Rare mechanical complication of myocardial infarction: isolated right ventricle free wall rupture

Akcay M, Senkaya E B, Bilge M, Yeter E, Kurt M, Davutoglu V

ABSTRACT

Rupture of the left ventricular free wall is a dramatic complication of acute myocardial infarction (MI) and occurs in about ten percent of patients with fatal acute MI. However, there are limited reports about right ventricular free wall rupture due to MI. In this case report, a patient with isolated right ventricular rupture following MI that was visualised with real-time transthoracic echocardiography is discussed for the first time in the literature.

Keywords: acute myocardial infarction, right ventricle rupture, transthoracic echocardiography.

Singapore Med J 2011;52(1):e7-e9

INTRODUCTION

Right ventricular (RV) rupture is a rare and dangerous complication of acute myocardial infarction (MI). Echocardiographic examination of patients with severe chest pain that is resistant to medical therapy, unresolved or new onset ST-segment elevation on electrocardiography (ECG) and tachycardia is useful in the diagnosis and management of RV rupture. RV rupture diagnosed by echocardiographic examination is discussed in this case report.

CASE REPORT

A 55-year-old male patient was referred to our hospital for rescue percutaneous transluminal coronary angioplasty (PTCA) after acute inferior MI. He had been admitted to a local hospital with severe chest pain four days ago. With the diagnosis of acute inferior MI, thrombolytic therapy was administered at the eighth hour of chest pain. Due to persistent chest pain and ST-segment elevation, the patient was referred to our clinic for rescue PTCA.

On admission, the patient's blood pressure was 110/70 mmHg and the heart rate was 90 beats per minute. Cardiac examination revealed no jugular venous distention or significant heart murmurs. ECG showed

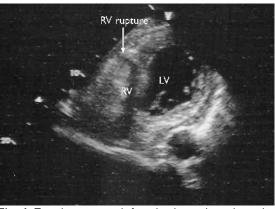


Fig. I Transthoracic apical four-chamber echocardiographic image shows rupture of the right ventricle free wall. LV: left ventricle; RV: right ventricle

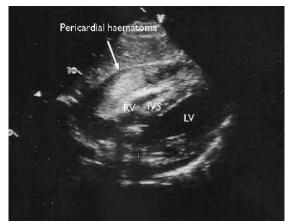


Fig. 2 Subcostal echocardiographic image shows pericardial haematoma.

IVS: interventricular septum; LV: left ventricle; RV: right ventricle

significant Q waves and ST-segment elevation in II, III and aVF leads. Right-sided ECG showed 1-mm STsegment elevation that was prominent in the lead V4R. Two-dimensional (2D) echocardiography revealed left ventricular regional wall motion abnormality with an ejection fraction of 20%–25% and a moderate pericardial effusion localised at the RV free wall. Cardiac rupture was detected at the 0.5-mm echo-free space of the right ventricle (Fig. 1). A flow from the right ventricle to the pericardial space was observed at that region by colour Doppler examination. No RV or right atrial diastolic Department of Cardiology, Ankara Ataturk Education and Research Hospital, Bilkent Avenue, Ankara 06800, Turkey

Akcay M, MD, Medical Officer

Senkaya EB, MD Consultant

Bilge M, MD Professor

Yeter E, MD, Consultant

Kurt M, MD, Medical Officer

Department of Cardiology, Gaziantep University Medical Faculty, University Avenue, Gaziantep 27310, Turkey

Davutoglu V, MD Associate Professor

Correspondence to: Dr Murat Akcay Tel: (90) 312 235 9162 Fax: (90) 312 291 2725 Email: dmuratakcay@ yahoo.com collapse as an indication of cardiac tamponade was noted (Fig. 2). An emergency coronary angiography revealed no hypotension or pulsus paradoxus. The left ventriculogram showed a dilated heart with akinesis of the diaphragmatic wall and hypokinesis of the anterolateral and apical walls. The global ejection fraction was 20%–25% and left ventricular end-diastolic pressure was 18 mmHg. The right coronary artery was occluded, and significant stenoses were observed in the left anterior descending artery, at the origin of the diagonal arteries and in the circumflex artery.

An emergency cardiac operation was planned. Bedside control echocardiography showed restriction of flow from the RV free wall to the pericardium, with the formation of thrombus probably due to the cessation of anticoagulant therapy. After a few seconds, the patient suffered sudden cardiac death due to ventricular fibrillation that was resistant to defibrillation attempts. Pericardiocentesis was also performed under the guidance of echocardiography to rule out possible pericardial tamponade. However, there was no drainage from the catheter that was deployed into the pericardial space. Although defibrillation was performed several times with cardiac massage, the patient was unresponsive to the resuscitation attempts.

DISCUSSION

This is the first case of an isolated RV free wall rupture as a complication of inferior MI after thrombolytic therapy, which was diagnosed by bedside echocardiographic examination. Left ventricular free wall rupture is the second most common cause of mortality in acute MI. There are limited reports on RV free wall rupture,⁽¹⁻⁵⁾ a rare but dangerous complication of acute MI.⁽⁶⁾ RV free wall rupture due to inferior MI is an uncommon finding during transthoracic echocardiographic examination. One of the reasons could be the limited evaluation of the right ventricle due to its crescent shape, substernal location and the presence of a large amount of artifact. The other reason could be the higher intraventricular pressure of the left ventricle as compared to the right ventricle. Figueras et al found that a higher intraventricular pressure in patients with systemic hypertension was one of the predictors of left ventricular rupture. In the same study, they also showed that patients with high ST elevation were at an increased risk for early rupture.⁽⁵⁾

The timing of administration of thrombolytic therapy and thrombolysis in myocardial infarction (TIMI) flow grade after reperfusion therapy has been shown to increase the risk of cardiac rupture after acute MI. Solodky et al reported that the use of thrombolytic therapy was independently associated with the occurrence of cardiac rupture.⁽⁷⁾ Sugiura et al showed that the incidence of free wall rupture was higher in patients with a TIMI flow grade less than 3 after thrombolytic therapy.⁽⁸⁾ Honan et al reported that late administration of thrombolytics increased the risk of cardiac rupture.⁽⁹⁾

In the light of these studies, we are able to speculate that late administration of thrombolytic therapy, accompanied by the failure of thrombolysis, could lead to RV rupture. The younger age and male gender of our patient, however, seem to be inconsistent with the findings in the literature. Although increased age was found to be a risk factor for cardiac rupture in the studies by Purcaro et al,⁽⁴⁾ Figueras et al⁽⁵⁾ and Sugiura et al,⁽⁸⁾ patients under the age of 60 years were also reported by Purcaro et al.⁽⁴⁾ The increased risk of women was reported in the study by Sugiura et al.⁽⁸⁾ However, Purcaro et al⁽⁴⁾ and Figueras et al⁽⁵⁾ both found that there was no gender difference in cardiac rupture. As the patients included in the abovementioned studies had left ventricular rupture, further studies are required in order to clarify the age and gender characteristics of patients with RV rupture.

Echocardiographic examination of patients with severe chest pain that is resistant to medical therapy, unresolved or new onset ST-segment elevation and tachycardia is useful in the diagnosis and management of cardiac rupture. Although echocardiography is a useful method for left ventricle imaging, echocardiographic evaluation of the right ventricle is limited by its crescent shape, substernal location and the presence of a large amount of artifact. In addition, in critically ill patients, the acquisition of optimal precordial images is often disturbed by mechanical ventilation, obesity, the inability of the patient to roll to the left lateral decubitus, as well as by chest or abdominal tubes.^(3-5,10) Due to these reasons, direct signs of cardiac rupture are infrequently visualised by echocardiography. We were able to directly visualise isolated RV free wall rupture and the flow from the right ventricle to the pericardial space by transthoracic 2D and colour Doppler echocardiography in our patient. Sherer et al also reported a patient with RV free wall rupture and cardiac tamponade.(11) However, although the diagnosis was made by transthoracic echocardiography, computed tomography revealed pericardial fluid surrounding the heart.⁽¹¹⁾ In Soriano et al's report, septal rupture with RV wall dissection was directly shown by echocardiography.⁽¹²⁾ Pijls et al reported biventricular free wall rupture with extracardiac left-to-right shunt

after MI, which was directly visualised by transthoracic echocardiography with contrast study.⁽¹³⁾

This case highlights the need to keep in mind the possibility of RV free wall rupture as a mechanical complication of inferior MI as well as the importance of echocardiography as a diagnostic tool for MI mechanical complications. It also demonstrates that RV MI leading to RV free wall rupture may be one of the causes of intractable ventricular fibrillation that is resistant to defibrillation.

REFERENCES

- Reddy SG, Roberts WC. Frequency of rupture of the left ventricular free wall or ventricular septum among cases of fatal acute myocardial infarction since introduction of coronary care units. Am J Cardiol 1989; 63:906-11.
- Oliva PB, Hammill SC, Edwards WD. Cardiae rupture, a clinically predictable complication of acute myocardial infarction: report of 70 cases with clinicopathologic correlations. J Am Coll Cardiol 1993; 22:720-6.
- López-Sendón J, González A, López de Sá E, et al. Diagnosis of subacute ventricular wall rupture after acute myocardial infarction: sensitivity and specify of clinical, hemodynamic and echocardiographic criteria. J Am Coll Cardiol 1992; 19:1145-53.
- Purcaro A, Costantini C, Ciampani N, et al. Diagnostic criteria and management of subacute ventricular free wall rupture complicating acute myocardial infarction. Am J Cardiol 1997; 15; 80:397-405.
- 5. Figueras J, Curos A, Cortadellas J, Sans M, Soler-Soler J.

Relevance of electrocardiographic findings, heart failure, and infarct site in assessing risk and timing of left ventricular free wall rupture during acute myocardial infarction. Am J Cardiol 1995; 76:543-7.

- Woldow AB, Mattleman SJ, Ablaza SG, Nakhjavan FK. Isolated rupture of the right ventricle in a patient with acute inferior wall MI. Chest 1990; 98:484-5.
- Solodky A, Behar S, Herz I, et al. Comparison of incidence of cardiac rupture among patients with acute myocardial infarction treated by thrombolysis versus percutaneous transluminal coronary angioplasty. Am J Cardiol 2001; 87:1105-8.
- Sugiura T, Nagahama Y, Nakamura S, et al. Left ventricular free wall rupture after reperfusion therapy for acute myocardial infarction. Am J Cardiol 2003; 92:282-4.
- Honan MB, Harrell FE Jr, Reimer KA, et al. Cardiac rupture, mortality and timing of thrombolytic therapy: a meta-analysis. J Am Coll Cardiol 1990; 16:359-67.
- Figueras J, Cortadellas J, Calvo F, Soler-Soler J. Relevance of delayed hospital admission on development of cardiac rupture during acute myocardial infarction: study in 225 patients with free wall, septal or papillary muscle rupture. J Am Coll Cardiol 1998; 32:135-9.
- Sherer Y, Levy Y, Shahar A, et al. Survival without surgical repair of acute rupture of the right ventricular free wall. Clin Cardiol 1999; 22:319-20.
- Soriano CJ, Pérez-Boscá JL, Canovas S, et al. Septal rupture with right ventricular wall dissection after myocardial infarction. Cardiovase Ultrasound 2005; 3:33.
- 13. Pijls NH, Fast JH, van der Meer JJ, van der Werf T. Biventricular free wall rupture with extracardiac left-to-right shunt after myocardial infarction. Am Heart J 1988; 115:186-9.