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Outcome of Conservatively Managed Left Ventricle Free Wall Rupture in Stemi, A Rare Case Scenario

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Introduction

In patients with acute myocardial infarction, left ventricular free wall rupture is an infrequent complication (2–4%) but it is associated with a high mortality from pericardial tamponade.[1-8] It accounts for 5–24% of all in hospital deaths related to acute myocardial infarction.[4,6,9,10]. To reduce this high mortality it is important to improve the way in which these patients are classified, as this may help clinicians anticipate myocardial rupture, prevent it occurring, and achieve better therapeutic results when it does occur. Left ventricular free wall rupture (LVFWR) is a rare complication that can occur after suffering a myocardial infarction (MI). The incidence of LVFWR has decreased dramatically over the years with the increased use of reperfusion strategies such as percutaneous coronary intervention (PCI) and fibrinolytic therapy, with an overall incidence ranging from 0.8% to 6.2%[1]. LVFWR is most likely to occur 1–4 days after the initial myocardial insult, andis one of the more deadly complications of MI[2]. The early diagnosis of LVFWR is critical and point of care echocardiography can help establish the diagnosis quickly by revealing evidence of pericardial effusion and tamponade. Characteristically, free wall rupture occurs in relatively elderly patients, generally older than 55 years and usually between 65 and 70,[1,3-5,11-13] without any apparent sex bias[3,14-17] although it may be relatively more common in female patients in view of the lower incidence of acute myocardial infarction in women.[4,5] In most cases the myocardial infarct is the first one recorded and it is usually transmural[2,4,12-14,18] but without overt heart failure.[2,13,14,18] There is an infrequent history of angina pectoris.[2,13,14,19] The incidence of diabetes is not particularly high, though arterial hypertension is common—often more than 50%, but with some exceptions.[1,5,6,12,14,18] These patients present with a rather prolonged episode of anginal pain, often lasting for four to six hours or more,[1,2,4,5,15,16,20] but sometimes with pain of shorter duration that has been preceded by other episodes lasting more than 30–60 minutes in the days leading up to the precipitating event.[16] Not infrequently these patients experience delay in admission to hospital,[2,9,16,18]often because of misdiagnosis but occasionally because of a silent acute myocardial infarct complicated by pericardial tamponade and manifested as syncope or systemic hypotension. This delay in hospital admission is often associated with maintenance of

some ambulatory activities.[2] ,[16] ,[18] Additional risk factors, or triggering factors, for free wall rupture are the presence of persistent arterial hypertension (⩾ 150 mm Hg) during the first 10–24 hours of the acute infarction while in hospital at rest, and any undue physical effort such as persistent coughing, vomiting, or agitation. In this paper, we provide an update on the clinical, electrocardiographic, echocardiographic, and angiographic features of a patient presenting with Left ventricular free wall rupture which was managed using conservative strategy that may be applied successfully to a selected subgroup of patients.

Case Presentation

A 70 year old male with a history of heavy smoking .The patient had been complaining of chest pain and dyspnea on exertion since last few days, for which he consulted local physician from where he was reffered to higher centre. He had history of chronic smoking. No past history of tuberculosis. Intially for few days they did not seek medical care due to some personal issues. Patient presented to our emergency department with severe pain and dyspnoea and a 12 lead electrocardiogram (ECG) performed revealed ST-segment elevations and pathologicalq-waves in leads II, III, and a VF consistent with inferior ST-segment elevation myocardial infarction (STEMI) and with low voltage QRS complexes (Fig. 1). Troponin I was positive.

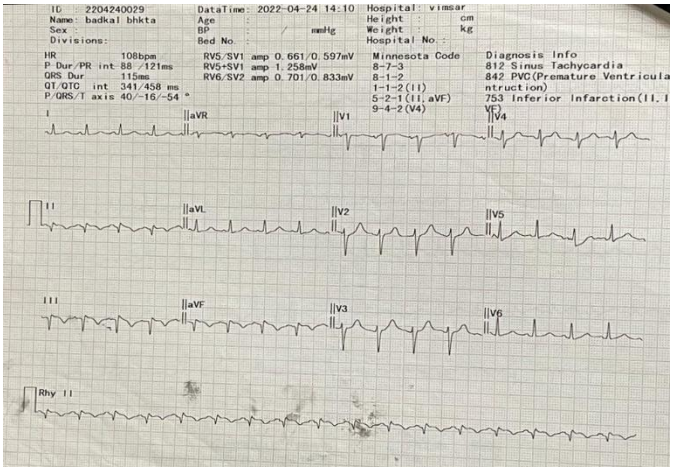

Fig. 1

Remaining lab investigation were within normal limit. Patient was hemodynamically unstable blood pressure was 80/46 mmhg; heart rate 104/ min; spo2 94% at room air; patient was subjected for chest x ray which showed mediastinal widening and increased cardiothoracic ratio (fig2) and echocardiography was done which revealed a depressed ejection fraction and hypokinesia in basal interventicular septum [ivs] and posterior wall associated with aneurysmal dilation of basal ivs and associated with large pericardial effusion with organised homogenous clot in pericardium attached to myocardium (fig 3).

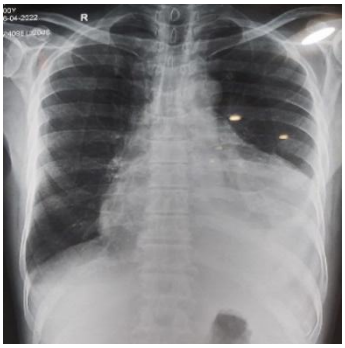

Fig2

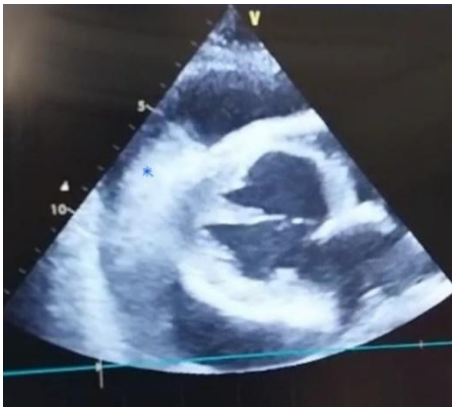

Fig 3 showing organised clot in pericardium

A pericardiocentesis was done with pigtail catheter (fig 4) in view of large pericardial effusion but there was risk of displacement of clot which could have lead to increased in pericardial effusion. Pigtail catheter was introduce in pericardial space under fluoroscopy guided and around 600 ml of fluid was removed and send for cytology and biochemistry which showed it was blood. Pigtail was left insitu for 5 days and then It was removed as there was no further drain coming from it.

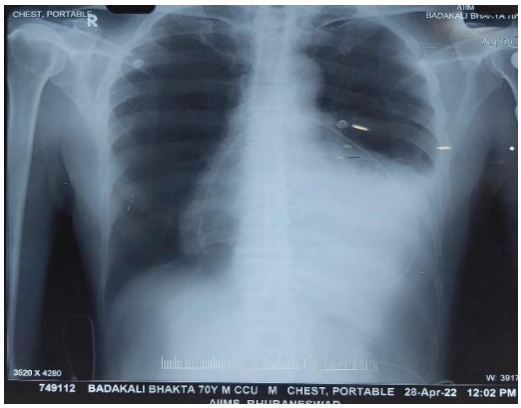

Fig 4

Patient hydration was maintained with i.v fluid and dopamine was started to maintain blood pressure and renal perfusion . Patient was taken coronary angiography which showed right coronary artery total occlusion and left coronary artery where having mild disease (fig 5). So patient was decided to be start on single antiplatelet ticagrelor 60 mg in twice daily dose to avoid risk of further bleeding in pericardial space and also during process of coronary angiography no heparin was given.

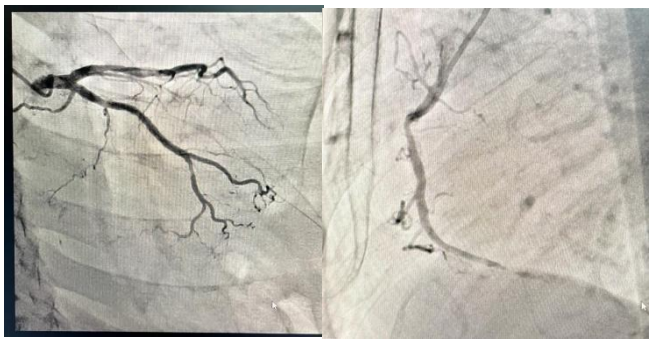

Fig 5

Repeat chest x ray (fig 6) after 10 days showed decrease in pericardial and pleural fluid.

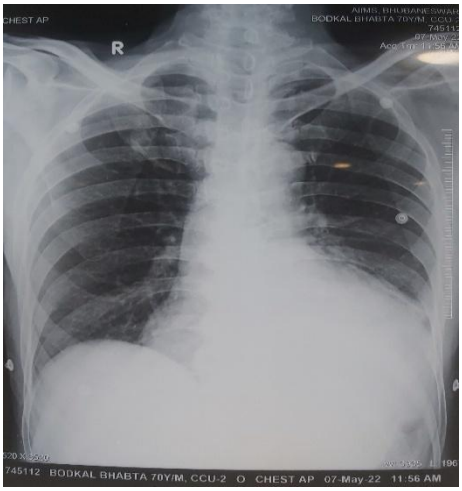

Fig 6

Echocardiography was repeated and showed mild pericardial effusion and also revealed a reduce ejection fraction and hypokinesia in basal interventicular septum [ivs] and posterior wall associated with aneurysmal dilation of basal ivs with fibrinous strand and organised clot in pericardial space (fig 7).

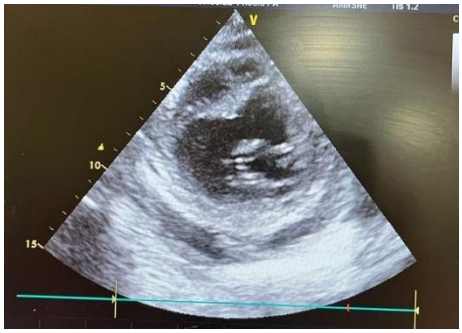

Fig 7

So the patient was finally discharged after 14 days with single antiplatelet at low dose ticagrelor 60 mg in twice daily dose. It is rare to encounter a case of LVFWR and in that also it's very rare to successfully manage LVFWR conservatively owing to its high mortality.

Conclusion

Diagnosis of free wall rupture is strongly suspected when a patient with a high risk profile—age > 55 years, first transmural acute myocardial infarct, absence of overt heart failure, persistent ST segment elevation, prolonged pain during the acute phase—presents with sudden hypotension or electromechanical dissociation, often preceded by recurrence of chest pain, together with raised jugular venous pressure and a moderate to severe pericardial effusion on echocardiography. To avoid semantic confusion, it is proposed that the term "acute free wall rupture" be reserved for patients with cardiac arrest caused by electromechanical dissociation or severe hypotension, and "subacute" rupture be applied to patients with hypotension of lesser degree.

When *cardiac arrest* has occurred, management includes cardiac massage, ventilatory support, administration of inotropic agents and colloids, and pericardiocentesis. If improvement occurs,

conservative management is then continued in close consultation with the surgical team. If initial management does not result in improvement, emergency thoracotomy is performed, preferably without cardiopulmonary bypass, and the rupture site is repaired with a Teflon patch glued to the epicardium. When the manifestation of free wall rupture is *hypotension* rather than cardiac arrest, initial management should involve colloid infusion, dobutamine infusion, and pericardiocentesis with aspiration of sufficient pericardial fluid (10–50 ml) to restore haemodynamic competence and urine output. Conservative management is then continued. If haemodynamic recovery cannot be achieved, surgical treatment is indicated. In all cases, conservative management involves blood pressure control (β blocker treatment) and avoidance of physical stress (bed rest for 5–7 days).Successful conservative strategy has little support in the literature, apart from a few articles describing conservative treatment, which is only possible in a self-selected group of oozing or subacute-type rupture. Nevertheless, it may be an option in extremely high-risk groups (especially older patients), where surgical intervention is considered futile

A conservative approach is particularly indicated in patients with important comorbidity factors that greatly increase the surgical risk, such as severe chronic lung disease, renal failure, extensive myocardial infarction, or serious peripheral vascular disease. This method of treatment, which represents a challenge in most settings and contravenes the standard surgical approach in all cases of suspected free wall rupture, will need to be validated by a substantial cohort of patients from more than one institution.

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