VENTRICULAR FREE WALL RUPTURE

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Background: Left ventricular free wall rupture (FWR) is reported to occur in 2–6% of cases presenting with acute myocardial infarction. Mortality of this complication is very high, accounting for 20–30% of all infarct related deaths. The objective of our study was to present our surgical experience of free wall rupture over a period of five years from 2004–2009 **Methodology:** A review of our records over this period of time was undertaken. **Results:** In our series of six patients collected over this time period, three (50%) presented with hypotension and three (50%) with persistent chest pain. ECG evidence of myocardial infarction was present in 4 (67%) cases, LVH in 2 (33.3%) cases and 2 (33.3%) showed diffuse ST&T changes. Echocardiogram was useful in the diagnosis of rupture but was not confirmatory. Coronary angiography and left ventriculography was performed in all the patients. Surgery was performed in all cases confirming the FWR. **Conclusion:** In this small series there was no surgical mortality which may reflect the favourable prognosis in sub acute rupture where haemodynamic stability is achieved with medical therapy prior to surgery.

Keywords: Free wall rupture, myocardial infarction, coronary artery bypass grafting.

INTRODUCTION

Left ventricular free wall rupture (FWR) is a dramatic complication of acute myocardial infarction.[1] It is infrequent occurring in 2–6% of acute myocardial infarctions but carries a high mortality due to sudden death, and is presumably responsible for as much as 20–30% of all infarct related deaths.[2] In recent years major advances in the management of myocardial infarction including thrombolysis and primary percutaneous coronary intervention has dramatically reduced the incidence of FWR, however, mortality due to it as a complication of myocardial infarction continues to be high.[4] A review of literature on FWR reveals that the majority of publications on the subject are single case reports with a review of the subject. There is one study that has reviewed their experience of FWR over a 30 year period.[3] It is apparent from this study that the incidence of FWR has reduced over the last three decades.

Surgical experience of FWR at any one centre is extremely limited because of the exceedingly high immediate mortality of acute rupture and difficulty of its early diagnosis. Sub acute rupture on the other hand may allow time for diagnosis and surgical management with gratifying long term results. To our knowledge there is no publication on the subject of acute or sub acute FWR from Pakistan, though there is one paper from Pakistan that describes the surgical experience of the patients with false aneurysms.[5]

The objective of our study was to review our experience over a period of five years of FWR and present our findings along with a review of literature on the subject.

The first free wall rupture of the heart after myocardial infarction was described by William Harvey in 1647. Hatcher and colleagues from Emory University reported the first successful operation for free wall rupture of the right ventricle in 1970. Fitzgibbon and associates in 1971 and Montegut in 1972 reported the first successful repair of left ventricular rupture associated with ischemic heart disease[5]. Left ventricular free wall rupture is third to cardiogenic shock and arrhythmias as the leading cause of death following a myocardial infarction.[6,7] There is a history of previous myocardial infarction in 25% of cases but often left ventricular free wall rupture can be the first presentation of ischemic heart disease.[7]

Free wall rupture may occur within 24 hours or between 5–7 days but rupture has been reported to occur as late as one month or even beyond.[1] Previous literature studies report the commonest site of rupture to be the anterior wall. Recent series have observed greater occurrence of lateral and posterior wall ruptures.[4]

Ruptures have been classified in several ways simple and complex ruptures, morphological types of rupture, acute subacute and chronic ruptures, blow-out and oozing type of rupture and early and late rupture.

A simple rupture results from a straight through and through tear that is perpendicular to the endothelial and epicardial surfaces. A complex rupture results from a more serpiginous tear often oblique to the endocardial and epicardial surfaces.

Morphological classification of rupture as type 1–4 has been described. Type 1 has little dissection or infiltration of the myocardium; Type 2 has a multicanalicular trajectory with extensive myocardial dissection; Type 3 rupture is protected either by a thrombus sitting at the orifice on the ventricular site or by pericardial adhesion; Type 4 rupture is incomplete as the trajectory does not traverse through the layers.[3]

Clinically ruptures can be divided into: acute rupture which results in death within a few minutes due to massive haemorrhage into the pericardial cavity; subacute rupture which is characterized by a smaller tear which may be temporarily sealed by a clot or fibrinous

pericardial adhesions which may be compatible with life for several hours or even longer; chronic rupture with false aneurysm formation which occurs when the leakage of blood is slow and when surrounding pressure on the epicardium temporarily controls the haemorrhage. Adhesions form between the epicardium and pericardium which reinforce and contain the rupture.[6]

An effort has been made to further discriminate the ruptures of the free ventricular wall depending on the clinical picture that they present.

Blow out ruptures are manifested with cardiogenic shock while stuttering ruptures have a less noisy clinical picture and varying severity of symptoms without haemodynamic instability. This latter category is clinically characterized by small rupture that is assessed during the operation and that most probably could tamponade spontaneously.[4] Ruptures have also been classified as early when it develops within the first 48 hours or late rupture when it develops beyond the second day.[6]

Factors facilitating free wall rupture include delayed hospital admission >12–24 hours, persistent systemic hypertension, unusual in hospital physical effort (agitation, repetitive vomiting, coughing), extension of myocardial infarction, expansion of myocardial infarction.[6]

These patients commonly present with acute tamponade and sudden electromechanical dissociation leading to sudden death, or severe hypotension. Patients may present with moderate to severe pericardial effusion and hypotension of varied severity, usually associated with sinus bradycardia or nodal rhythm and jugular venous distention[6]. Symptoms also include nausea, hypotension, angina, pericardial type of chest discomfort or pain.[7]

The pathophysiological process of FWR involves thinning of the myocardial wall with the intensity of necrosis occurring at the distal end of the vessel (watershed area) where there is often poor collateral flow. The shearing effect of myocardial contraction against a stiffened necrotic area causes rupture. The most common rupture location is on the anterior or lateral wall of the left ventricle. A mid-ventricular position along the apex to base axis is most common.[7]

On admission the ECG is usually consistent with transmural acute myocardial infarction, with a notably elevated ST segment which tends to remain persistently elevated over the ensuing hours or days.[6] Echocardiography is the procedure of choice for the diagnosis of left ventricular free wall rupture and usually reveals a pericardial effusion with intrapericardial echoes consistent with haematoma. Occasionally, diastolic collapse of the right ventricle can be seen. Echocardiography has a diagnostic sensitivity of 100% and a specificity of 93%.[8] The role of new imaging techniques in this setting such as cardiac MRI although promising remains observational and anecdotal. The wide availability of ultrasound makes this technique difficult to beat in the clinical arena, as the expensive technology and expertise needed for cardiac MDCT or MRI imaging are not promptly available in the majority of hospitals.

The experience of any surgical team in dealing with this catastrophic complication is likely to be limited and so the approach to it is not clearly standardized and long term outcome after repair is limited. Emergency surgery is usually the only therapeutic option available.[3] However mortality of surgically treated patients with several different surgical techniques varies widely among centers ranging from 11–80% in acute FWR where as in sub acute rupture hospital mortality is about 25% with long term survival of 48%.[10] Some authors have suggested that primary angioplasty reduces and even avoids the risk of free wall rupture especially when a successful angiographic result is obtained, however limited data is available in this regard.[11]

MATERIAL AND METHODS

We reviewed our computerized database of admissions for a period of five years from Jan 2004 through June 2009. No patients were excluded from the review based on age, sex or co-morbid conditions.

Surgical Technique

After coronary angiogram all six patients underwent surgery. Five of the patients underwent surgery on bypass while one was operated off pump. Only one patient underwent CABG along with the free wall rupture repair as that was the only one with significant triple vessel coronary artery disease.

All six patients underwent emergency/urgent surgical repair. Standard median sternotomy was performed in all patients and they were fully heparinised.

Pericardium was carefully opened without disturbing the haematoma around the rupture site. One patient with small apical rupture was repaired without using cardio pulmonary bypass (CPB) using Teflon pledget with 3/0 proline mattress stitches. In the remaining five patients after limited opening of pericardium, ascending aorta and bicaval cannulation was performed and cardio-pulmonary bypass was instituted. LV was vented via right superior pulmonary vein (RSPV). Routine 28 °C systemic hypothermia and cold intermittent ante grade blood cardiolegia was used.

Haematoma around the rupture was removed and rupture site identified. Rupture sites in our patients

were lateral wall, inferolateral, inferoapical, lateral wall and posterolateral wall.

All necrotic myocardium was excised to the healthy margin. Defect was repaired with Teflon felt using double layer continuous 2/0 proline suture and haemostasis secured. All patients had routine temporary ventricular pacing wire inserted.

All five patients were weaned off CPB with routine inotropic support without any problem and were transferred to ICU in stable condition. None of our patients needed IABP or reopening for bleeding post operatively.

RESULTS

During this period of time only six cases of FWR were recorded and surgically treated who form the basis of this report.

Five of these patients were men and 1 was a female. Three of the men were over 70 years of age, one was 50 years old and two were in their 30's (35 and 38) Table-1.

Two of these patients had a history of hypertension with poor drug compliance, one had LVH but no history of hypertension. None of them were diabetic or smokers. Five of them had a history of coronary artery disease, two had previous myocardial infarctions and three had a history of angina.

All six presented with a history of chest pain of several hours to several days duration (two presented within 12 hours of chest pain, two 5–7 days after chest pain and two 1–2 weeks after onset of symptoms). Two of them had associated shortness of breath, four had associated vomiting two had generalized weakness; nausea and sweating was experienced by one patient each. Four of these patients were haemodynamically unstable at the time of admission with a blood pressure of less than 90/60 and were supported with I/V fluids and ionotropes. Two had evidence of left ventricular failure in the form of bilateral basal crepitations; one had developed a gallop rhythm; the rest of the systemic examination was unremarkable.

The ECG showed an anterior wall infarction in one patient; inferior wall MI in two patients; lateral wall MI in one patient. All these patients showed diffuse ST and T changes; one patient had diffuse ST and T changes but no Q waves; while one had evidence of left ventricular hypertrophy and diffuse T changes. The lab workup of only one patient was completely normal. Only one patient had raised cardiac enzymes, Ck-MB and Trop-I. Three patients had a raised urea and creatinine level, two had deranged liver function tests. The haemoglobin of two patients was low and one had a raised ESR.

Transthoracic echocardiogram was done in all six patients, three of whom exhibited chamber dilatation with an otherwise preserved LV function; one with a moderately impaired and the third with a severely impaired LV function. A total of three patients had left ventricular impairment (2 moderate, 1 severe). Three showed evidence of apical aneurysmal dilatation, 2 were suggestive of pseudoaneurysm formation. One echo revealed multiple clots and 2 showed large LV thrombi. In 3 of the echocardiograms a defect was visualised in the LV wall, in one it was seen in the posteroinferior wall (12 mm), 4 cm in the posterior wall, 2.5 cm in the lateral wall. One echo was suggestive of VSD and pseudoneurysm and mitral regurgitation; pericardial effusion was seen in only one case.

All patients underwent a coronary angiogram. One patient had single vessel coronary artery disease with impaired LV function and an apical clot and had been advised medical management prior to this admission. One had mild coronary artery disease with posterolateral rupture at the apex; 2 had normal coronary arteries; only 1 had significant TVCAD (triple vessel coronary artery disease) with LV rupture and pseudoaneurysm formation.

All these patients were started on drug therapy while they were undergoing investigations. All received clopidogrel except one; 4 of them received aspirin, 3 of them received a statin; 3 received anticoagulants in the form of heparin and clexane. Digoxin and nitrates were given to 4 patients; diuretics were given to 3 patients; 2 were given ACE inhibitors and 1 received amiodarone. None of them received lytic therapy.

All patients tolerated the surgical procedure very well; they remained in the ICU as per routine with a smooth uneventful recovery and were shifted to the floor on the second post operative day as per protocol. Only one patient received amiodarone for the treatment of arrhythmias. All 6 patients were discharged one week after surgery. Post operative follow up echocardiograms were available which showed improvement of LV function.

Table-1: Characteristics of the patients with ventricular free wall rupture

No.	Age (Yrs)	Sex	Area of rupture	Initial Presentation	Thrombolysis	LV dysfunction	Previous history of IHD	History of co-morbids
1	50	M	Inferoapical	Cardiogenic shock	Nil	Moderate	+ve	None
2	75	M	Posteroinferior	Cardiogenic shock	Nil	Mild	+ve	None
3	35	F	Apical	Cardiogenic shock	Nil	None	-ve	None
4	38	M	Apicolateral	Angina	Nil	None	+ve	HTN
5	74	M	Posterolateral	Angina	Nil	Moderate	+ve	LVH
6	70	M	Lateral wall	Angina	Nil	Severe	+ve	HTN

DISCUSSION

In our series of six patients the clinical presentation was of the sub acute type where no acute cardiac tamponade or electro-mechanical dissociation occurred. Three of the patients presented with chest pain and three with hypotension. All the patients presented several hours to days after the onset of symptoms suggesting a sub acute course. As reported in other series[12] left ventricular hypertrophy with or without hypertension was seen in three patients (50%). Diabetes mellitus was not a predisposing factor similar to other published reports.[12] Previous diagnosis of coronary artery disease was present in two of the six patients; consistent with other reported series.

Echocardiography is the diagnostic modality of choice in patients with both acute and sub acute FWR.[1,2,9,10] The presence of echo dense pericardial fluid more than 5 mm in the setting of chest pain, hypotension and pericardial tamponade or electromechanical dissociation is highly sensitive and specific. In some patients a tear in the ventricular wall may be seen.[9,10] Myocardial tears in the sub acute form are seen more frequently in the lateral or inferior areas as compared to the anterior wall. In our cases tears were seen in the postero-inferior, posterior and lateral wall in one patient each. Pericardial effusion was seen in only one patient which might indicate that the rupture in our cases may have been old and extension of tear may have occurred into a false aneurysm or were small localized tears effectively sealed by a clot or the pericardium. Left ventricular function was impaired in three of our patients which is consistent with other reported series.[8–10] The impaired systolic function in our patients may indicate the late presentation in some of our patients with prior coronary disease. FWR into the pleura and mediastinum has been described in a patient in whom a previous pericardiectomy had been performed.[13]

Coronary angiography and cardiac catheterization in the setting of acute FWR is not advisable as it delays urgent surgery. However in the sub acute form where the patient can be stabilized it is helpful for surgical decision making and performance of coronary artery bypass grafting where indicated.[15] Autopsy series have shown significant coronary artery disease in cases of FWR hence when possible coronary angiography and coronary artery bypass grafting when performed may improve long term survival but may have no effect on short term mortality. Coronary angiography may be performed in the operating room at the time of surgery as recently reported.[14]

Coronary angiography was performed in all our cases. Only 1 patient had severe triple vessel disease in whom coronary artery bypass grafting was performed. Two patients had normal coronary arteries, 1 had single vessel disease and 2 had mild disease in the infarct related vessels. These findings are similar to other series.[3] Ventriculography may outline the rupture in some acute cases.[15]

Medical management of FWR is dependant on early diagnosis and prompt management. Intravascular volume expansion by colloid solutions, inotropes and pericardiocentesis are required for stabilization in the majority of patients. Continuation of medical management with beta blockers; prolonged bed rest; repeated pericardiocentesis and strict blood pressure control has been advocated by Figueras *et al*[3] as an alternate to surgical management with good long term survival. However, there is a paucity of data on the subject from other institutions. One study from Japan has reported a small series of cases where intra-pericardial injection of glue has been used for repair of the ruptured wall with acceptable long term results.[16] In situation where other co-morbidities preclude surgery it may be considered a viable alternative. In all other cases after stabilization surgical repair is the treatment of choice.

Four main surgical techniques have been advocated:

1. Horizontal continuous sutures reinforced with Teflon patches. In this technique the sutures are placed in the necrotic zone and hence has a high risk of recurrence.
2. Resection of the necrotic area and closure of deficit with Teflon reinforced sutures.
3. The third method requires continuous suture reinforced with a double Teflon layer.
4. The fourth technique requires gluing of a Teflon patch or bovine pericardium over the involved area with bio compatible glue or fibrin.

All these methods have pros and cons and as of now there is no uniform method of repair. Each case is dealt with individually by the surgeon.

In all our cases surgery was performed. Five of the patients were placed on bypass for ease of repair of the ventricular rupture on the posterior and lateral wall. Five patients had repair with Teflon and Prolene sutures and in one patient the rupture was repaired with pledgeted purse string sutures. None of our patients required intra aortic balloon insertion the routine use of which is controversial.[4,17] In one patient repair was performed on the beating heart. One patient required coronary artery bypass grafting. All our patients did well post operatively and were discharged from the hospital on the seventh post operative day. Our surgical results compare well with other reported case series. In our small series of six patients we did not have any surgical mortality which compares favourably with other similar series where mortality has been 25–80%.[2,4,17] This may reflect the better prognosis of patients who present with sub acute FWR

and can be stabilized on medical treatment prior to surgery.

Post operative echocardiograms showed improved systolic function in whom it was impaired preoperatively. No pericardial effusion was seen.

Long term follow up of 3½ years; 4 years; and 5 months with no recurrence is available in 3 patients. The other 3 patients are lost to follow-up after 1 month of follow-up. The recurrence rate after FWR repair where reported has been low. Long term results in 3 of our patients and short term in the other 3 cases are similar to the other series reported. There is a single case report of over 10 years survival.[18]

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