Post-Infarction Left Venticular Free Wall Rupture

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Introduction

Left ventricular free wall rupture (LVFWR) is an unpredictable complication of acute myocardial infarction (AMI) with an incidence of 4-6% accounting for 8-17% mortality[1] only second to pump failure. LVFWR may not be suddenly fatal, upto 40% of deaths occur sub-acutely over a matter of hours. If this condition goes untreated, death is almost certain with only 17 survivals reported without surgical repair[2,3]. First report for surgical repair in LVFWR was by Montegut4 in 1972, since then scattered case reports and small series of surgical experiences have appeared in European, American and Japanese literature[1]. At Medline search from 1966, no report from Southeast Asia was found and to the best of our knowledge this is the first case report form Pakistan.

Case Report

A 43 year old man presented with in six hours duration of left pre-cordial and arm pain with electrocardiographic changes suggestive of extensive anterolateral myocardial infarction. He had new onset exertional angina for two months. He was a current smoker and had elevated cholesterol levels. Prompt thrombolysis was initiated with 1.5 million units of streptokinase infusion intravenously, which had to be discontinued ten minutes later for severe allergic skin rash and shivering, requiring intravenous hydrocortisone and clemastine administration. He was subsequently managed with intravenous nitrates and heparin along with aspirin and angiotensin converting enzyme inhibitors. On second hospital day his echocardiogram showed severe left ventricular systolic dysfunction and an apical thrombus and no evidence of pericardial effusion(PE). Same day he had clinical left ventricular failure requiring diuretics. On third hospital day he had pleuritic chest pain, without any audible rub, managed with non-steroidal anti-inflammatory drugs and episodes of self-limiting supraventricular tachyarrhythmia. On fourth hospital day he continued to have pleuritic chest pain, became hypotensive and bradycardiac. Repeat electrocardiogram revealed global ST-elevation Portable echocardiogram showed PE with tamponade effect and indistinct LVFWR. He was immediately taken to operating room, intra-aortic balloon pump (IABP) inserted and peripheral cannulation established. After median sternotomy complete cardiopulmonary bypass was established. LVFWR was identified at lateral wall adherent to adjacent pericardium. Successful surgical repair was performed with Gelatin resorcin - for maIm (GFR) adhesive glue and suture reinforcement. Post-operatively, patient required extended mechanical support due to pump failure. By seventh post-operative day he was weaned off from mechanical support and discharged on eleventh postoperative day. At four months follow-up, he remained well with New York Heart Association Class - II congestive heart failure.

Comments

Incidence

In a prospective study[1] the overall incidence of LVFWR among all AMI patients was 6.5%, with 4.1% having an acute course. As in recent years the frequency of death from arrhythmias has decreased due to improvement of critical care along with use of beta-blockers and thrombolytics, the relative

contribution of LVFWR to mortality after infarction has risen, making it now the second leading cause of death and accounting for 8 to 17% mortality postinfarctio[1].

Risk Factors

Risk factors for LVFWR includes advanced age greater than 60 years, female gender, pre-existing hypertension, first AM!, no previous angina, no evidence of left ventricular hypertrophy, lack of mural thrombus and presence of transmural infarct[1].

Rupture usually occurs early in the clinical course of infarcts, with upto 40% occurring within the first twenty-four hours of onset of recognized symptoms and 85% occurring with in the first week[1]. This correlates well with studies of changes in the biomechanical strength of myocardium after infarction in animal models, showing equal or greater strength of infarcted territory compared to normal muscle after seven days of infarction. Much debate has arisen concerning the association of thrombolytic use with myocardial rupture. The Thrombolysis In Myocardial Infarction (TIMI) trial investigators reported that among patients with fatal first myocardial infarctions, those randomized to treatment with thrombolytics (average time to therapy 3.1+1.3 hours) had a lower incidence of free wall rupture compared with those who did not (22% vs 46%, $p = 0.045$)[5]. Meta-analysis by Honan and colleagues[6] showed that the odds ratio of cardiac rupture increased significantly with delay in mean time to treatment, only therapy within seven hours was associated with a decreased risk of rupture.

In contrast Late Assessment of Thrombolytic Efficacy (LATE)[7] study by Becker and associates, done on 5,111 patients randomized to receive either recombinant tissue-type plasm inogen activator (rt-PA) or a placebo, demonstrated the highest number of ruptures among group receiving il-PA within 6 to 12 hours of symptom onset, although the data was not statistically significant.

Morphologic Patterns

In a prospective series[8] six pathologic varieties of ventricular free wall rupture were recognized based on findings during surgery or autopsy. For reasons poorly understood the most common site of rupture is the anterior or lateral wall and a mid-ventricular position along the apex to base axis is most frequent (66%). One theory involves the innate anatomic muscular arrangements of the left ventricle at that site whereas in another explanation it may be associated with the site of papillary muscle insertion, causing stress at insertion site leading to rupture[9].

Clinical Presentations

Clinical presentation may be acute or sub-acute but high index of suspicion is required to suspect this ominous complication. In particular an abrupt hemodynamic collapse in a post infarct patient with absence of cardiac murmurs of mitral regurgitation or ventricular septal defect is highly suggestive. Symptom occurring hours or days before the final event includes pleuritic chest pain, repetitive and unprovoked vomiting, restlessness and agitation unexplained hypotension, transient bradycardia, syncope and physical signs of tamponade[10]. Transient electromechanical dissociation is described in majority of patients, and has high predictive value especially when it occurs in patients who had their first myocardial infarction and were not in heart failure before rupture[11].

Diagnosis

The most common electrocardiographic change is a persistent or progressive ST-segment elevation in the absence of reinfarction, pericarditis and ventricular aneurysm formation. Ueda et al[12] have shown that patients with persistently high levels of C-reactive protein, particularly greater than 2Omg/dI, have a high probability of rupture. Two dimensional(2-D) transthoracic echocardiography is the most sensitive and expeditious diagnostic modality for LVFWR. The most consistent finding is PE. Only rarely is the point of rupture identified either on 2-D or Doppler investigation. In addition, PE with echodense masses overlying the heart, independently of cardiac tamponade is highly suggestive of LVFWR. The role of invasive testing is unclear. There are scattered reports of individual cases identified by other techniques i.e. transesophageal echocardiography[13], cineventriculography and

technetium-99m-labelled pyrophosphate scanning[14]. Pericardiocentasis can relieve pericardial tamponade physiology in patients with free flowing pericardial effusion and stabilize the patients hemodynamic while they await surgery. A serous fluid on pericardial tap effectively rules out free wall rupture.

The role of pre-operative coronary angiography is unclear. These patients usually have multi-vessel disease (81%), and all have severe obstruction of at least one major epicardial artery[14]. The benefit of doing angiography in hemodynamically stable patients verses the risk of the patients dying because of delayed treatment is unknown, as only nine of the eighty seven reported long term survivors of free wall rupture had coronary artery bypass grafting as part of their surgical treatment1. However, surgery should not be delayed unnecessarily for cardiac catheterization in these critically ill patients.

Management

The first step is to achieve stable hernodynamics in an unstable patient by using rapid infusions of fluids along with inotropic support[15]. The next step is Pericardiocentasis, if successful provides relief from pericardial tamponade physiology and improves hemodynamics. The theoretic concern of excessive hemorrhage is not of clinical significance. In patients not improving with pericardial tap, IABP , if available should be placed early[16]. The use of partial femoral bypass has also been reported[14]. Without surgical repair death is almost certain, so far only 17 survivals have been reported without surgical repair[2,3]. First report for surgical repair in LVFWR was by Montegut4 in 1972. The classical approach is to remove the area of infarction and replace it with a prosthetic patch under cardiopulmonary bypass (CPB). In cases in which a large portion of the left ventricular free wall is infarcted and infarctectomy can lead to small ventricle, a Dacron prosthesis should be used. In centers where facilities for CPB are not available, patients with anterior or lateral wall rupture, the most common site, can be directly closed with pledged sutures. Recently application of a patch to the epicardial surface with biologic glue has been reported to lead to a successful outcome[17]. In order to prevent early post-operative ischemia and improve long term prognosis, many surgeons opt for grafting all major vessels empirically guided by palpable disease. There are no conclusive data to support or refute revascularization strategy at the time of repair because of the number of reported survivors is small. Data on operative mortality rates are few and even limited by publication bias, as numerous cases of attempted repair Sendon et al[18] reported an immediate operative mortality of 24% and hospital mortality rate of 52%. These mortality rates are high but less than the virtually 100% eventual mortality of an untreated ventricular free wall rupture. No patient that survived to discharge died over a mean 30-month's period of follow-up[1].

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