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Kasuistika | Case report

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

Shi-Min Yuan[a], Jin-Zao Chen[b], Yao-Hua Yu[c], Liang-Qing Lin[c], Xiang-Kui Fang[c], Jing Lin[c]

[a] *Department of Cardiothoracic Surgery, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian 351100, China*
[b] *Department of Cardiology, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian 351100, China*
[c] *Department of Anesthesiology, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian 351100, China*

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SOUHRN

Poinfarktová ruptura myokardu je vzácné a fatální postižení. U 60leté ženy s akutní prekordiální bolestí přetrvávající po dobu dvou hodin došlo k hemodynamickému kolapsu; byly potvrzeny akutní tamponáda a kardiogenní šok. U pacientky byla provedena urgentní perikardiotomie s použitím intraaortální balonkové kontrapulsace a infuzemi inotropních látek. Při akutní bolesti na hrudi je velmi důležitá diferenciální diagnóza. Pro záchranu pacienta má naprosto zásadní význam časné stanovení diagnózy a rychle provedená chirurgická intervence.

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. An early diagnosis and prompt surgical intervention of acute tamponade are crucial for rescuing the patient.

Address: Shi-Min Yuan, MD, PhD, Department of Cardiothoracic Surgery, The First Hospital of Putian, Teaching Hospital, Fujian Medical University, Putian 351100, China, e-mail: shiminyuan@126.com.
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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. 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 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 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 1–5 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 tamponade secondary to myocardial rupture after AMI. Coronary angiography was given up and intraaortic balloon pump was immediately inserted. Her blood pressure was kept higher at about 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 massage.

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 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 × 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.

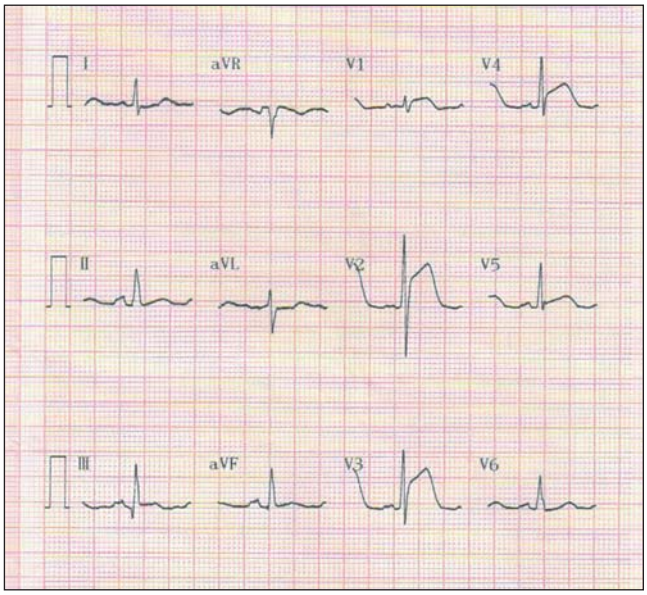

Fig. 1 – Electrocardiogram showed extensive anterior infarction.

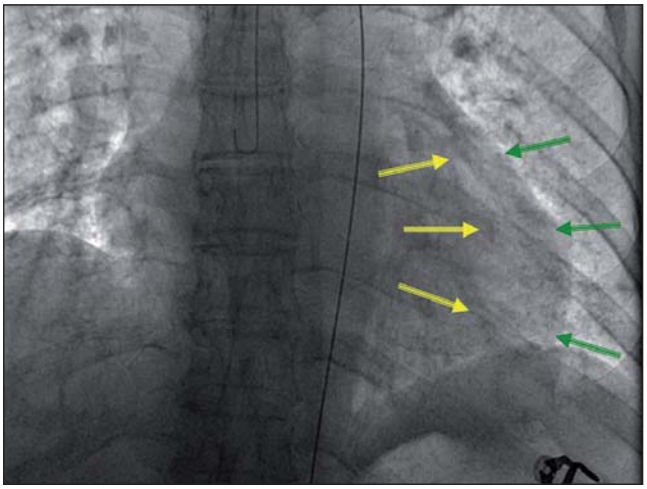

Fig. 2 – Chest roentgenogram showed massive pericardial effusions (between the yellow and green arrows). Note the bright belt indicating the cardiac silhouette (rightward arrows) and the lateral margin of the pericardial sac (leftward arrows).

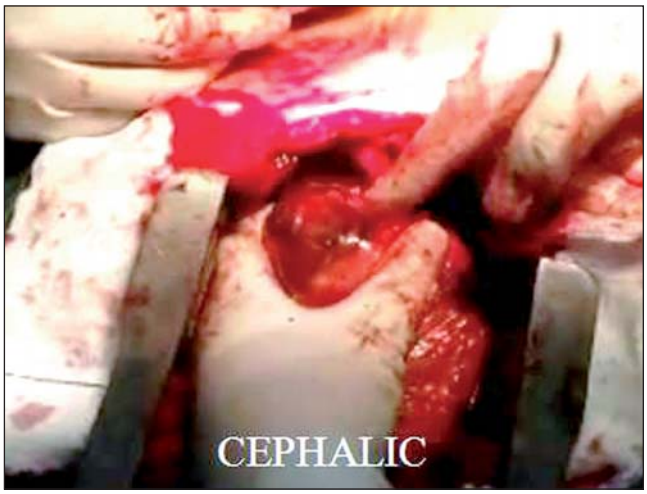

Fig. 3 – Operative view of the hematoma on the apex.

Table 1 – Laboratory tests.

Date	LDH (313–618 U/L)	CPK (26–174 U/L)	Amylase (30–110 U/L)	CPK isoenzyme MB (0–32 U/L)	Troponin I (0–0.06 μg/L)	Pro-BNP (0–900 pg/mL)	Glucose (3.89–6.11 mmol/L)	Leucocyte (× 10^9/L)	Platelet (125–350 × 10^9/L)	C-reactive protein (0–8 mg/L)
Day 0	1819	178	381				14.26	12.17	82	
Day 1	3908	7255	1982		218.04	2528	9.86	11.34	70	
Day 2	1271	2971	1287	33	53.44		9.49	9.28	54	
Day 3							6.74	9.28	43	
Day 5			179					7.08	92	66.2
Day 6					6.6	1899	5.78	10.17	79	27
Day 11	361	29	101	7	0.34	2244	7.02	7.21	60	17.4

CPK – creatine kinase; LDH – lactate dehydrogenase; Pro-BNP – N-terminal pro brain-type natriuretic peptide.

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 2, 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 × 15 × 14 mm in the left ventricular cavity, which was expected to be solved under dual anti-platelet therapy. She was planned for further coronary angiography and surgical interventions in a later stage. Due to her economic hardship, she declined definite surgical operations and lost for follow-up since her discharge.

Discussion

Myocardial rupture predisposes to develop in patients with AMI of the anterior wall, especially of extensive anterior wall [5]. The onset symptoms of the patients may be severe chest pain, arrhythmia, heart failure, tamponade or cardiogenic shock [6]. Yuan et al. [3] reported myocardial enzymes, including aspartate aminotransferase, lactate dehydrogenase, creatine kinase, creatinine kinase MB isoenzyme and troponin I, peaked on day 2 after myocardial rupture. The peak values of the myocardial enzymes were 4–11 folds higher in non-survivors than in survivors of myocardial rupture. The myocardial rupture could be disclosed by ventriculography, transthoracic echocardiography and MRI [2]. Cardiac compression and tears of the ventricular wall may be visualized by echocardiography except for hypokinesis [4].

In most patients, immediate surgery is necessary. Free wall rupture is treated by resecting the infarcted area and closing the rupture zone with Teflon or Dacron patches or by using of biologic glues. Ventricular septal defects can be closed directly or with a patch. Papillary muscle rupture is usually treated with mitral valve replacement. Surgical repair of pseudoaneurysm and (or) coronary artery bypass surgery are often warranted. Despite the aggressive treatments, the operative mortality remains high [4].

For oozing type myocardial ruptures, direct suture and either synthetic or autologous patch with glue or suture are conventional techniques for myocardial rupture repair [7]. Sutureless repair with TachoComb was praised to be the most effective technique for myocardial rupture repair with rapid hemostasis and without the need of cardiopulmonary bypass [8]. However, sutureless techniques may be associated with some problems, such as difficulty in locating and exposing the coronary arteries in coronary artery bypass grafting in a later stage, risks of recurrent rupture, left ventricular pseudoaneurysm formation and mitral regurgitation [9]. There have been several reports describing the non-surgical treatment of oozing type myocardial rupture, by percutaneous injection of fibrin-glue into the pericardium [10], by combined prolong rest, blood pressure control and β-blocker [11], or by percutaneous cardiopulmonary support [12].

It has been estimated that thrombus formation in patients with AMI was independently associated with base-

line infarct size [13]. By cardiovascular MRI, a thrombus was found in 8.8% of the patients with AMI, 88.2% of which had a thrombus resolution under dual anti-platelet therapy (a loading dose of clopidogrel 300 or 600 mg and thereafter 75 mg/day in addition to an initial loading dose of aspirin followed by 80–100 mg/day) [13].

Mechanisms of thrombocytopenia in acute coronary syndrome can be: glycoprotein IIb/IIIa inhibitor-induced, heparin-induced or thienopyridine derivatives-induced [14]. Moreover, myocardial rupture is likely a stress that induces hyperglycemia. A close correlation between hyperglycemia and increased risks of congestive heart failure, cardiogenic shock, and mortality after AMI has been found [15]. Hyperglycemia enhances myocardial Txnip expression, possibly through reciprocally modulating p38 MAPK and Akt activation, leading to aggravated oxidative stress and subsequently, amplification of cardiac injury following myocardial ischemia-reperfusion injury [16]. Other possible mechanisms that hyperglycemia contributes to poor prognosis of AMI might be increased inflammatory, electrophysiological alterations, free fatty acid production, altered coagulation status and impaired microvascualr flow, etc. [15]. In addition, brain-type natriuretic peptides can be shown to be diagnostic and prognostic in both acute coronary syndrome and detecting the sequelae of post-infarction cardiac dysfunction [17]. Plasma brain-type natriuretic peptide levels increased without symptomatic and hemodynamic changes and reached their highest level immediately before cardiac rupture [18].

This particular case report refers an oozing type of ventricular rupture. Even if there was no continuous bleeding discovered, the hematoma in the apex of the heart was probably result of the bleeding spot revealed by postoperative echocardiography for apical dyskinesia and ventricular wall thinning, which was already clotted. Therefore, this rupture site should be secured using any of sutures technique to prevent possible recurrent rupture and recurrence of bleeding especially the patient was treated with dual anti-platelet therapy. Nevertheless, we could only perform a rescue operation by reliving tamponade in this near-basic-level hospital that is lack of any materials for definite operations, including synthetic patches, biologic glues and facilities for coronary artery bypass grafting.

Conclusions

This article reports a successful lifesaving procedure for a critical patient with myocardial rupture after AMI. Timely diagnosis is a prerequisite for subsequent correct treatment. We advise an urgent surgical intervention for the patients with tamponade and a rapid hemodynamic deterioration. However, the causative relations between hyperglycemia and thrombocytopenia remain to be further determined.

Conflict of interest
None declared.

Funding body
None.

Ethical statement
Authors state that the research was conducted according to ethical standards.

Informed consent
Informed consent was obtained from the patient before discharge.

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