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Echocardiographic Capture of Right Ventricular Wall Rupture During Inferior Wall Acute Myocardial Infarction

Bradley A. Maron, MD^{a,*}, Kumaran Kolandaivelu, MD, PhD^a, David K. Rhee, MD, PhD^b, Alice K. Bukhman, MD, MPH^b, and Elazer R. Edelman, MD, PhD^a

^aDepartment of Internal Medicine, Division of Cardiovascular Medicine,

^bDepartment of Internal Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts.

Abstract

We present the case of a 57-year-old woman with no previous cardiovascular history in whom fatal right ventricular wall rupture was diagnosed by bedside echocardiography early in the management of an inferior wall acute myocardial infarction.

We report a novel case of right ventricular (RV) wall rupture captured by bedside echocardiography during acute myocardial infarction (AMI).

Case Report

A 57-year-old woman with no previous cardiovascular events presented to a community hospital in Boston, Massachusetts, after 3 days of abdominal discomfort, nausea, and weakness that culminated in syncope at her home. Electrocardiogram obtained at the scene showed ST-segment elevations in leads II, III, aVF, V₅, and V₆ with ST-segment depression in leads I, aVL, and V₁ to V₃ (Figure 1). In the emergency department, troponin I level was 10.1 ng/ml. The patient was administered aspirin 325 mg, clopidogrel 600 mg, intravenous heparin, and intravenous eptifibatide. Because of sustained hypotension, the patient was intubated and an intra-aortic balloon pump was placed. Arteriogram revealed total occlusion of the proximal right coronary artery (Figure 2) with diffuse narrowing of the left anterior descending and left circumflex coronary arteries. Right coronary artery thrombectomy was performed 86 minutes after hospital presentation, resulting in removal of a 5-cm thrombus. Two drug-eluting stents were deployed to the proximal and mid-right coronary artery, resulting in restoration of Thrombolysis In Myocardial Infarction grade 3 flow (Figure 2). Right- and left-sided heart hemodynamic measurements indicated near equalization of end-diastolic atrial and ventricular filling pressures (Figure 3). Left ventriculogram obtained before completion of the procedure, however, revealed only akinesis of the posterior and inferior walls, without evidence of cardiac rupture or impaired diastolic function. The patient was transferred to our facility for further management.

*Corresponding author: Tel: 617-525-4803; fax: 781-634-0375., bmaron@partners.org (B.A. Maron).

On arrival to the coronary intensive care unit, approximately 7 hours after initial presentation to the other hospital, the patient was hypotensive, tachycardic, and anuric despite intra-aortic balloon pump counterpulsation and escalating concentrations of intravenous pressor agents. Electrocardiogram showed persistent 3-mm ST-segment elevation in the inferior limb leads, and blood analysis revealed severe anion gap acidosis and a significantly increased serum lactate level of 12.1 mmol/L. Two-dimensional echocardiogram revealed an underfilled right ventricle and a 1.2-cm circumferential pericardial fluid collection (Figure 4). There was severely decreased biventricular systolic function and globally impaired diastolic filling. An abnormal communication between the right ventricle and the pericardial space was observed, representing RV wall rupture; in addition, a large thrombus was observed in the pericardial space (Figure 4). Repeat echocardiogram with contrast demonstrated evidence of a small ventricular septal defect at the cardiac apex (Figure 4). The patient died 12 hours after admission to our hospital.

Discussion

In the present patient, nonspecific gastrointestinal symptoms persisted for 3 days before hospital presentation and almost surely represented AMI onset. Therefore, the possibility cannot be excluded that RV rupture occurred before hospitalization but was sustained by physiologic hemostasis. Although the use of thrombolytic and antiplatelet medications for AMI treatment is associated with a decrease in the prevalence of cardiac rupture,¹ the magnitude of this benefit may also be somewhat offset by an increase in the risk of intrapericardial hemorrhage and cardiac tamponade that is associated with these therapies.²

RV wall rupture is an exceedingly rare AMI complication, and to our knowledge, this report constitutes the first published account to include echocardiographic images documenting the natural history of this catastrophic event. The preferred treatment strategy in patients with cardiac rupture is immediate mechanical closure by surgical patch repair, although the efficacy of this on outcome remains controversial.^{3,4} In 1 report of nearly 40 cases of successful surgical repair of left ventricular free wall rupture, posterior and anterolateral infarct locations appeared favorable for successful surgical outcome.⁵ The present case underscores the importance of a high index of clinical suspicion for infarct-associated mechanical complications, irrespective of the temporal relation to hospital course.

References

1. Figueras J, Alcalde O, Barrabes J, Serra V, Alguersuari J, Cortadellas J, Lidón R. Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period. *Circulation* 2008; 118:2783–2789. [PubMed: 19064683]
2. Renkin J, de Bruyne B, Benit E, Joris JM, Carlier M, Col J. Cardiac tamponade early after thrombolysis for acute myocardial infarction: a rare but not reported hemorrhagic complication. *J Am Coll Cardiol* 1991; 17:280–285. [PubMed: 1898952]
3. Menon V, Webb G, Hillis L, Sleeper L, Abboud R, Dzavik V, Slater J, Forman R, Monrad E, Talley J, Hochman J. Outcome and profile of ventricular septal rupture with cardiogenic shock after myocardial infarction: a report from the SHOCK Trial Registry. *J Am Coll Cardiol* 2000;36(suppl A): 1110–1116. [PubMed: 10985713]

4. Lemery R, Smith H, Giuliani E, Gersh B. Prognosis in rupture of the ventricular septum after acute myocardial infarction and role of early surgical intervention. *Am J Cardiol* 1992; 70:147–151. [PubMed: 1626498]
5. Chemnitz J, Schmidt T, Wojcik J, Ruschewski W, Kreuzer H, Tebbe U. Successful surgical management of left ventricular free wall rupture in the course of myocardial infarction. *Eur J Cardiothorac Surg* 1991; 5:51–55. [PubMed: 2018650]

Figure 1.

Initial 12-lead electrocardiogram obtained by emergency medical technicians at the patient's home shows ST-segment elevations in leads II, III, aVF, V₅, and V₆. There are ST-segment changes in leads V₁ to V₄. These findings indicate active MI and are suggestive of current inferolateral injury.

Figure 2.

(A) Emergency coronary angiogram demonstrates the presence of acute total occlusion to the proximal right coronary artery. (B) After thrombectomy and placement of drug-eluting stents to the proximal and midright coronary artery, Thrombolysis In Myocardial Infarction grade 3 flow is established throughout the course of the target vessel.

Figure 3.

Hemodynamic tracings indicative of cardiac tamponade. (A) Hemodynamic tracings after cardiac catheterization show severely decreased left ventricular filling pressures. In contrast, right atrial (B) and pulmonary capillary wedge (C) pressures are significantly increased. Atrial and ventricular end-diastolic filling pressures and pulmonary artery pressure (D) approach equalization, suggesting globally impaired diastolic function consistent with cardiac tamponade.

Figure 4.

Bedside echocardiograms demonstrating RV wall rupture. (A) Apical 4-chamber view on presentation to the coronary care unit shows a 1.2-cm circumferential pericardial effusion (*asterisk*). (B) Thirty minutes later, a subcostal short-axis image demonstrates an underfilled right ventricle (RV) (*asterisk*) and fresh thrombus in the pericardial space (*arrowheads*) that is now larger. The site of RV wall perforation is visible (*arrow*). (C) Contrast-enhanced apical 2-chamber view obtained on repeat echocardiography several hours later reveals a ventricular septal rupture at the cardiac apex. Contrast evisceration from the left ventricle (LV) through the apex is visible (*arrow*). A = anterior wall; I = inferior wall; LA = left atrium; P = pericardium; RA = right atrium.