Managing Complications in Acute Myocardial Infarction

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Abstract

Acute myocardial infarction (AMI) due to coronary artery disease is a leading cause of death in both the developed and developing countries. The advent of coronary care units and early reperfusion therapy (Thrombolytic and Percutaneous Coronary Intervention) has substantially decreased in-hospital mortality rates and has improved the outcome in survivors of the acute phase of MI.

Complications of AMI include mechanical, arrhythmic, ischemic, and inflammatory (early pericarditis and post-MI syndrome) sequelae, as well as left ventricular mural thrombus. In addition to these broad categories, right ventricular (RV) infarction and cardiogenic shock are other common complications of AMI. The onset of each of these complications usually results in explicit symptoms and physical manifestations. Thus, a basic knowledge of the complications that occur in the postinfarction period and the clinical syndromes associated with each, will allow the physician to evaluate and treat the complication appropriately.

Compared to AMI patients who receive aggressive therapy (6.5-7.5%) versus those AMI patients in the community (15-20%), the former has a significant decrease in the short term mortality rate.1,2 From a global perspective, the burden of MI in developing countries may soon approach those now afflicting developed countries. Most of these deaths are the direct result of pathophysiologic changes which occur as a result of the AMI. Many more patients suffer from complications of AMI. These patients require prompt recognition of their condition and aggressive management in order to prevent unnecessary morbidity and mortality. The advent of coronary care units (CCU) and early reperfusion therapy has substantially decreased in-hospital mortality rates and has improved the outcome in survivors of the acute phase of MI.

Complications of AMI can be broadly classified into:

A. Mechanical
B. Arrhythmic
C. Ischemic
D. Inflammatory
E. Embolic

A. Mechanical Complications

1. Left Ventricular (LV) Failure and Cardiogenic Shock.

LV dysfunction is the single most important predictor of mortality following AMI.3 Hemodynamic classification of AMI was done by Killip and Kimball on the basis of clinical presentation and physical findings at the onset of AMI and by Forrester et al on the basis of invasive monitoring.4,5

Incidence of Heart Failure in AMI is given in Table 1.

Table 1: Incidence of Heart Failure in AMI.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Patients (%)</th>
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<tbody>
<tr>
<td>I No evidence of congestive heart failure</td>
<td>85</td>
</tr>
<tr>
<td>II Rales, ↑ jugular venous distension, or S3</td>
<td>13</td>
</tr>
<tr>
<td>III Pulmonary oedema</td>
<td>1</td>
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<tr>
<td>IV Cardiogenic shock</td>
<td>1</td>
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The 30-day mortality rate for patients with cardiogenic shock in GUSTO I trial was 58%.6

Hemodynamic monitoring with an arterial line and pulmonary artery catheter is helpful. Echocardiography (2D-Echo) determines the extent of myocardial involvement and other complications of AMI which contribute to cardiogenic shock.

In patients with cardiogenic shock an intraaortic balloon pump (IABP) insertion is priority as it reduces the afterload, improves cardiac output (CO) and decreases the myocardial oxygen requirement. Temporary Percutaneous Left Ventricular Assist Device (LVAD) allow time for recovery of stunned or hibernating myocardium. Inotropic and vasopressor agents may be given at the lowest possible doses. Adding vasodilators in conjunction with IABP and inotropic agents increase CO and improve coronary perfusion pressure. Survival improvement is associated only with percutaneous coronary intervention (PCI) or coronary artery bypass grafts (CABG).7 The SHOCK study revealed that long-term survival improved significantly in patients with cardiogenic shock who underwent early revascularisation.

2. Right Ventricular Myocardial Infarction (RVMI)

Mild right ventricular (RV) dysfunction is common after inferior or inferoposterior wall MI; however hemodynamically significant RV impairment occurs in 10% of patients. As RV has lower O2 requirement, is thin walled, is perfused during systole and diastole, and has collateral blood flow from the left anterior descending artery and the thebesian veins, RVMI rarely causes irreversible damage. The triad of hypotension, JVP distension with clear lungs and absence of dyspnea is specific for RVMI. Kussmaul’s sign may be present.

ECG usually shows inferior wall MI (IWMI) and ST elevation in V4R has a positive predictive value of 80%. 2D-Echo demonstrates RV dilation, severe RV dysfunction and associated LV dysfunction. PA catheterisation reveals high right atrial (RA) pressures with low PCWP.

Management of RVMI involves volume loading, to increase preload and CO but the target CVP is...
approximately 15mm Hg. If volume loading fails to increase CO, inotropes may be added. Successful reperfusion improves RV function and decreases 30-day mortality rates.

Tricuspid valve replacement or repair with annuloplasty rings is required for severe tricuspid regurgitation caused by RVMI. RV assist device is indicated in patients who remain in cardiogenic shock despite the foregoing measures.

3. Ventricular Septal Rupture (VSR)

VSR occurred in 1-2% patients after acute MI in the prethrombolytic era but the incidence has decreased in the post thrombolytic era. VSR may develop as early as 24 hours after MI but is usually seen 2 to 5 days after MI. The diagnosis should be suspected when a new pansystolic murmur develops, especially associated with worsening hemodynamic profile and biventricular dysfunction. The defect is usually located in the apical septum with AMI and in the basal posterior septum with IWMI (Figure 1). 2D-Echo with colour flow imaging is the test of choice for diagnosis of VSR.

Transesophageal echocardiography (TEE) in some cases may help and right heart catheterisation with oximetry demonstrates step up in oxygen saturation in the RV and pulmonary artery. Early surgical closure and revascularisation is the treatment of choice. The mortality rate for patients with VSR treated medically is 24% at 72 hours and 75% at 3 weeks. Cardiogenic shock and multisystem failure are associated with high surgical mortality. An IABP should be inserted as a bridge to surgery. After IABP insertion, vasodilators can be started with hemodynamic monitoring to decrease left to right shunt and increase systemic flow. Emerging data suggest that percutaneous closure with paediatric VSD occluder device may be useful for high risk surgical patients and for whom surgical closure has failed.8

4. Mitral Regurgitation (MR)

MR of mild to moderate severity occurs in 13% to 45% of patients with AMI and is associated with increased mortality. Multiple mechanisms account for MR which include mitral annular dilation as a result of LV dilation; papillary muscle dysfunction and partial or complete rupture of the chordae or papillary muscle. Most MR is transient, asymptomatic, and benign. However, severe MR caused by papillary muscle dysfunction is a life threatening complication of AMI. Papillary muscles rupture (PMR) accounts for 7% of the cases of cardiogenic shock and 5% of mortality after AMI. The overall incidence is 1%. PMR usually involves the posteromedial papillary muscle as it is solely supplied by the right coronary artery (RCA). Patients may develop severe respiratory distress due to pulmonary oedema. A new pansystolic murmur audible at the cardiac apex with radiation to the axilla or the base of the heart suggests an acute MR. 2D-Echo with Doppler and colorflow imaging is the diagnostic modality of choice. TEE and Pulmonary artery catheterisation may also be helpful in quantifying MR. Priority of therapy is aggressive medical therapy with vasodilators and emergency surgical repair. Vasodilator therapy is very useful as it reduces SVR, regurgitant fraction, and increases stroke volume and CO. In patients with hypotension, IABP should be promptly inserted. PCI in patients with AMI and severe MR have shown improvement in hemodynamic values and reduction in MR. Surgical therapy (CABG + mitral valve replacement) should be considered immediately for patients with PMR. The prognosis is very poor among patients treated medically. Patients with moderate MR may do well with mitral valve repair.

5. Cardiac Free Wall Rupture

It accounts for approximately 10% of mortality after AMI. Free wall rupture (FWR) occurs in the first 5 days in 50% of patients and within 2 weeks in 90% of patients. With acute rupture, patients have electromechanical dissociation and sudden death. Some patients have a subacute course as a result of contained rupture. In subacute rupture, patients have distended jugular veins, pulsus paradoxus, diminished heart sounds, and a pericardial rub. A new to and fro murmur may be heard. In acute rupture, there may be no time for diagnostic testing.
In subacute rupture, 2D-Echo reveals findings of cardiac tamponade: RA systolic collapse, dilated inferior vena cava, and marked respiratory variation in mitral (>25%) and tricuspid (>40%) inflow (Figure 2). Reperfusion therapy has reduced the overall incidence of cardiac rupture after AMI. Immediate pericardiocentesis should be performed as soon as the diagnosis is made while arrangements are being made for transport to the operating room. Emergency thoracotomy with surgical repair is the definitive therapy for patients with cardiac rupture (Figure 3).

Pseudoaneurysm is caused by a contained rupture of the LV free wall. They may remain small or undergo progressive enlargement and the outer wall is formed by the pericardium and mural thrombus. Pseudoaneuerysms communicate with the body of the LV through a narrow neck, the diameter of which is <50% of the diameter of the fundus. Chest x-ray may show cardiomegaly with an abnormal bulge on the cardiac border and ECG may reveal a persistent ST elevation. 2D-Echo, MRI, CT-Scan may help confirm the diagnosis. Spontaneous rupture may occur in one third of the patients. Surgical resection is recommended for patients regardless of the size of the pseudoaneurysm.

7. True Ventricular Aneurysm

It can be acute or chronic. Acute development of a large LV aneurysm can result in CHF and even cardiogenic shock. They usually occur in transmural MI involving the apex of the LV. Chronic aneurysms which persist more than 6 weeks after MI, occur in 10% to 30% of patients with AWMI and such patients may have heart failure, ventricular arrhythmias, and systemic embolism but may be asymptomatic. An ECG reveals a persistent ST elevation and 2D-Echo helps accurately depict the aneurysmal segment and mural thrombus as well. 2D-Echo helps differentiate between a true aneurysm which has a wide neck and a pseudoaneurysm which has a narrow neck in relation to the diameter of the aneurysm (Figure 4).

Heart failure with acute aneurysms is managed with intravenous vasodilators and an IABP. Heart failure with chronic aneurysm should be managed with ACE inhibitors, digoxin and diuretics. ACE inhibitors have been shown to reduce infarct expansion and progressive remodelling and should be started within the first 24 hours of an AMI. Anticoagulation with warfarin sodium is indicated for patients with a mural thrombus with a target international normalized ratio (INR) of 2 to 3 for 3 to 6 months. PCI has beneficial effects on LV remodelling that are independent of myocardial salvage such as decreased 1-year mortality, LV dilation, and expansion. Implantable cardioverter-defibrillator (ICD) is indicated in patients with chronic aneurysm and intractable ventricular arrhythmias refractory to medications. Those with refractory heart
failure and refractory ventricular arrhythmias should be considered for surgical resection (Dorr’s procedure). Revascularisation is beneficial to patients with a large amount of viable myocardium in the aneurysmal segment.9

8. Dynamic Left Ventricular Outflowtract (LVOT) Obstruction

Dynamic LVOT obstruction is an uncommon complication of AMI and occurs due to hyperkinesis of basal and mid segments of the LV which decrease the cross sectional area of the LVOT. The resulting increased velocity of blood through the outflow tract decreases pressure below the mitral valve and result in the leaflet being drawn anteriorly towards the septum (venturi effect) which further increases the LVOT obstruction and MR. 2D-Echo helps evaluate the hyperkinetic segments, the LVOT obstruction, and the presence of systolic anterior motion (SAM) of the mitral leaflet. Medical treatment centers on decreasing myocardial contractility and heart rate with beta blockers while expanding intravascular volume with several small boluses of normal saline to increase preload and decrease LVOT obstruction and SAM.

B. Arrhythmic Complications

The incidence of arrhythmias is higher in patients the earlier they are seen after the onset of symptoms. Major mechanism of arrhythmias is re-entry caused by electrical inhomogeneity of the ischemic myocardium. Reperfusion arrhythmias occur due to washout of various ions such as lactate, potassium, and toxic metabolic substances that have accumulated in the ischemic zone. Arrhythmias have an adverse hemodynamic consequence as patients with LV dysfunction have a relatively fixed stroke volume and depend on changes in heart rate to alter CO. Tachyarrhythmias after AMI include ventricular fibrillation (VF), ventricular tachycardia (VT), and atrial fibrillation or flutter. In general, ventricular and atrial arrhythmias in the setting of AMI are treated according to the advanced cardiac life support (ACLS) guidelines. It is reasonable to manage refractory arrhythmias by aggressive attempts to reduce myocardial ischemia and adrenergic stimulation with beta-blockers, IABP use, emergency PCI-CABG surgery and maintaining serum potassium levels ≥4.5 mEq/liter and serum magnesium level ≥2 mEq/liter.10 Routine use of prophylactic anti-arrhythmic drugs is not indicated for suppression of isolated premature ventricular beats (PVC), couplets, runs of accelerated VT and nonsustained VT.

Accelerated idioventricular rhythm (AIVR) occurs in up to 20% of AMI patients and is often observed shortly after successful reperfusion. AIVR is thought not to affect prognosis, and is not routinely treated. Nonsustained VT (NSVT) occurring early after presentation does not appear to be associated with an increased mortality risk.

VT occurring late in the course of AMI is more common in patients with transmural infarction and LV dysfunction and more likely to be sustained and causes marked hemodynamic deterioration, with increased rate of hospital and long term mortality. Rapid abolition of sustained VT in AMI patients (cardioversion/defibrillation) is mandatory as it reduces the LV function and frequently deteriorates into VF.10 Patients with recurrent or refractory VT should be considered for specialised procedures such as implantation of AICD or surgery. Urgent attempts at revascularisation (angioplasty or CABG) can help control refractory VT.

Ventricular fibrillation (VF) can occur in 3 settings in hospitalized patients with AMI. Primary VF occurs suddenly and unexpectedly in patients with no signs or symptoms of LVF. Secondary VF is often the final event of a progressive downhill course with LV Failure and cardiogenic shock. Late VF develops more than 48 hours after AMI and mostly occurs in patients with large infarcts and LV dysfunction. Treatment includes unsynchronised electrical countershock with at least 200 to 300 joules as rapidly as possible. Intravenous amiodarone can be used for successful interruption of recurrent episodes. When synchronous cardiac electrical activity is restored by countershock but contraction is ineffective (i.e., pulseless electrical activity), the usual cause is extensive myocardial ischemia or necrosis or rupture of the ventricular free wall or septum.

The CAST Trial (Cardiac arrhythmia suppression trial) and SWORD trial (survival with Oral D-Sotalol) were both discontinued prematurely due to increased mortality observed in the active treatment group.11,12 The CAMIAT (Canadian Amiodarone Myocardial Infarction trial) and EMIAT (European Amiodarone Myocardial Infarction trial) showed a reduction in arrhythmic deaths but no reduction in total mortality.13,14 Several trials that included post AMI patients in the study population have shown significant mortality reductions in patients randomized to ICD implantation versus conventional medical therapy. At present, prophylactic implantation of an ICD after AMI is indicated in patients with LVEF <35% and at least 40 days post-AMI with New York Heart Association (NYHA) class II or III symptoms.15

Supraventricular Tachy Arrhythmias

Sinus tachycardia occurs due to anxiety, persistent pain, LV failure, fever, pericarditis, hypovolemia, pulmonary embolism, and the administration of cardioaccelerator drugs such as atropine, epinephrine, or dopamine. It results in augmentation of myocardial oxygen consumption, and reduction in the duration of coronary perfusion thereby, intensifying myocardial ischemia. Persistent sinus tachycardia can signify persistent heart failure and connotes poor prognosis and excess mortality. Treatment includes analgesics for pain, diuretics for heart failure, beta blockers, and nitroglycerine for ischemia, and aspirin for fever or pericarditis.

Atrial flutter (AFL) and Atrial fibrillation (AF) is usually transient and is a consequence of augmented sympathetic stimulation of the atria, LV failure, pericarditis and ischaemic injury to the atria and RVMI. The increased ventricular rate and loss of the left atrial (LA) contribution to LV filling result in a significant reduction in CO. AF is associated with increased mortality and stroke, particularly in patients with AWMI. Acute management of AF involves rate control with intravenous diltiazem or esmolol. If the patient is hemodynamically unstable, immediate transthoracic cardioversion may be appropriate.16 If the AF has been present for more than 48 hours or if the duration is unclear, cardioversion ideally should be preceded by TEE to rule out a LA thrombus. If the patient is hemodynamically stable, cardioversion can be done pharmacologically
Second degree AV block: Type I second-degree AV block does generally not require specific treatment. Beta blockers and calcium antagonists (other than nifedipine) prolong AV conduction and discontinuation of these drugs in the setting of AMI increases ischemic injury. If the block is associated with sinus bradycardia and hypotension, atropine can be administered. Continued electrocardiographic monitoring is important in view of the possibility of progression to higher degrees of block.

Second degree AV block: Type I second-degree AV block does not affect survival and is most commonly associated with occlusion of the RCA and is caused by AV node ischemia. Specific therapy with atropine is required if the patient becomes symptomatic, ventricular rate falls < 50bpm, PVCs and Bundle branch blocks (BBB) develop or patient develops heart failure. Temporary pacing is almost never needed.

Type II second-degree block usually originates from a lesion in the conduction system below the His bundle. Because of its potential for progression to complete heart block, type II second-degree AV block should be treated with a temporary external or transvenous temporary pacemaker. Complete (third degree) AV block (CHB) can occur in patients with AWMI and IWMI. CHB in patients with IWMI results from an intranodal or supranodal lesion and is usually transient. The escape rhythm is usually stable, with a rate exceeding 40bpm and a narrow QRS complex and pacing is not usually necessary unless the patient has slow ventricular rate (<40 to 50 bpm), ventricular arrhythmias, hypotension or pump failure. Permanent pacemaker is almost never indicated.

In patients with AWMI, the CHB often occurs suddenly after AMI and is usually preceded by intraventricular block and often type II AV block. Such patients have unstable escape rhythms with wide QRS complexes and rates less than 40 bpm and ventricular asystole. The AV block usually develops as a result of extensive septal necrosis that involves the bundle branches. In AWMI with CHB, temporary pacing protects against transient hypotension and asystole but has no shown survival benefit.

Intraventricular Block: Pre-existing bundle branch blocks (BBB) or divisional blocks is less commonly associated with development of CHB in patients with STEMI. Isolated fascicular blocks are less likely to progress to complete AV block. Right bundle branch block (RBBB) alone can progress to AV block because it is often a new lesion, associated with anterosetal infarction. Isolated RBBB is associated with an increased mortality risk in patients with AWMI. Bifascicular block, i.e. RBBB with left anterior or posterior fascicular block, or with left BBB, in the presence of first degree AV block is associated with high risk of developing CHB, severe pump failure and high mortality and temporary pacing is advisable. Permanent pacemaker insertion may be required if CHB persists throughout the hospital stay in a patient with AMI, when sinus node function is markedly impaired, or when type II second or third degree block occurs intermittently.

C. Ischemic Complications

It is a critical task for clinicians to distinguish postinfarction angina or recurrent angina from nonischemic causes such as pericarditis, pulmonary embolism and noncardiac cases. Recurrent postinfarction angina may be due to either an infarct extension or reinfarction in a separate territory or reocclusion of the infarction related artery. Within the first 18 to 24 hours following AMI, recurrent infarction should be strongly suspected when there is a repeat ST-segment elevation on the ECG. Beyond the first 24 hours, recurrent infarction can be diagnosed by the re-elevation of the cardiac markers or the presence of new Q waves on the ECG. Pericarditis is an inflammatory condition of the pericardium caused by an infective process, an autoimmune reaction or as a result of myocardial infarction. Postinfarction angina is important because it increases morbidity and mortality. If the 12-lead ECG reveals ST segment re-elevation, urgent catheterisation and PCI is recommended and if PCI is unavailable, repeat fibrinolysis can be considered. Hemodynamically stable symptomatic patients are treated with nitroglycerine and beta blockade. When hypotension, heart failure or ventricular arrhythmias develop during recurrent ischemia, urgent catheterisation and revascularisation are indicated. With increasing use of PCI in the management of patients with AMI, stent thrombosis may be a cause of recurrent ischemia.

D. Inflammatory

Pericarditis results from an area of localized pericardial inflammation overlying the infarcted myocardium and the inflammation is fibrosus in nature. Pericarditis after MI is classified as early pericarditis or late pericarditis. Early pericarditis complicates AMI in approximately 10% of patients and the inflammation usually develops 24 to 96 hours after MI. Patients report progressive severe chest pain that lasts for hours. The pain is postural; worse when supine and alleviated when sitting up and leaning forward. Radiation of pain to the trapezius ridge is nearly pathognomonic for acute pericarditis. A transient pericardial friction rub may be heard and correlates with a larger infarct and hemodynamic derangements. ECG shows ST elevation with concave upward or saddle-shaped curve and the changes are generalised and not limited to a particular territory (Figure 5). 2D-Echo may reveal a pericardial effusion. Treatment of pericarditis consists of aspirin in doses of 650mg every 4 to 6 hours. Colchicine may be used in those with recurrent pericarditis.

Late pericarditis (Dressler’s syndrome) occurs 1 to 8 weeks after AMI. Clinically, these patients present with pericardial chest discomfort, malaise, fever, leukocytosis, raised ESR and 2D-Echo may reveal pericardial effusion. Treatment is with aspirin, however, if >4 weeks have elapsed since AMI, NSAIDs and even steroids may be used for severe symptoms.
The incidence of clinically evident systemic embolism after MI is approximately 2%, mural thrombus around 5% (Figure 6) and the incidence is higher among patients with AWMI. Systemic embolism most often results in a stroke, although patients may have limb ischemia, renal infarction and mesenteric ischemia. Most episodes of systemic emboli occur in the first 10 days after AMI. Treatment involves use of intravenous heparin for 3 to 4 days to elevate aPTT to 1.5 to 2 times that of control followed by oral anticoagulation for 3 to 6 months for patients with mural thrombus and those with large akinetic areas detected by 2D-Echo.17

**Conclusion**

AMI may be complicated by a number of pathophysiologic mechanisms. The onset of each of these complications usually results in explicit symptoms and physical manifestations. Thus, a basic knowledge of the complications that occur in the postinfarction period, and the clinical syndrome associated with each, will allow the physician to evaluate and treat the complication in a confident and timely manner.

**References**


