital diagnosis established by a physician at the hospital using telemedicine and subsequent pre hospital thrombolysis (PHT) delivered by paramedics in the ambulances seem as efficient in reducing time delays as physician-staffed ambulances.7 The results of a nationwide prospective registry of patients admitted to intensive care units for acute myocardial infarction in November 2000 in France show that prehospital thrombolysis therapy with liberal use of early angioplasty offers 1-year mortality results that are at least as satisfactory as those with primary coronary angioplasty. In patients treated very early (those admitted within 3.5 hours of onset of chest pain), PHT offers superior efficacy compared with any other mode of reperfusion therapy.8 Here, Vaishnav et al have dealt with the need for domiciliary thrombolysis and the need for TIMI 3 flow. They have also pointed out the increased effect by the addition of aspirin. At the same time they have cautioned the use in NESTMI.

Approach to STEMI and Non STEMI

The ACC and AHA guidelines suggest an approach that integrates the information from history, examination, electrocardiogram, cardiac biomarkers to assign the patient to four categories- noncardiac chest pain, stable angina, possible acute coronary syndrome or definite acute coronary syndrome.9

In these guidelines, patients with STEMI are triaged immediately for reperfusion therapy and those with unstable angina or non ST elevation MI are admitted to hospital for risk stratification and treated conservatively or an early invasive strategy is adopted depending on the quantum of risk that is involved.
Thrombolysis in the Era of Intervention

Once a patient with STEMI presents, then an assessment is made of the different reperfusion options.\textsuperscript{10}  

**Step 1: Assess time and risk**  
- Time since symptom onset.  
- Risk of STEMI  
- Role of fibrinolysis  
- Time required for transport to a skilled PCI laboratory  

**Step 2: Determine if fibrinolysis or invasive strategy is preferred**  
If presentation is < 3 hours and there is no delay to invasive strategy, there is no preference for either strategy.  
Fibrinolysis is preferred if:  
- Early presentation (<3 hours from symptom onset and delay to an invasive strategy)  
- Invasive strategy is not an option  
- Catheterisation laboratory is occupied or not available  
- Vascular access difficulty  
- Lack of access to a skilled PCI laboratory (Individual operator experience of > 75 primary PCI cases/year or team experience of > 36 primary PCI cases/year)  
- Delay to invasive strategy  
- (Door to balloon)-(Door to needle time) more than 1 hour.  
- Medical contact to balloon or door to balloon time more than 90 minutes.  

Kaul et al have emphasised on the early use of fibrinolytic agents and antiplatelet drugs.

Recent Advances in the Management of AMI

An invasive strategy is preferred if:  
- Skilled PCI laboratory is available with surgical back up.  
- High risk STEMI  
  - Cardiogenic shock  
  - Killip class >3  
- Contraindications to fibrinolysis including increased risk of bleeding and intracranial haemorrhage.  
- Late presentation  
  - Symptom onset was more than 1 hour ago.  
- Diagnosis of STEMI is in doubt.  

Haemodynamically unstable patients, patients in cardiogenic shock and rarely those with refractory ischaemia, may require additional support with intraaortic balloon counter pulsation (IABP). IABP reduces preload and increases coronary perfusion in diastole. Percutaneous left ventricular assist devices may also allow time for the stunned or hibernating myocardium to recover.\textsuperscript{11}  

Discussing recent advances Mardikar et al have highlighted the different modalities available, including thrombus aspiration. The TAPAS trial showed a decline in mortality rate at the end of 30 days in patients who had undergone aspiration. Newer antiplatelet agents like prasugel and ticagrelor have also helped in lowering the restenosis rate. They have drawn atten to the possibility of stem cells in revascularisation.

Guidelines of AMI Management\textsuperscript{12}  

Each geographic locality should have its plan for management of STEMI patients, including which hospitals should receive them from the community. Units capable of obtaining a rapid ECG diagnosis and risk stratifying these patients should transfer them expeditiously to hospitals with facilities for early percutaneous coronary intervention. Early pharmacotherapy including antiplatelet agents, anticoagulant, statin should be initiated in the receiving hospital. In this issue Iyengar and Godbole have stated that even with PAMI being practised more frequently thrombolysis still has a part to play in early revascularisation, especially with the ready availability of newer antithrombolytics like tenectaplaste. However, they also stress that TAMI 3 flow may not be achieved always and that PAMI may have to be taken recourse to. Banerjee and Kumar have emphasised the early and late modalities of management.

Management of Complications in AMI

Complications in AMI may be electrical (tachy or brady arrhythmias) or mechanical. It is important to recognise these early to ensure survival and recovery of the patient with AMI. Cardiogenic shock may result from right ventricular infarction, free wall rupture, ventricular septal rupture, papillary muscle rupture or pump failure resulting from infarction of a large territory of myocardium.

Early echocardiography can help in identifying and differentiating the causes of mechanical failure. Five therapeutic modalities are used to treat patients in Cardiogenic shock which include vasopressors, mechanical support with IABP or ventricular assist devices, fibrinolysis, PCI and CABG. The first two treatment modalities are useful but temporary modalities. Revascularisation is associated with an improvement in survival. The SHOCK trial\textsuperscript{13} evaluated the outcome in patients with AMI in Cardiogenic shock. 86% patients received IABP and two groups were randomised to early revascularisation with PCI or CABG or to initial medical stabilisation. The mortality was lower and long term survival was better in the group randomised to early revascularisation.

Free wall rupture, ventricular septal rupture and papillary muscle rupture require early operative intervention after early stabilisation with IABP. Surgical survival is determined by early surgery, short duration of Cardiogenic shock and milder degree of systolic biventricular impairment.\textsuperscript{14}  

First degree and second degree Type I blocks do not affect survival and do not require invasive therapy with pacing. However if the ventricular rate falls below 50 beats/min and the patient is symptomatic treatment with atropine is indicated. Transvenous pacing is almost never required in this situation.

Complete AV block can occur in patients with anterior and inferior infarction. In patients with anterior infarction complete heart block can result suddenly after 12-24 hours and occurs due to extensive septal necrosis which involves the bundle branches. The prognosis is poor in this group of patients even after pacing because of the extensive myocardial necrosis which has occurred. In this issue Mullassari et al have dealt extensively with the complications arising from infarctions. Cardiac failure and arrhythmias are commonly seen though complications like myocardial rupture and aneurysm formation are relatively uncommon. Heart blocks are quite common and often transitory.
Future of Thrombolytic Therapy-An Indian Perspective

An estimated 31.8 million in India have coronary heart disease (CHD). This translates into 11% prevalence in urban India. However, hospitals with PCI capabilities may not be available to all, especially in rural areas and smaller cities. If patients present to the community hospitals (which have no facilities for PCI) in these areas within 12 hours of onset of infarction (LATE trial), thrombolysis is an option that may be used. The data from 22 trials of fibrinolytic therapy was pooled and it was seen that the earlier the treatment was started after the onset of symptoms, the greater was the benefit. The benefit decreased in a non-linear fashion with incremental delays in time to thrombolysis after onset of symptoms. The greatest benefit was in the first hour after the onset of symptoms (the Golden Hour). Also one must keep in mind that many of the patients may not be affording PCI and majority are not insured. In this scenario, thrombolysis if given within the Golden hour may prove to be not only life saving but also a cost-effective approach to treat AMI in our country.

Cardiac Rehabilitation after MI

The effect of exercise training after MI is to improve exercise capacity and quality of life. Exercise training helps in decreasing the myocardial oxygen demand and also helps in improving the endothelial function. All patients after acute MI should be encouraged to enroll for a physician supervised rehabilitation program. Many hospitals have started offering this rehabilitation as a package included with the PCI, where patients do not have to bear any additional cost. However if approved and reimbursed by insurance companies, then it will see a greater rise in utilisation by patients. In this issue Contractor has dealt extensively on the process of early and late rehabilitation.

References

4. Fibrinolytic Therapy Trialists’ (FTT) Collaborative Group, Indications for fibrinolytic therapy in suspected acute myocardial infarction: Collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1000 patients. Lancet 1994;343:311-322