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Case report

Acute temponade due to postinfarction myocardial rupture successfully managed with urgent pericardiotomy

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ARTICLE INFO

Article history:
Received 14 January 2017
Received in revised form
28 March 2017
Accepted 12 June 2017
Available online xxx

Keywords:
Acute myocardial infarction
Cardiac tamponade
Heart rupture
Pericardiotomy

ABSTRACT

Postinfarction myocardial rupture is rare and fatal. A 60-year-old female developed hemodynamic collapse after 2-h acute precordial pains. Acute tamponade and cardiogenic shock were confirmed. The patient underwent urgent pericardiotomy under the aid of intraaortic balloon pump and inotropic infusions. The differential diagnosis of acute chest pain is important. Early diagnosis and prompt surgical intervention of acute temponade are crucial for rescuing the patient.

Introduction

Myocardial rupture is a fatal complication of acute myocardial infarction (AMI). It is not only a serious threat for patients' life, but also a surgeon's nightmare as well. Myocardial free wall rupture is a biphasic clinical course: the acute (emergent) phase is usually accompanied by cardiac tamponade and hemodynamic deterioration; while in the chronic phase after the patient survives the acute (emergent) phase, the patient has to face with additional complications, such as left ventricular pseudoaneurysm that is pending to rupture, cardiac dysfunction and mitral regurgitation, etc. Despite valuable experiences shared from continuously reported cases that were successfully managed, however, evidence-based suggestions regarding the management of myocardial rupture were still scanty [1].

The incidence of myocardial rupture was reported to be 1% of the surviving patients with AMI, but representing 20% of the

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http://dx.doi.org/10.1016/j.crvasa.2017.06.006

Please cite this article in press as: S.-M. Yuan et al., Acute temponade due to postinfarction myocardial rupture successfully managed with urgent pericardiotomy, Cor et Vasa (2017), http://dx.doi.org/10.1016/j.crvasa.2017.06.006

mortality [2]. Myocardial rupture can develop involving the free wall (42.1%), papillary muscle (26.3%), ventricular septum (26.3%), or double structures (ventricular septum plus free wall) (5.3%) [3]. Of the free wall ruptures, half of the cases were identified as a blow-out type with a tear ranging from 1 to 5 cm in diameter, and half, an oozing type, instead [4]. Apical myocardial rupture has not been described until recently Waterhouse et al. [2] presented their unique case that was diagnosed by cardiac magnetic resonance imaging (MRI) and successfully surgically repaired.

Case report

A 60-year-old female, with no significant medical history, presented to the Emergency Department of this hospital with complaints of acute sustained precordial pain for 2 h on August 17, 2015. Electrocardiogram showed the extensive anterior wall AMI (Fig. 1). She was sent for an immediate coronary angiography. However, she had hemodynamic collapses on table in the Digital Subtraction Angiography Room with her systolic pressure decreasing to 50 mmHg. Chest roentgenogram revealed faint heart beating and massive pericardial effusions (Fig. 2). She was diagnosed as cardiogenic shock and temponade secondary to myocardial rupture after AMI. Coronary angiography was given up and intraaortic balloon pump was immediately inserted. Her blood pressure was kept at 90 mmHg. Bedside echocardiography confirmed the presence of tamponade and left ventricular dysfunction with a significantly dyskinetic apex. She was sent to the operating room at once. As she was on the operating table, cardiac arrest occurred twice, but she was resuscitated successfully by closed chest cardiac massages.

Urgent sternotomy revealed a full and tight pericardium. The incisions on the pericardium by pericardiotomy incurred immediate block by massive blots. As a result, a total of 300 mL

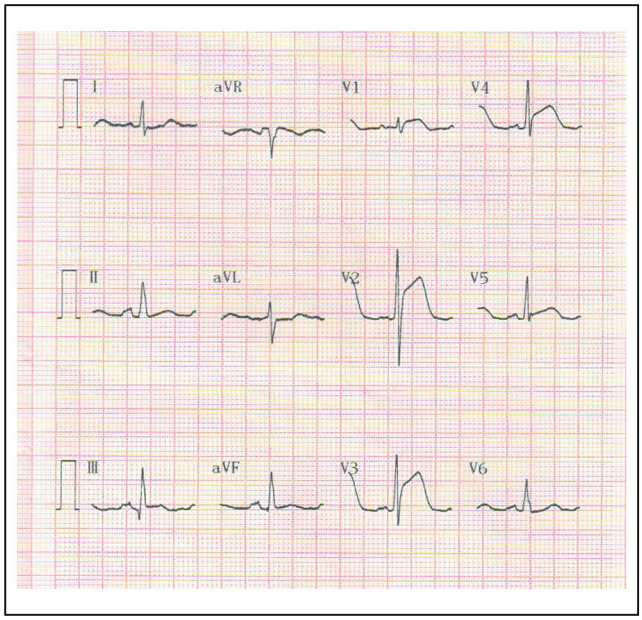

Fig. 1 – Electrocardiogram showed extensive anterior infarction.

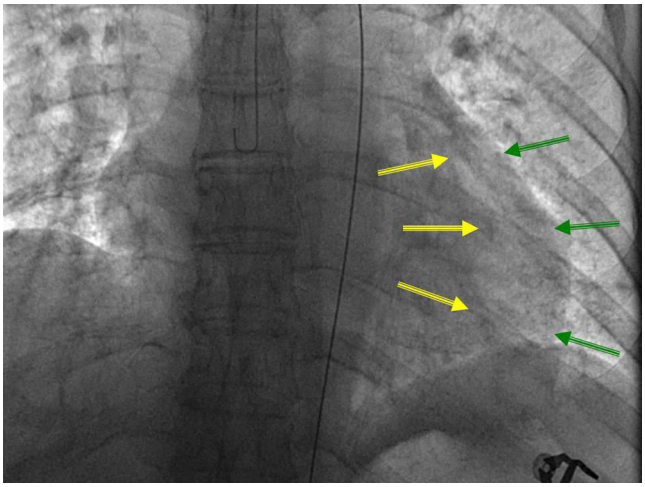

Fig. 2 – Chest roentgenogram showed massive pericardial effusions (between the right- and leftward arrows). Note the bright belt indicating the cardiac silhouette (rightward arrows) and the lateral margin of the pericardial sac (leftward arrows).

blood as well as massive blots were removed. The patient's blood pressure returned normal as soon as the resolution of the tamponade. Lifting up the apex, an exploration revealed a hematoma 2 cm × 2 cm on the apex. No rupture could be visible (even with saline flushing) or palpable (Fig. 3). There was no infarct lesion, either. While doing hemostasis, no active bleeding was found. Thus, no further maneuvers were performed.

After the operation, she was hemodynamically stable with inotropic infusions and intraaortic balloon pumping. She was extubated on Day 3 and weaned from intraaortic balloon pumping on Day 5. She was managed according to acute coronary syndrome. Laboratory investigations showed peaked cardiac enzymes on Day 1, transient hyperglycemia and sustained thrombocytopenia in spite of platelet infusions (Table 1). Echocardiography revealed apical dyskinesis with a minimal thickness of 3 mm and an intramural thrombus measuring 27 mm × 15 mm× 14 mm in the left ventricular cavity, which was expected to be solved under dual anti-platelet

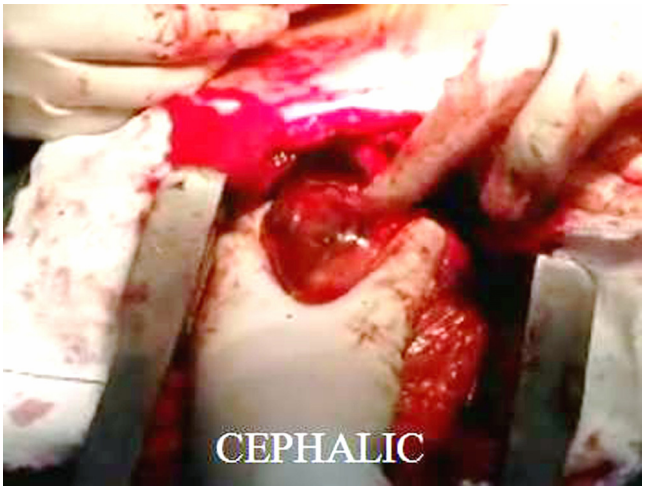

Fig. 3 – Operative view of the hematoma on the apex.

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