Abstract : Ventricular septal rupture (VSR) following acute myocardial infarction (AMI) is a rare complication. It produces progressive hemodynamic instability which may be fatal. This is a case study of a patient who had developed VSR following anterior wall MI post thrombolysis, With a typical natural history.

Keyword : Ventricular septal rupture, Acute myocardial infarction, Echocardiography, Intra-Aortic balloon pump

VENTRICULAR SEPTAL RUPTURE AFTER ACUTE MYOCARDIAL INFARCTION

55 year old lady was admitted in ICCU of TVMCH on 2/11/10- 2.15pm. The history revealed that she was a known hypertensive on regular treatment ,was admitted with sudden onset of chest pain since 4 am, i.e for 10 hours. Chest pain was retrosternal with radiation to back which was associated with sweating and vomiting. Physical examination revealed that she was dyspnoeic with pulse rate of 80 bpm – regular, normal in volume and character, felt in all peripheral blood vessels. Blood pressure-170/100 in right upper limb in supine position. Cardiovascular system examination revealed normal heart sounds without any murmur. There was fine inspiratory crackles in the base of the lungs. There was no dependent oedema or elevated jugular venous pulse. ECG revealed extensive anterior wall ST elevation MI. Troponin T was positive. Transthoracic echocardiography revealed hypokinesia of interventricular septum and left ventricular apex with normal valves and mild LV systolic dysfunction with ejection fraction of 51%. No other abnormality was revealed.
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She was successfully thrombolysed with streptokinase after giving loading dose of Aspirin,Clopidogrel and Atrovastatin. She also received Enalapril and Metoprolol. Post thrombolysis BP was 140/80. She was given heparin after 6 hours.The 2nd day, 3/11/10 8 am She was complaining of chest pain which was prickling type in theretrosternal region. On physical examination she was found to be drowsy and dyspnoeic. There was no dependent oedema or elevated JVP. Her pulse rate was 84bpm and blood pressure was 140/90. Cardiovascular system examination revealed a harsh pansystolic murmur in the lower left sternal border. Respiratory system examination was normal. Echocardiography on 3/11/10 at 11am showed- Normal chambers and normal valves. Ventricular septal defect, which is serpegenious extending from lower 2/3rd of IVS to LV apex with Left to Right Shunt is present. Hypokinetic contraction of remaining part of IVS and LV apex suggestive of VSR. Mild LV systolic dysfunction with 2.6/24- Gradient across VSD. LV ejection fraction was 49 %. No clot or pericardial effusion.

Fig-1, ECG
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Fig-2, Echocardiography of VSR
Closure of the VSR was not undertaken as the patient’s family were not willing to transfer her to a cardiovascular surgery center. Therefore, she received only supportive medical treatment. The hemodynamic status worsened progressively requiring inotropic support with dopamine and dobutamine. Repeated echocardiography examinations during the following days showed the VSR with increasing gradient. 3rd day, patients condition worsened. She was drowsy and dyspnoeic with a pulse rate of 98bpm. Her peripheries were cold and clammy. Her blood pressure was
90/60. With same findings in cardiovascular respiratory system as previous day. She was put on inotropic agents. Her condition continued to worsen with hypotension and pulmonary oedema. On 4th day she eventually succumbed to her illness.

Discussion

VSR is a rare but serious complication of acute myocardial infarction that is, in almost all cases, fatal without early surgical intervention. It had an incidence of 1-3% in the era before reperfusion therapy, decreasing with the introduction of thrombolytic therapy. VSR is less frequent than a rupture of the ventricular free wall. VSR complicates 1-2% of cases of acute myocardial infarction. First successful correction reported by Cooley in 1957. High mortality despite various improvements in therapy with 30 day mortality-74% and 1 year mortality-78%. Relative Improvement in survival due to earlier diagnosis, earlier flow restoration and more aggressive surgical intervention. The predictors of VSR are advanced age, anterior location of infarction, female sex, non smokers and thrombolysis after 12 hours also suggested as a predisposing factor. Ventricular septal rupture is equally common after anterior and inferior myocardial infarctions. Infarct expansion and extensive myocardial thinning may lead to intolerable wall stress, particularly at areas with marked distortions in normal geometry, as in this patient. The average time to development of a ventricular septal rupture is 2-4 days. It can range from few hours to 2 weeks. Time course may be accelerated by thrombolysis, possible related to intramyocardial hemorrhage. The defect may range from very small to a 1-cm hole in the interventricular septum. The ventricular septal defect may take a winding course through the necrotic myocardium, leading to an exit into the right ventricle far displaced from the left ventricular site of the defect. Clinical presentation The diagnosis should be suspected clinically when a new pansystolic murmur which is loud/harsh pansystolic murmur, within the first week post AMI. The murmur is best heard at left lower sternal border, less loud at the apex associated with a palpable thrill. Depending on the location, may radiate to the axilla mimicking MR. Up to 50% of patients experience chest pain associated with the development of murmur. CHF and shock are often associated with the development of murmur

Coronary anatomy and VSR Post MI VSRs more commonly associated with 100% occlusion of the infarct related artery. There are mainly two types of VSR - VSD Simple: through and through defect usually located anteriorly Complex: serpiginous dissection tract remote from the primary septal defect most commonly an inferior VSD.

Diagnosis Echocardiography with color flow Doppler imaging is the diagnostic tool of choice for identifying a VSR. Its sensitivity and specificity have been reported to be as high as 100%. It can also be used to define the site and size of septal rupture, assess the LV and RV function, estimate the RV systolic pressure, and quantify the left-to-right shunt. Cardiac catheterization is usually required to confirm the diagnosis, quantify the degree of left-to-right shunt, differentiate VSR from other conditions, such as acute MR, plus visualize the coronary arteries. In patients with VSR, right-heart catheterization shows a step-up in oxygen saturation from the right atrium to the RV, in contrast no step-up in oxygen saturation among patients with MR.

Management As with other cardiac ruptures, surgical management is advocated, although the outcome is
not as gratifying as in acute mitral regurgitation, because the extent of myocardial necrosis is generally larger.\textsuperscript{1,5}

The key to management of VSR is prompt diagnosis and an aggressive approach to hemodynamic stabilization, angiography, and surgery. The optimal approach includes hemodynamic stabilization with the administration of oxygen and mechanical support with use of an intra-aortic balloon pump, as well as the administration of vasodilators (to reduce afterload and thus LV pressure and the left-to-right shunt), diuretics, and inotropic agents. Cardiac catheterization is needed to define the coronary anatomy; this is followed by urgent surgical repair.

Medical therapy is intended only for temporary stabilization before surgery, as most patients' conditions deteriorate rapidly and they die in the absence of surgical intervention.

In the GUSTO-I trial, the 30-day mortality rate was lower in patients with VSR who underwent surgical repair than in patients treated medically (47\% vs 94\%), as was the 1-year mortality rate (53\% vs 97\%).\textsuperscript{1} Lemery et al had reported that a 30-day survival rate of 24\% in patients treated medically compared with 47\% in those treated surgically.\textsuperscript{5}

Current guidelines by the American College of Cardiology/American Heart Association for the treatment of patients with septal rupture complicating AMI highlighted the urgent surgical intervention, regardless of the clinical status. Surgical management of septal rupture includes the following elements:

- Prompt establishment of hypothermic cardiopulmonary bypass
- An approach to the septal rupture via the infarct area and the excision of all necrotic, friable margins the geometric configuration of the ventricles and heart function of the septum and ventricular walls to avoid postoperative haemorrhage, residual defect, or both.

Reconstruction of the septum and ventricular walls by using prosthetic material and preservation of This case demonstrated typical risk factors for ventricular septal rupture – an elderly lady, who is a hypertensive, a non smoker with extensive anterior wall infarction, who had a septal rupture after 24 hours of infarction. She had natural course of illness who eventually succumbed to her disease.

BIBLIOGRAPHY:


