Left Ventricular Oozing Rupture following Acute Myocardial Infarction


Abstract

We describe the case of an 85-year-old woman in whom pericardiocentesis, prolonged bed rest and blood pressure control were performed without surgery to successfully treat an oozing-type myocardial rupture due to myocardial infarction.

Key words: acute myocardial infarction, oozing-type cardiac rupture, pericardiocentesis

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Case Report

An 85-year-old woman was admitted to our hospital 4 h after the onset of severe chest pain. Upon admission, she was alert, with a systolic blood pressure (SBP) of 110 mmHg and a heart rate (HR) of 80 beats/min. Electrocardiography showed ST elevation in leads II, III, aVF (Fig. 1). Echocardiogram revealed severe hypokinesis of the left ventricular (LV) inferior-posterior wall from the base to the apex, no thinned or echo-dense wall was observed, and there was no pericardial effusion. Acute myocardial infarction (AMI) was confirmed on the basis of creatine kinase (CK) elevation. We provided the patient with anti-platelet agents (100 mg aspirin and 150 mg clopidogrel), and she was taken to the catheterization laboratory. Coronary angiogram showed 100% obstruction of the distal right coronary artery (RCA) and no significant stenosis of the left coronary artery (Fig. 2). Subsequently, direct percutaneous coronary intervention (PCI) was performed for the lesion, and during this procedure 8,000 IU of heparin was administered. The RCA was successfully recanalized without mechanical complications and the patient was transferred to the coronary care unit. We then started the intravenous administration of heparin at an infusion rate of 500 U/h.

One hour later, the patient’s level of consciousness changed to somnolence following repeated emesis. Suddenly, SBP dropped to about 60 mmHg, and HR was 116 beats/min. Echocardiogram revealed cardiac tamponade with an oozing-type myocardial rupture (Fig. 3). Pericardiocentesis was immediately performed using a 4F long sheath. Blood fluid was obtained (200 mL), vital signs recovered to a BP of 110/60 mmHg and HR of 80 beats/min and she became alert. On day 2 after admission, we started angiotensin II receptor blocker and beta-blocker agents to control BP at approximately 100/70 mmHg.

Peak CK was 6,300 IU/L (10 h after onset of chest pain). The pericardial discharge from the drainage tube gradually diminished to 40 mL on day 3 and 10 mL on day 4. After day 6, no echo-free space was observed in the pericardium, and therefore the pericardial drain was removed. The patient resumed taking 100 mg aspirin on that day. Bed rest was maintained for 12 days, and ambulation was started at 14 days. One month later, coronary multi-slice computed tomography (MSCT) demonstrated the stent site at the RCA lesion without reobstruction (Fig. 4) and no pseudoaneurysm. Therefore, the patient was discharged without any complications and has remained asymptomatic.

Discussion

Cardiac rupture following AMI is considered to be sudden...
Figure 1. Electrocardiogram on admission showed ST-segment elevation in leads II, III, aVF and ST-segment depression in leads I and aVL.

Figure 2. Left: emergent coronary angiography shows 100% obstruction of the distal right coronary artery. Right: an angiogram after PCI using a bare metal stent (4.0×20 mm Liberte Stent)

Figure 3. Echocardiography showed an echo-free space (arrows) 1 hour after PCI; Left: para-sternal view, Right: subxiphoidal view. RV: right ventricle, IVC: inferior vena cava

and unanticipated (1). This complication of AMI occurs more often in women, hypertensive patients and patients > 60 years old who have sustained a first infarction (2, 3). It is generally considered that there are 2 distinct types, based
on the clinical course: blow-out type and oozing type. There have been few reports of patients with oozing-type cardiac rupture who were treated without surgical repair. Murata et al (4) reported the effectiveness of percutaneous fibrin glue therapy and Masaki et al (5) reported the successful treatment of oozing-type cardiac rupture with percutaneous cardiopulmonary support without the need for surgical repair. Pericardiocentesis resulted in remarkable hemodynamic improvement, and prolonged rest and blood pressure control led to a favorable outcome in our case. Figueras et al (6) treated 19 selected free wall rupture patients with prolonged bed rest, BP control and pericardiocentesis. These patients had a higher incidence of inferior and lateral wall infarction. They suggested that medical management might be of particular value in patients with a lateral or infero-posterior AMI or in those at very high surgical risk, such as those with a large infarct area or those >75 years old. Since the present case had high surgical risk [pleak CK (6,300 IU/L) and 85 years old] and relatively stable hemodynamics without catecholamine infusion, pericardiocentesis, prolonged bed rest and BP control may have contributed to the favorable outcome.

Recently, MSCT has been reported to provide high diagnostic accuracy for detecting coronary stenosis and occlusion (7). In this case, MSCT was performed at 1 month after AMI, and there was no coronary stenosis after PCI. Since this technique also provides useful cross-sectional information and is more sensitive for the evaluation of the morphology of LV, LV aneurysm and intracardiac thrombi may be easy to identify. Therefore, MSCT, a non-invasive tool can provide variable information at follow-up after AMI.

Conclusion

Surgical intervention should be considered for patients in whom cardiogenic shock persists after the removal of pericardial effusion. The findings in the present case suggest that an oozing-type cardiac rupture with hemodynamic recovery after pericardiocentesis might respond favorably to conservative treatment and evaluation with a non-invasive tool, such as MSCT.

References


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