Case Report

Timely Diagnosis of Left Ventricular Posterior Wall Rupture by Echocardiography: A Case Report

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Abstract

Left ventricular free wall rupture is responsible for up to 10% of in-hospital deaths following myocardial infarction. It is mainly associated with posterolateral myocardial infarction, and its antemortem diagnosis is rarely made.

One of the medical complications of myocardial infarction is the rupture of the free wall, which occurs more frequently in the anterolateral wall in hypertensives, women, and those with relatively large transmural myocardial infarction usually 1-4 days after myocardial infarction.

We herein present the case of a 66-year-old man suffering inferior wall myocardial infarction with abrupt hemodynamic decompensation 9 days after myocardial infarction. Emergent transthoracic echocardiography revealed massive pericardial effusion with tamponade, containing a large elongated mass measuring 1 × 8cm suggestive of hematoma secondary to cardiac rupture. In urgent cardiac surgery, the posterior wall between the left coronary artery branches was ruptured.

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Introduction

Left ventricular (free wall) rupture is the major culprit in up to 10% of in-hospital deaths following acute myocardial infarction (MI), usually between 3 to 6 days after the infarction. It typically involves the anterior or lateral wall, at the terminal region of the left anterior descending coronary artery distribution. It is associated with transmural infarctions involving at least 20% of the left ventricle (LV), and it rarely occurs in areas with good collateral blood supply.[1, 2] The local factors that beget myocardial rupture are thinness of the apical wall at the terminal end of blood supply, poor collateral flow, and shearing effect of muscular contraction against an inert and stiffened necrotic area.

Occurring more frequently in elderly, female,[2, 3] and hypertensive patients,[1, 4] myocardial rupture is detected more often in the LV than in the right ventricle (RV)[3] and tends to strike between day 1 and week 3 but more commonly within 1 to 4 days post infarction.[1] In addition, it is known to happen most commonly in patients with delayed hospital admission and its concomitant maintained physical activity.[5]

Rupture of the LV free wall usually gives rise to hemopericardium and death from cardiac tamponade. The course of rupture varies from a catastrophic event, with an acute tear leading to immediate death (acute rupture), to a slow and incomplete tear causing a late rupture (sub-acute rupture). An incomplete rupture may occur when the thrombus and hematoma together with the pericardium seal the rupture of the LV and may develop into a diverticulum or a false aneurysm. The clinical recognition of the rupture is often first suggested by the development of profound RV failure and shock progressing to electromechanical dissociation. Immediate pericardiocentesis will temporarily

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relieve tamponade, followed by cardiopulmonary bypass and coronary artery bypass graft surgery to repair the wall.

Although there is often insufficient time for diagnostic tests in patients in whom an acute LV free wall rupture is suspected, echocardiography is the examination of choice. Echocardiography may demonstrate a pericardial effusion and typical findings of cardiac tamponade. Be that as it may, identification of the rupture site is rarely possible.

Case report

A 66-year-old man with a history of systemic hypertension was admitted to our hospital due to neglected inferior wall MI (6 days before) and recurrent chest pain. Physical examination was unremarkable. Initial twelve-lead electrocardiogram displayed q wave and ST segment elevation in leads II, III, aVF, and V_6, while cardiac markers showed elevated creatine kinase (CKMB = 54 Iu/L) and cardiac troponin I (11.4 µg/l). Chest pain was controlled by medical treatment. Transthoracic echocardiography (TTE) showed normal LV size with moderate concentric hypertrophy and moderate LV systolic dysfunction (ejection fraction = 40%); hypokinesia of the base and mid septal, base and mid inferior, and mid posterior segments; mild to moderate RV dysfunction; and no pericardial effusion. On the third day after admission, the patient suddenly developed hypotension; his blood pressure dropped to 90/50 mmHg and he exhibited cold perspiration, tachycardia, and paleness. ECG showed no new ischemic changes.

Emergent bedside TTE revealed severe LV systolic dysfunction (LVEF = 25%) and akinesia of the base and mid inferior, base and mid septal, and mid posterior segments as well as large pericardial effusion associated with diastolic collapse of the RV, RV outflow tract, and right atrium (Figures 1 & 2). Over and above the massive effusion in the pericardial space, an elongated echo-dense mass measuring 1 × 8cm was detected over the right atrium and RV, suggestive of hematoma. The patient, therefore, underwent emergent coronary angiography followed by urgent cardiac surgery with the diagnosis of myocardial rupture.

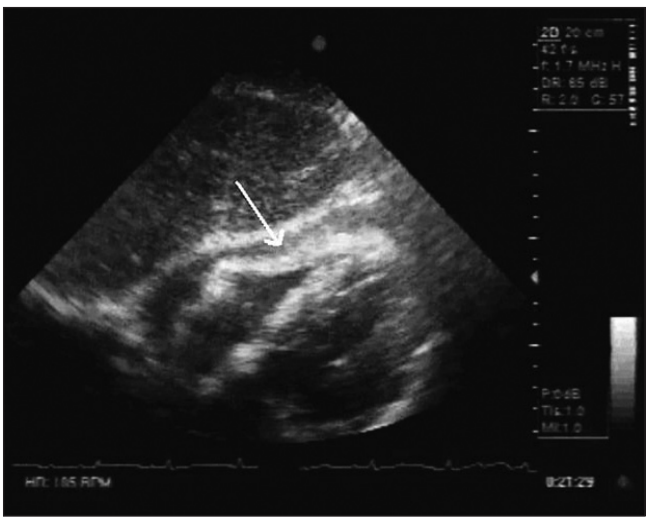

Figure1. Subcostal 4-chamber view echocardiography revealing mixed effusion and hematoma around the right atrium and right ventricle responsible for the right ventricular collapse (arrow)

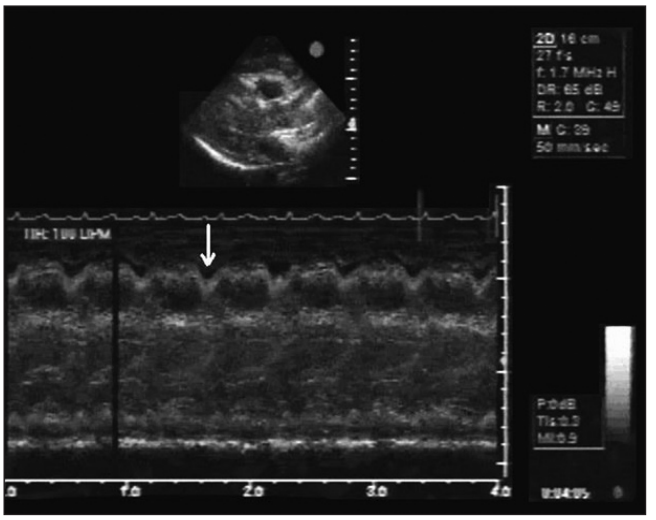

Figure 2. M-mode echocardiography from the parasternal long-axis view demonstrating marked right ventricular collapse (arrow)

During surgery, one liter bloody effusion was initially drained from the pericardial space, and the site of the cardiac rupture at the posterior wall between the left coronary artery branches was repaired so that coronary artery bypass grafting could be performed.

The patient's post-operative course was uneventful, and he was discharged one week after cardiac surgery.

Discussion

One of the fatal complications of MI, myocardial rupture accounts for up to 10% of in-hospital deaths in the wake of acute MI.[1] The course of rupture varies from catastrophic, with an acute tear leading to immediate death, to sub-acute, accompanied by nausea, hypotension, and pericardial type of chest discomfort.[3] Factors contributing to this complication are delayed hospital admission and the resultant maintenance of physical activity as well as recurrent and intense chest pain.[5]

Our patient was hypertensive with delayed hospital admission. His inferior wall was involved and cardiac rupture had already occurred without the preceding chest discomfort. Fortunately, however, the timely diagnosis and emergent cardiac surgery brought about the successful sealing of the rupture site with the hemopericardium and the patient's life was saved.

The prognosis of a cardiac rupture is very poor even when

surgical therapy is contemplated;[1] we would, therefore, maintain that patients may fare better if they are afforded early diagnosis and management of this frequently fatal condition.

Conclusion

The case presented here highlights the merit of TTE as regards the early diagnosis of LV rupture. Echocardiography can be performed at bedside; consequently, it has the potential to identify this catastrophic condition in patients with acute MI and to determine the next course of action.

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