CASE REPORT

An 8-year follow-up of a patient with acute myocardial infarction complicated with ventricular rupture and cerebral thrombosis treated conservatively: a cohort study

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SUMMARY

Cardiac rupture is a complication of myocardial infarction (MI) with extremely high mortality. Poor prognosis is usually predicted in conservatively treated patients. The treatment for cardiac rupture and thrombosis is in conflict. We reported an MI case of cardiac rupture and cerebral thrombosis using a conservative method. This case indicated that (1) It is noteworthy that patients suffering from the subacute form of rupture may remain haemodynamically stable. This type of patients may benefit from a conservative treatment without anticoagulation or antiplatelet at the onset of cardiac rupture. (2) Thirty days following cardiac rupture, it is safe to use warfarin anticoagulation for cerebral thrombosis treatment. The patient suffered from acute MI, cardiac rupture and subsequently cerebral thrombosis. We treated this patient in a conservative manner and the patient remained alive for 8 years. This case suggested that it may be safe to use warfarin anticoagulation treatment for cerebral thrombosis 30 days following cardiac rupture.

BACKGROUND

Cardiac rupture is one of the most devastating complications of myocardial infarction (MI) accounting for up to 20% of mortality. Cardiac rupture after acute MI often involves the free wall of the left ventricle (LV), the interventricular septum or papillary muscles. The incidence, timing of occurrence, prognostic factors, clinical features and outcomes of acute MI complicated by LV free wall rupture in both the prethrombolytic and thrombolytic therapy eras have been debated extensively. Although thrombolytic therapy has been shown to reduce the incidence of cardiac rupture,¹ this therapeutic management for patients with acute MI also may accelerate early cardiac rupture. We reported a case on the importance of conservative therapy in this complication.

CASE PRESENTATION

A 32-year-old male patient was admitted to our hospital with a chief complaint of aggressive dyspnoea, accompanied with vomiting, diarrhoea and jaundice for 2 weeks prior to the admission. His main cardiovascular risk factors included hypertriglyceridaemia. He was first admitted to a local infectious medical centre to treat his apparent jaundice. However, laboratory tests were negative for hepatitis (hepatitis

B, C, D and E). Further the echocardiogram displayed massive pericardial effusion while pericardiocentesis showed bloody fluid. The qualitative troponin T test was positive. ECG revealed Q waves in V1–V4 leads (figure 1). The primary diagnosis for the patient was subacute MI (anterior wall) and LV free wall rupture. The patient was then transferred to the cardiology department of our hospital. Upon arrival, his blood pressure was 108/83 mm Hg with pulmonary wet rales audible at the base of the left lung. Heart valve murmur and neurological signs of focal lesion were negative. He had a leucocyte count of 15.1 G/l and a platelet count of 459 G/l. Serum levels of urea nitrogen were 8.73 mmol/l, creatine 137 µmol/l, total bilirubin 33.2 µmol/l, direct bilirubin 21 µmol/l, alanine transaminase 73 U/l, C reactive protein 207.9 mg/l, fibrinogen 8.493 g/l and erythrocyte sedimentation rate 74 mm. The patient underwent pericardiocentesis to remove 200 ml bloody fluid which contained erythrocyte 350 000 million/l, leucocyte 2700 million/l with percentages of polykaryocyte and monocyte at 59 percent; and 41%, respectively. The mucoprotein was positive in the bloody effusion without coagulation. Echocardiogram displayed extensive LV anterior, apical wall motion abnormalities and a ventricular thrombus located at the apex of LV (figure 2). Ejection fraction of LV was 38% and movement of LV apical wall was in contradiction. Echocardiogram further revealed massive pericardial effusion. The patient underwent ^{99m}Tc single-photon emission CT which revealed hypoperfusion in the anterior, apex and inferior wall, and ventricular aneurysm formed in the rest of the images (figure 3). His systolic blood pressure was maintained below 110 mm Hg. The patient was treated with β-blocker, nitrates, atorvastatin calcium and diuretics. However, no aspirin, clopidogrel, low-molecular-weight heparin and warfarin were administered.

Thirty days following cardiac rupture, the patient complained of visual acuity loss with poor vision. Ophthalmic examination revealed visual acuity of both the eyes at 0.1. There was a solid exudate beside the inferior temporal vascular arch in the left eye and a cotton wool spot near the temporal inferior vascular arch in the right eye. MRI results suggested presence of acute cerebral infarction of caudate nucleus, corpus callosum and occipital cortex on the right side. Transoesophageal echocardiography showed pericardial effusion was limited

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Figure 1 ECG upon admission revealed Q waves in V1–V4 leads and inverted T waves in V5 lead.

in the lateral and posterior wall of the LV. Pericardial effusion was almost completely absorbed. The patient was then treated with warfarin with a target international normalized ratio (INR) of 2.5–3.5 for approximately 4 weeks. His visual acuity recovered to 0.4 and 0.8, respectively, for the left and right eye. Subtle opacity was seen in the bilateral lens. Examination of the visual field exhibited bilateral homonymous hemianopsia on the right side with macular sparing. The patient was later discharged without dyspnoea. Ten months later, the patient was admitted again to our hospital for coronary-artery angiography procedure. The results indicated diffuse thrombosis in the anterior descending artery middle segment, diagonal branch and right coronary artery (figure 4). Three years later, the patient was admitted to our cardiac surgery department for dyspnoea and coronary-artery bypass graft (CABG) procedure, ventricular aneurysm repair and ventricular thrombus surgical removal were performed. Tissue biopsy revealed collagen proliferation and glassy degeneration (figure 5). The patient was then discharged without chest pain and dyspnoea. Eight years later, the patient exhibited clinical manifestation of heart failure with a moderate exercise tolerance under treatment with ACE inhibitor, β -blocker, nitrates, atorvastatin calcium, diuretics and aspirin.

TREATMENT

The primary diagnosis for the patient was subacute MI (anterior wall), and LV free wall rupture. The patient was treated with

β -blocker, nitrates, atorvastatin calcium and diuretics. However, no aspirin, clopidogrel, low molecular weight heparin and warfarin were administered. The second diagnosis for the patient was acute cerebral infarction. He was then treated with warfarin with a target INR of 2.5–3.5 for approximately 4 weeks.

Three years later, the patient was admitted to our cardiac surgery department for dyspnoea and CABG procedure, ventricular aneurysm repair and ventricular thrombus surgical removal were performed. Tissue biopsy revealed collagen proliferation and glassy degeneration. Eight years later, the patient exhibited clinical manifestation of heart failure with a moderate exercise tolerance under treatment with ACE inhibitor, β -blocker, nitrates, atorvastatin calcium, diuretics and aspirin.

OUTCOME AND FOLLOW-UP

Eight years after LV free wall rupture, the patient is still alive.

DISCUSSION

The most devastating complication of MI may be cardiac rupture. Although it carries an incidence of merely 1–4% following a heart attack, it accounts for up to 20% of mortality associated with infarction.² Approximately 90% of the ruptures occur during the first 2 weeks following infarction, with 22% occurring within the first 24 h.³

Free wall rupture is usually divided into two distinct forms based on presentation, namely acute and subacute.⁴ The acute form (also referred to as blowout) involves cardiac arrest resulting from severe hypotension and electromechanical dissociation secondary to cardiac tamponade.⁵ The subacute form of free wall rupture consists of moderate to several pericardial effusion and hypotension of varied severity associated with sinus bradycardia or nodal rhythm.³ The present case appeared to be of cardiac rupture that had presumably occurred after the onset of MI. It is noteworthy that patients suffering from the subacute form of rupture may remain haemodynamically stable.⁶ Surgical resection is often recommended for free ventricular wall rupture patients⁷ while poor prognosis was reported in conservatively treated patients.⁸ The patient was treated in a conservative manner without aspirin, clopidogrel and low weight heparin anticoagulation or antiplatelet at the beginning of cardiac rupture. At the same time, serum total number of platelet and concentration of fibrinogen were high. These factors may prompt the formation of thrombus to stop ventricular bleeding.

Figure 2 Transthoracic echocardiograph: the two-dimensional view of the left ventricular apex depicting left ventricular free wall rupture, thrombosis in the left ventricle apex and excessive circumferential pericardial fluid. FWR, ventricular free wall rupture; TH, thrombosis; PE, pericardial effusion.

Figure 3 Myocardial ^{99m}Tc single-photon emission CT in rest of the image. Hypoperfusion was found in the apex, anterior and inferior wall (arrowheads). (A) Apex, (B) anterior and (C) inferior wall. VLA, vertical long-axis view; SA, short-axis view; and HLA, horizon long-axis view.

The rupture site was protected either by a thrombus on the endocardium side or by pericardial adhesion.⁹

The patient suffered from MI and cardiac rupture 30 days later. He felt visual acuity injury and MRI indicated cerebral thrombosis. The thrombosis is likely to originate from the LV. Interestingly, treatment strategy for cardiac rupture and cerebral thrombosis is completely opposite, making a rather difficult choice for the physician with regard to the use of warfarin for anticoagulation treatment. If warfarin was used early on, there is a high propensity of ventricular bleeding. Paradoxically, prevention of warfarin anticoagulation may promote the recurrence of cerebral thrombosis.

Three years later, CABG procedure was performed on the patient and he was discharge uneventfully. To the best of our knowledge, a survival of up to 8 years in a conservatively treated MI patient with ventricular free wall rupture and cerebral thrombosis was not previously reported.

CONCLUSIONS

(1) It is important to note that patients suffering from the subacute form of rupture need to remain haemodynamically stable. Such patients can be treated conservatively without aspirin, clopidogrel and low weight heparin anticoagulation or antiplatelet at the beginning of cardiac rupture to avoid mortality. (2) Another critical point that can be drawn from this case is that it

Figure 5 H&E staining in ventricular aneurysm tissue biopsy. The slide displays fibrous tissue proliferation and hyaline degeneration.

is safe to use warfarin anticoagulation for cerebral thrombosis purpose patient suffered cardiac rupture 30 days later. We still need more cases to support this issue.

Figure 4 Coronary angiography of the left and right coronary arteries 3 years after myocardial infarction and cardiac rupture. (A) Thrombosis (TH) in the left anterior coronary artery (arrowhead). (B) TH in the right coronary artery (arrowhead).

Learning points

- ▶ Myocardial infarction cardiac rupture acute cerebral infarction anticoagulation treatment.
- ▶ It is noteworthy that patients suffering from the subacute form of rupture may remain hemodynamically stable. This type of patients may benefit from a conservative treatment without anticoagulation or antiplatelet at the onset of cardiac rupture.
- ▶ We should do echocardiogram examination before anticoagulation or antiplatelet treatment at the onset of acute myocardial infarction to make sure the security of the patient.
- ▶ Thirty days following cardiac rupture, it is safe to use warfarin anticoagulation for cerebral thrombosis treatment.

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Contributors XL collected the images, QZ prepared and amended the manuscript.

Competing interests None.

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