ORIGINAL RESEARCH PAPER

Cardiology

KEY WORDS:

A CASE OF VENTRICULAR SEPTAL RUPTURE AFTER ACUTE MYOCARDIAL INFARCTION

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INTRODUCTION –

When ventricular septal rupture (VSR) complicates acute myocardial infarction the mortality is usually high. Reperfusion therapy has reduced its incidence. However if VSR has developed then rapid diagnosis, aggressive medical management, and surgical intervention are required to optimize recovery and survival. In the era before reperfusion therapy, septal rupture complicated 1 to 3 percent of acute myocardial infarctions. Among the 41,021 patients in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) trial, ventricular septal rupture was suspected in 140 patients (0.34 percent) and confirmed by a retrospective review in 84 (0.2 percent). Thus, reperfusion therapy has decreased the incidence of septal rupture. Septal rupture occurs more frequently with anterior wall acute myocardial infarction than other types of acute myocardial infarction. Risk factors for septal rupture included hypertension, advanced age (60 to 69 years), female sex and the absence of a history of angina or myocardial infarction. Angina or previous infarction may lead to myocardial preconditioning as well as to the development of coronary collaterals, both of which reduce the likelihood of septal rupture. In patients undergoing thrombolysis advanced age, female sex, and the absence of smoking are often associated with an increased risk of septal rupture, whereas the absence of antecedent angina has not been associated with an increased risk. In the GUSTO-I trial, there was a nonlinear relation between the systolic and diastolic blood pressures at enrolment and septal rupture, since hypertension and extensive myocardial infarction and right ventricular infarction are also risk factors for septal rupture.

PATHOGENESIS -

The septum adjacent to the rupture is thin and necrotic. Without reperfusion, coagulation necrosis develops within the first three to five days after infarction with numerous neutrophils entering the necrotic zone. The neutrophils undergo apoptosis and release lytic enzymes causing the disintegration of necrotic myocardium. The pathogenic process of the rupture changes over time. In first 24 hours, coagulation necrosis is beginning and there are relatively few neutrophils within the infarcted tissue. Early ruptures occur in infarcts with large intramural hematomas that dissect into tissue and rupture. If patients survive for several weeks, the septum becomes fibrotic. Becker and van Mantgem classified the morphology of free-wall rupture into three types, which are also relevant to ventricular septal rupture:

Type I ruptures have an abrupt tear in the wall without thinning

Type II, the infarcted myocardium erodes before rupture occurs and is covered by a thrombus

Type III has marked thinning of the myocardium, secondary formation of an aneurysm, and perforation in the central portion of the aneurysm.

The size of septal rupture ranges from millimetres to several centimetres. Morphologically, septal rupture is categorized as simple or complex. Simple septal rupture - discrete defect and a direct through and through communication across the septum. The perforation is at the same level on both sides of the septum. Complex septal rupture - Extensive haemorrhage with irregular, serpiginous tracts within necrotic tissue. Septal ruptures in patients with anterior myocardial infarction are generally apical and simple. Conversely, in patients with inferior wall myocardial infarction, septal ruptures involve the basal infero-posterior septum and are often complex. Occasionally, muscles of the ventricular free wall or papillary muscles may tear especially in the case of complex septal ruptures. Ventricular septal ruptures associated with an inferior or anterior myocardial infarction generally involve right ventricular infarction.

TIME COURSE -

Without reperfusion, septal rupture generally occurs within the first week after infarction. There is a bimodal distribution of septal rupture, with a high incidence on the first day and on days 3 and rarely more than two weeks after infarction. The median time from the onset of symptoms of acute myocardial infarction to rupture is generally 24 hours or less in patients who are receiving thrombolysis. The median time from the onset of infarction to septal rupture was 1 day in the GUSTO-I trial and 16 hours in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) trial. Although thrombolytic therapy reduces the size of the infarct, it may in some cases promote haemorrhagic dissection in the myocardium, accelerating the onset of septal rupture.

CLINICAL MANIFESTATIONS –

Symptoms of septal rupture include chest pain, shortness of breath, and symptoms of low cardiac output and shock. Acute septal rupture produces a harsh, loud holosystolic murmur along the left sternal border, radiating toward the base, apex, and right parasternal area, and a palpable parasternal thrill in half of patients. With cardiogenic shock and a low output state complicating septal rupture, there is rarely a thrill, and the murmur is difficult to identify because turbulent flow across the defect is reduced. Right and left ventricular S3 gallops are common. The pulmonic component of the second heart sound is accentuated by pulmonary hypertension. Tricuspid regurgitation may also be present. Biventricular failure generally ensues within hours or days. As compared with acute mitral regurgitation, septal rupture has a loud murmur, a thrill, and right ventricular failure but is less often characterized by severe pulmonary oedema. In patients with a low cardiac output, distinguishing between these two entities can be difficult. In addition, severe mitral regurgitation may occur in 20 percent of patients with septal rupture.

DIAGNOSIS –

Pump failure in patients with myocardial infarction may be related to the major mechanical complications, such as ventricular septal rupture, papillary-muscle rupture, or free-

wall rupture. Alternatively, it can result from infarction or ischemia of a large area, ischemic mitral regurgitation, right ventricular dysfunction, or hypovolemia. Doppler echocardiography is diagnostic and can be used to define the site and size of septal rupture, left and right ventricular function, estimated right ventricular systolic pressure, and the left-to-right shunt. The sensitivity and specificity of colour Doppler echocardiography is as high as 100%. In severely ill patients who are receiving assisted ventilation, the image quality of transthoracic echocardiography may not be sufficient for diagnosis, and trans-oesophageal echocardiography is more sensitive. Pulmonary artery catheterization can be helpful. Severe mitral regurgitation may result in an increase in oxygen saturation in the peripheral pulmonary arteries. Coronary angiography is useful for assessing the coronary anatomy if concomitant revascularization is being considered. Radionuclide scintigraphy is an alternative non-invasive technique for diagnosing septal rupture, assessing ventricular function, and calculating the size of the intracardiac shunt.

MANAGEMENT

- Ventricular septal rupture (VSR) in the thrombolysis era
- thrombolysis has reduced the incidence of VSR 10–20 fold
- with thrombolysis VSR presents earlier after myocardial infarction and is more often complex than simple
- surgical mortality for surgical repair is higher in the thrombolysis era
- Echocardiography provides the following information:
- differentiation from papillary muscle rupture
- site and size of interventricular rupture
- Right and left ventricular function
- size of shunt
- Surgical principles of VSR closure:
- hypothermic cardiopulmonary bypass with myocardial protection
- trans-infarction approach to the VSR
- trimming of infarcted muscle around the VSR
- closure of the VSR with a patch to avoid tension
- closure of the ventricle without tension with buttressed sutures

Medical Therapy –

Medical therapy consists of mechanical support with an intra-aortic balloon pump, afterload reduction, diuretics, and usually inotropic agents. Oxygenation should be maintained with the administration of oxygen by mask, continuous positive airway pressure, bilevel positive airway pressure, or intubation with mechanical ventilation. Nitroprusside may reduce left-to-right shunting and improve cardiac output, but it may also cause hypotension. Its use is contraindicated in patients with acute renal failure. Patients with hypotension often need inotropic agents and vasopressors to maintain arterial blood pressure.

However, an increase in left ventricular pressure increases left- to-right shunting. Attempts to stabilize the patient's condition with medical therapy are only temporizing, because most patients have a rapid deterioration and die. Most patients require surgical intervention. Even patients whose condition appears to be clinically stable are at risk for abrupt hemodynamic deterioration, because the size of the septal rupture can increase without warning. The mortality rate among patients with septal rupture who are treated conservatively without mechanical closure is approximately 24 percent in the first 24 hours, 46 percent at one week, and 67 to 82 percent at two months.

Mechanical Closure –

Current guidelines of the American College Of Cardiology – American Heart Association for the Treatment of Patients with Acute Myocardial Infarction recommend immediate Operative Intervention in Patient With Septal Rupture, Regardless of their Clinical Status.

CASE REPORT

A 58 years old female admitted to our Hospital with chief complaints of Acute onset Left sided chest pain radiating to left shoulder and left side of jaw since with Shortness of breath (NYHA 3) since 3.5 hours. Patient had no Known co morbidities and had no significant past medical or surgical history. On presentation she was Conscious , oriented to Time, Place and Person , GCS – E4V5M6 , afebrile , HR 104 BPM , BP 110/60 mmHg , RR 20/min , SpO2 88% on RA 97 % on 4 lit NP. S1S2 was heard with no added sound. On chest auscultation – BL Basal Crepitation was heard. ECG – Sinus Tachycardia, Qs pattern in V_{1-4} / I / aVL , ST elevation V_{2-4} . Trop I – 5.6 . 2 D Echo – LVEF 55% with Hypokinetic IVS and Anterior wall. Patient was Thrombolysed with Inj. Tenecteplase 40 mg IV and following which patient was admitted in CCU. After 6 Hours of Thrombolysis patient became drowsy and her GCS – E3 V4 M5 . patient was Afebrile , HR – 150 BPM , Bp – 120/60 mmHg , RR – 26/min , SpO2 – 96% on 8 lit FM. On auscultation Pan Systolic Murmur Present in Tricuspid and Mitral Area. Repeat 2D ECHO – s/o Ventricular Septal Rupture. Patient was immediately taken for Coronary Angiography – which was s/o Triple Vessel Disease. Following which Patient was Taken for CABG with VSD closure. Patient was kept Under IABP support for the next 3 days. Patient was discharged on day 28 of admission. On follow up after 7 days patient was vitally stable. Pansystolic Murmur was still heard in tricuspid area.

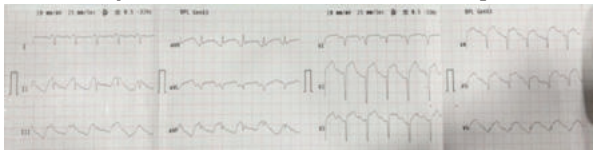

Fig : ECG after 6 hours of Admission

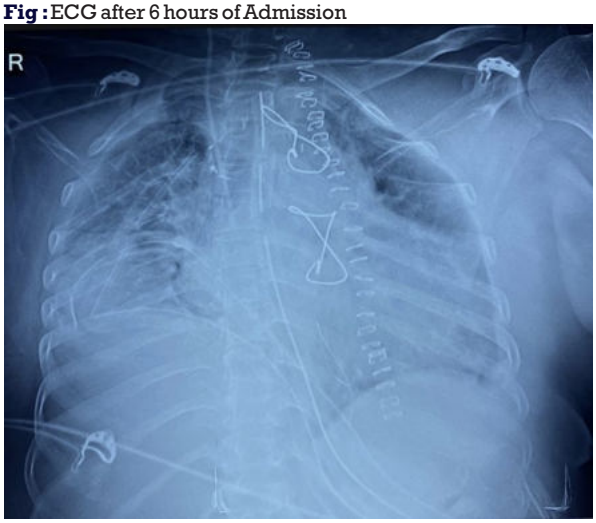

Fig : Post OP Day 2

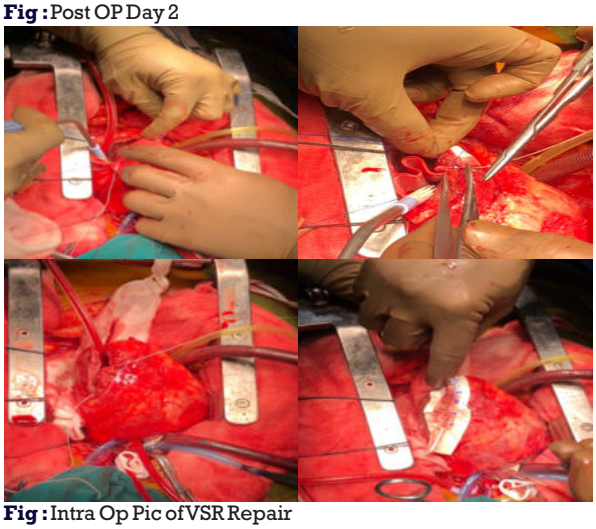

Fig : Intra Op Pic of VSR Repair

CONCLUSION

Ventricular Septal Rupture is a Complication that can happen following Acute Myocardial Infarction. In the pre-thrombolytic era, outcomes after septal rupture were extremely poor, with an in hospital mortality rate of approximately 45 percent among surgically treated patients and 90 percent among those treated medically. Patients Immediately taken up for surgery after development of Ventricular Septal Rupture has better out come.

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