Healthy Heart

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Dr. Hemang Baxi

From the Desk of Hon. Editor:

Dear Friends,

Warm Greetings & Happy Diwali in advance. It is a great please to interact with you periodically and share scientific knowledge.

In this issue, I have tried to summerise Complications of Acute Myocardial Infarction (AMI). Although the incidence of various post MI complications have decreased significantly with the advent of newer pharmacological agents, early reperfusion techniques (Pharmacological or mechanical), prognosis remains dismal for several important post-AMI complications.

We will be discussing various clinical aspects & management issues of all these complications.

With warm regards

- Dr. Hemang Baxi

Complications of Acute Myocardial Infarction

Introduction

Each year, approximately ten million The patients in India suffer an AMI, leading directly to 8,00,000 deaths. Despite new pharmacologic agents, the emergence of reperfusion therapy, and the growing acceptance of early revascularization, prognosis remains dismal for several important post-AMI complications. Thus, to achieve the best possible outcomes, cardiology providers need to be able to recognize, diagnose, and treat AMI complication, including : 1) cardiogenic shock, 2) right ventricular (RV) infarction/ischemia, 3) ischemic mitral valve regurgitation (MR),4) ventricular septal defect (VSD), and 5) left ventricular (LV) free wall rupture.

Cardiogenic shock

Incidence

Cardiogenic shock remains the leading cause of death for patient hospitalized with AMI, and complicates 6-7 % of acute ST-elevation and non-ST-elevation MIs (NSTEMIs). In the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occuded Coronary Arteries (GUSTO) IIb trial, 4.2 % of ST-segment elevation patients developed cardiogenic shock compared to only 2.5 % of patients with non-ST-segment elevation; however, mortality secondary to cardiogenic shock is similarly high among patients with and without ST-segment elevation.

Severe dysfunction of the LV due to infarction and ischemia is the most common cause of cardiogenic shock. However, it can also be precipitated by other complications of AMI such as RV dysfunction, acute MR, VSD, or cardiac free wall rupture.

Clinical presentation

In addition to the MI symptoms, patients with cardiogenic shock will develop respiratory distress and signs of hypoperfusion including cool, clammy extremities, oliguria, and mental status changes from cerebral hypoperfusion. Physical exam reveals hypotension, rales, S3 gallop, and cold extremities. Hemodynamically, the systemic systolic pressure is lower than 90 mm Hg, the cardiac index is lower than 1.8 L/min/m2, and systemic vascular resistance and pulmonary artery wedge pressure are elevated. Laboratory data reveal lactic acidosis, elevated blood urea nitrogen/creatinine, and arterial

hypoxemia. Chest X-ray reveals pulmonary edema.

Diagnosis

In the Should we emergently revascularize occluded coronaries for cardiogenic shock (SHOCK) trial, the diagnosis of cardiogenic shock was confirmed by both clinical and hemodynamic criteria. The clinical criteria were:

- Hypotension (systolic blood pressure <90 mm Hg for at least 30 minutes)
- End-organ hypoperfusion (cool extremities or a urine output < 30 mL/h, and a heart rate > 60 beats/min)

The hemodynamic criteria were :

- Cardiac index < 2.2 L/min per square meter of body-surface area
- Pulmonary-capillary wedge pressure of at least 15 mm Hg

The median time from MI to onset of cardiogenic shock in the SHOCK trial was 5.5 hours, with 75 % of patients developing shock within 24 hours.

Management

The goals of the therapy for cardiogenic shock included hemodynamic stabilization to ensure tissue perfusion and prompt assessment for reversible cause of cardiogenic shock. Intra-aortic balloon pump (IABP) counterpulsation should be considered. Positives inotropes (dobutamine and dopamine) should be added if necessary to improve cardiac output and hemodynamics. Diuretics should be used to treat pulmonary congestion and enhance oxygenation.

Reperfusion therapy is likely to improves post-AMI cardiogenic shock mortality. This has been suggested by GUSTO-1 trial subgroup analysis, which studied 1,320 patients with shock. Reperfusion therapy was associated with reduced in-hospital (50 %) and one year mortality. The SHOCK trial is the largest and most important randomized trial evaluating early revascularization in patients with cardiogenic shock. Patients with shock due to LV failure complicating MI were randomized to emergency revascularization or initial medical stabilization.

Revascularization was accomplished by either coronary artery bypass grafting (CABG) or angioplasty. The primary endopoint was morality from all causes at 30 days, and the secondary endpoint was six-month survival. Revascularization was associated with a nonsignificant mortality reduction at 30 days (absolute risk reduction 9 %. relative risk reduction 17 %); however, the six-month mortality rate was significantly reduced (50.3 % vs 63.1 %, P = 0.027). Subgroup analysis suggested that patients younger than age 75 benefited proportionally more from early reperfusion strategy.

RV infarction /ischemia

Incidence

Approximately 50 % of patients who develop an inferior MI will also experience a RV infarction/ischemia, although only about 10 % of those infarcts will be clinically and hemodynamically significant.

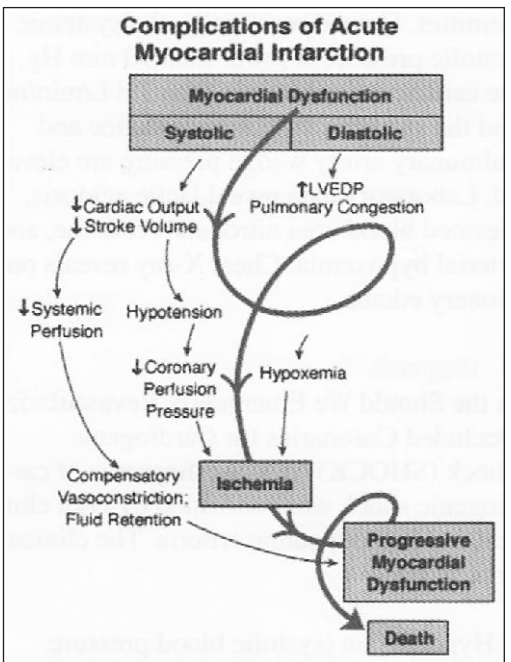

Figure-1 : The downward spiral in cardiogenic shock

Clinical Presentation

The clinical triad of hypotension, clear lung fields, and elevated jugular venous pressure (JVP) in a patient with an inferior infarction is virtually pathognomonic for RV infarction/ischemia. Although quite specific, this triad has a sensitivity of <25%. Physical exam reveals elevated JVP and RV S3 with clear lungs (in patients without concomitant LV dysfunction). Kussmaul's sign (inspiratory increase in JVP) can also be seen. The predominant atrial descent is systolic (x descent) and the diastolic (y) descent is blunted, reflecting an increased resistance to diastolic filling.

Diagnosis

ECG : Lead V4R is the single best lead for diagnosis, 1 mm ST elevation in lead V4R is approximately 70% sensitive and 100%

specific for acute RV infarction. Thus, right-sided ECGs should be done in all patients with acute inferior infarction.

Echocardiography is the study of choice for RV infarction. It provides information about the size and function of the RV.

Pulmonary artery catheterization reveals elevated RA and RV pressure and low pulmonary capillary wedge pressure (unless there is concomitant LV failure).

Management

Optimal management of RV involvement in AMI or cardiogenic shock requires early recognition. Once suspected, the treatment should be directed at early maintenance of RV preload, reduction of RV afterload, inotropic support of the dysfunctional RV, and early reperfusion. Intravenous fluids (usually normal saline) should be given to enhance the RV and eventually the LV filling pressure. Agents that decrease the RV preload such as nitrates, diuretics and morphine may worsen hemodynamic condition and should be restricted. Inotropes can be used in cases where fluid resuscitation is not sufficient to improve the hemodynamic status of the patient. Maintenance of atrioventricular synchrony is also important to optimize RV filling. Most importantly, patients with RV involvement in either LV or RV shock can benefit as much from early revascularization as other patients with cardiogenic shock. Patients who undergo successful reperfusion have improved RV function reduced 30-day mortality.

Acute mitral valve regurgitation

MR following AMI results from regional or global LV ischemia, papillary muscle dysfunction or papillary muscle rupture. Mitral regurgitation is fairly common following AMI and will persist in 15% cases. Papillary muscle rupture causing MR occurs much less frequently and complicates approximately 2 % of AMIs, often leading to cardiogenic shock and worse outcome. In the SHOCK trail registry, acute severe MR was the cause of shock.

Clinical manifestations

The diagnosis of ischemia-induced acute MR and papillary muscle rupture is often difficult. Severe MR and heart failure symtoms may develop following a small or moderate-sized AMI. Most patients develop acute hypotension and flash pulmonary edema with a new holosystolic murmur. Although the murmur is often loud, a palpable thrill is generally not present (in contrast to VSD). The intensity of the murmur frequently does not correlate with the severity of the regurgitant flow. If the LV function is severly impaired or if the left atrial pressure is very high, the murmur may be of low intensity or even absent. If a pulmonary artery catheter is already in place, the pulmonary capillary pressure tracing usually shows giant V waves secondary to decreased left atrial compliance in the setting of acute regurgitaion. This phenomenon is also seen in acute VSD.

Diagnosis

The diagnosis is uaually suggested by the combination of hemodynamic compromise and a new systolic murmur in the setting of an AMI. the main differential diagnosis is postinfarction VSD.

Echocardiography with color flow Doppler is valuable to confirm the diagnosis and mechanism of ischemia-induced acute MR.

Treatment

Emergent arterial vasodilators such as nitroprusside may improve the hemodynamic status temporarily because it decreases systemic vascular resistance. This reduces the regurgitant fraction and increases the forward stroke volume and cardiac output. Most cases of ischemia-induced MR resolve or improve without surgery following reprefusion/revascularization and treatment of the underlying ischemia/infarction. Surgery is usually required in presence of papillary muscle rupture, much less commonly in the absence of rupture. Emergent surgical intervention remains the treatment of choice for papillary muscle rupture. Observational data suggest that surgery for acute MR should be performed acutely, even in patients who appear to stabilize with medical therapy, because its unpredictable. The perioperative mortality associated with postinfarct papillary muscle rupture is high (27%), but two-thirds of the survivors are alive at seven years. The only factor that improved both immediate and long-term

survival was the concomitant performance of CABG.

Rupture of the interventricular septum (Post AMI VSD)

Incidence

Rupture of the interventricular septum occurred in about 2 % of all acute infarctions in the prethrombolytic era. This figure has decreased to 0.2 % in patients treated with reperfusion therapy. It may develop as early as 24 hours after MI, but typically occurs 3-5 days after the acute event. In the GUSTO-I trail, VSD was associated with a very high 30-day mortality (74 % vs. 7 % for patients with and without VSD)

Clinical manifestations

Patients with a ruptured septum usually present with hypotension, congestive heart failure (CHF), and a new murmur. The murmur is harsh, loud, and holosystolic, best heard at the lower left sternal borders and accompanied by a palpable thrill in 50% of cases. VSD can present as cardiogenic shock.

Diagnosis

Two-dimensional TTE with color flow imaging is the recommended test to diagnose post –AMI VSD.

Management

The key to the management of postinfarction VSD is early recognition and early surgical intervention. Unless there is significant aortic regurgitation, an IABP should be inserted emergently as bridge to a surgical procedure. Early

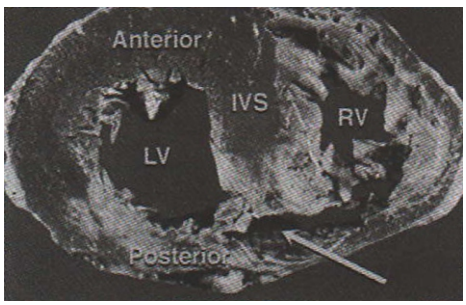

Figure-2 : Posterior ventricular septal rupture (VSD)

surgical closure is recommended and likely yields better results than attempting to wait for days, weeks until the conditions for surgery improve. In the SHOCK trial registry, surgical repair was performed on 31 patients with rupture, of whom six (19 %) survived. Of the 24 managed medically, only one survived. In the GUSTO-I trial, surgical repair was performed in 40 percent of patients with a VSD within a median of 3.5 days after onset of infarction symptoms. The 30-day and one-year survival for those undergoing surgery was 53 % and 47 %, respectively, while it was only 6 % and 3 % for those treated medically.

In summary, postinfarct VSD carries a very high mortality, and the key to the management of these patients is rapid stabilization and prompt surgry.

Rupture of the LV free wall

Incidence

Rupture of the free ventricular wall is almost always fatal. Fortunately, reperfusion therapy has decreased the incidence of free wall rupture (0.2 %). More than one-half of myocardial rupture occurs within the first five days after MI. Independent predictors of rupture was anterior location of the infarction, age>70, and female sex.

Clinical presentation

The most common presentation of myocardial rupture is cardiac arrest leading to sudden death. Electromechanical dissociation is usually the underlying cardiac rhythm. Premonitory chest discomfort and intermittent brady-cardia signal impending rupture in most cases. Complete rupture of the LV free wall usually leads to hemopericardium and death from acute cardiac tamponade.

Diagnosis

High clinical suspicion and immediate echocardiography during resuscitation with demonstration of hemopericardium and cardiac tamponade is essential for diagnosis.

Treatment

Suivival of AMI patients with LV free wall rupture and cardiogenic shock is dismal. Very few patients can be resuscitated or temporarily stabilized in order to be considered for surgical repair. Aggressive volume replacement with blood transfusion and intravenous fluids, pressor agents, and emergent pericardiocentesis may be considered if LV rupture is suspected or diagnosed. Still, medical therapy has no role in the treatment for acute LV wall rupture. The only hope for these patients is emergency surgical repair, thus emphasizing the importance of early recognition diagnosis and surgical triage.

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INTERESTING FACTS ABOUT HEART TRANSPLANT

Who can get a new heart?

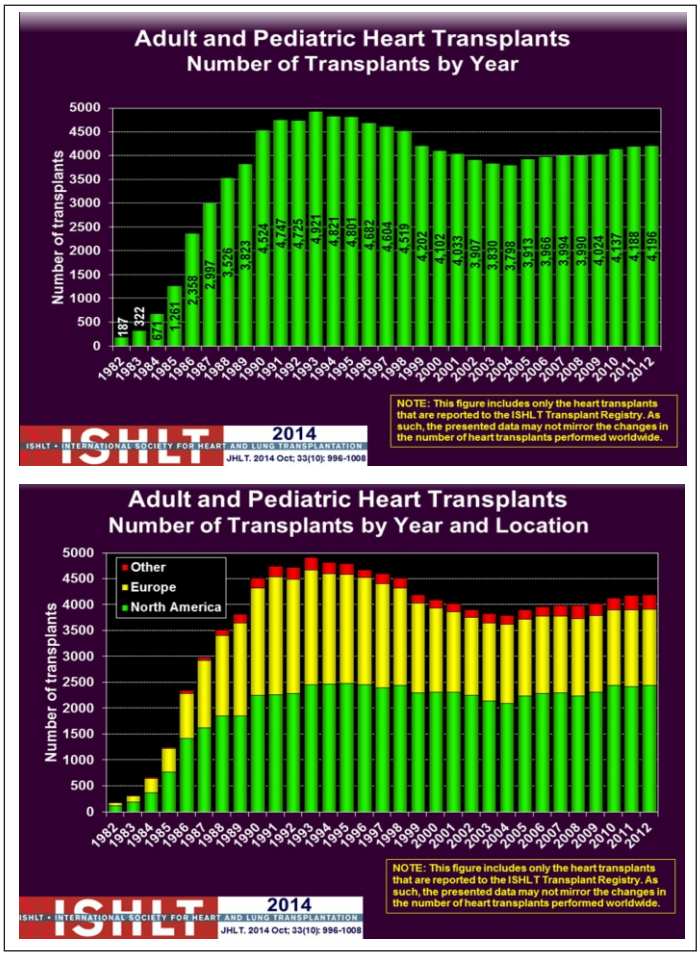
- These are not absolute guidelines, since each transplant center is allowed to set its own rules for who is eligible.
- The patient must be less than 69 years old when put on the waiting list.
- The patient must show no evidence of active infections or cancer at time of transplant.
- The patient must show no evidence of disease affecting arterial circulation to your brain or legs (significant underlying disease in major arteries lowers chances of long-term survival after transplant).
- Results of the patient's physical evaluation tests must be considered adequate and must be psychologically "suitable"
- The patient must fully understand the risks and requirements for taking medications.
- The patient must be committed to actively participating in the rehabilitation process after transplant.
- The patient must not have smoked or used alcohol for at least 3 months before being put on the transplant waiting list, and must be trusted not to smoke or drink afterwards.
- The patient cannot be overweight.
- The patient must not have dental treatment in progress.

How does a donor heart get to the patient?

- A potential donor who has been confirmed brain dead must be identified.
- Next of kin is told of the chance to donate their relative's organs and tissues. They must give their permission.
- An OPO - Organ Procurement Organization - is called to decide if any of the donor's organs are acceptable. If so, they get the family's official permission and attempt to match the donor with the most suitable recipient(s).
- The organs are surgically removed from the donor.
- The donor organs are taken to the transplant center where the surgery will be done.
- The longest the patient has survived after heart transplant as of 2002 is 24 years.

Others

- Heart transplant survival rates are now 84.8% at one year, 77.1% at 3 years, and roughly 50% at 10 years after transplant surgery.
- The number of patients (65 years and older) who receive heart transplants is increasing faster than any other age group. From 1996 to 1999, heart transplants in the 65-plus age bracket rose by 28%.
- Acute heart rejection is more likely to happen when the heart donor was female regardless of the recipient sex.
- The signs of beginning artery blockages are seen in almost half of patients after one year, using sensitive tests like multi-vessel IVUS imaging.
- Heart failure is the leading cause of death in most of the developed world. About 730,000 Americans die each year from it.



- In 1998, only 2,345 heart transplants were done in USA.
- Last year (2014) in India we crossed figure of 50 transplants in one year.
- Average waiting time to transplant is more than 3-7 months. In some parts of the country, as many as 40% of patients die while waiting.
- Failure to take post-transplant meds properly is the third leading cause of transplant failure!
- Pharmaceutical companies tell many of their employees to bring back samples of dirt from any foreign places they visit on vacations. That's because cyclosporine and some other very valuable drugs come from fungus found in dirt from different parts of the world.
- As of January, 2000, there were 141 heart transplant programs operating in USA.
- A donor family's financial responsibility ends when the The patient is declared dead. After that, the hospital does not bill the family for any charges. The Organ Procurement Organization (OPO) is billed for those charges.
- CAD (Coronary Artery Disease) is the leading cause of death for heart transplant recipients who have survived at least one year after surgery.

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