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Case Report

Myocardial rupture after small acute myocardial infarction in the absence of coronary artery disease

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ABSTRACT

A 73-year-old woman with a past medical history of hypertension suffered a cardiac arrest. After successful resuscitation, she was hypotensive and tachycardic and the ECG showed ST elevation in the inferior and lateral precordial leads. Coronary angiography did not show evidence of obstructive coronary artery disease. A bedside echocardiogram demonstrated a large pericardial effusion with signs of cardiac tamponade. The echocardiogram and subsequent aortic root angiography did not reveal evidence of dissection. Pericardiocentesis removed 700 cc of bloody fluid with relief of tamponade. A few minutes later the patient again arrested. Fluid was again drained but she suffered recurrent hemodynamic collapse and could not be resuscitated. Autopsy revealed a small transmural myocardial infarction with external rupture and hemopericardium. There was only mild coronary artery disease without evidence of plaque rupture. This case illustrates that mild coronary artery disease and a small myocardial infarction can lead to catastrophic mechanical complications.

1. Introduction

Free wall rupture in the setting of transmural myocardial infarction (MI) has an incidence of <2% in the modern reperfusion era [1]. Mortality, however, remains as high as 39–77% [2, 3]. We present a case of free wall rupture after myocardial infarction in the absence of coronary artery disease, as documented by angiography and histology.

2. Case report

A 73-year-old female with a past history of hypertension presented as a transfer from another facility following cardiopulmonary arrest in her physician's office. She was successfully resuscitated, but during transport she developed pulseless electrical activity. Upon arrival to our emergency department, her EKG demonstrated ST elevation in the inferior and precordial lateral leads (Fig. 1a). In the cath lab, she was hypotensive and tachycardic and required vasopressor support. Her initial troponin I was elevated at 6.14 ng/mL (Normal 0.0–0.029 ng/mL). Diagnostic angiography showed no obstructive coronary artery disease (Fig. 1b and c). Left ventricular (LV) end diastolic pressure was elevated at 24 mmHg. An urgent bedside echocardiogram demonstrated normal LV function with a medium to large sized pericardial effusion with coagulum. There was evidence of right ventricular (RV) collapse and echodense material adherent to the RV in the pericardial space, confirming a diagnosis of cardiac tamponade (Fig. 1d). Suprasternal and parasternal long axis views by echocardiogram did not show an intimal flap, and aortic root angiography did not show evidence of dissection (not shown).

Pericardiocentesis was performed with removal of 700 cc of bloody fluid. This resulted in relief of tamponade with normalization of blood pressure and heart rate. Cardiovascular surgery was consulted regarding management of the residual pericardial hematoma. A transesophageal echo showed no aortic dilatation, aortic valve insufficiency, or dissection of the ascending aorta. A few minutes later, the patient again experienced cardiopulmonary arrest with pulseless electrical activity. The pericardial drain was aspirated with removal of 200 cc of bloody fluid, with return of spontaneous circulation. Recurrent arrest and hemodynamic instability ensued. Bedside discussion was held with the family regarding possible emergent exploratory cardiovascular surgery. The family decided to stop resuscitation efforts given the patient's previously expressed wishes. She expired moments later.

Post mortem examination revealed an acute small (less than 3 mm thick on short axis) transmural myocardial infarction (MI) with free wall rupture along the posterior septum and posterior wall, with the hemorrhagic tract extending from the posteromedial papillary muscle to the epicardium (Fig. 2a, b). Histology showed coagulation necrosis with prominent neutrophilic infiltrate (Fig. 2c), indicative of an infarction approximately 24 h old adjacent to the rupture tract. Examination of the right coronary artery revealed mild atherosclerotic disease, but

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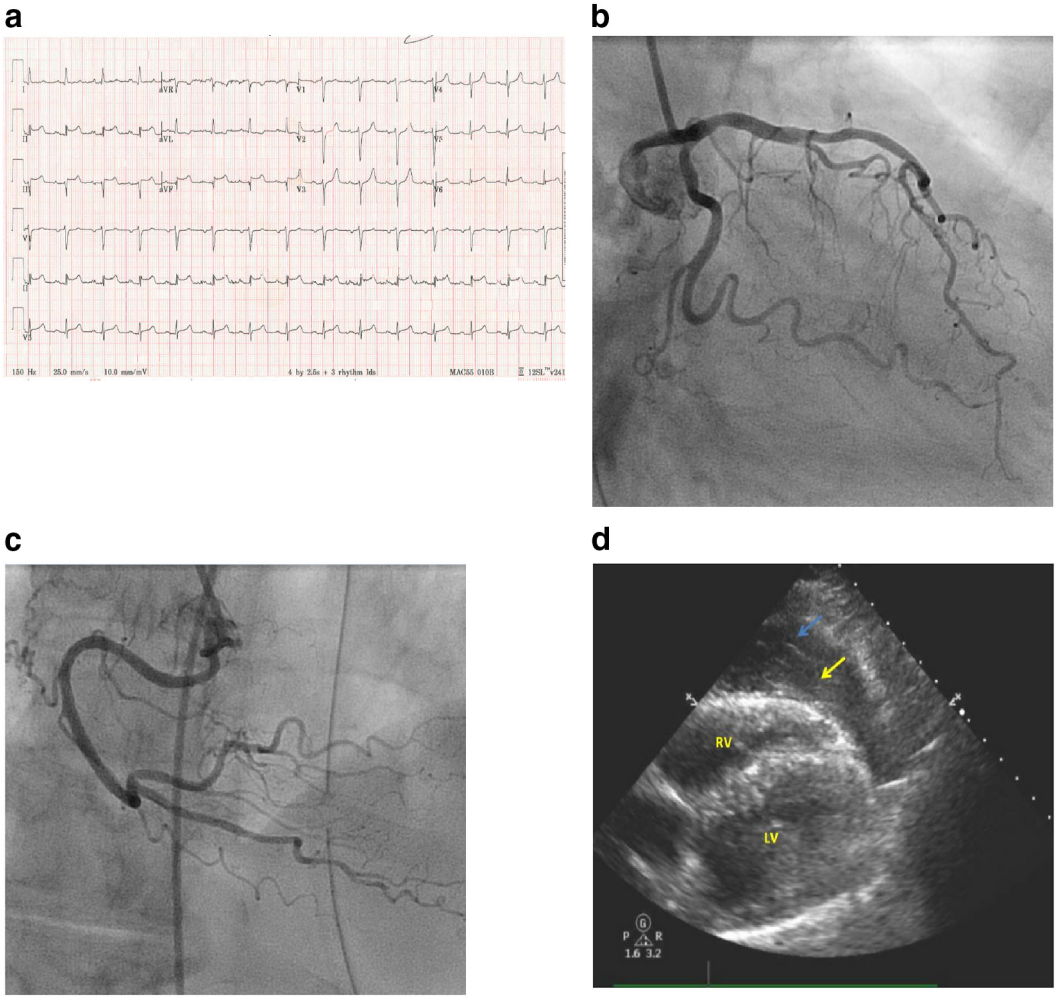

Fig. 1. (a) Electrocardiogram with ST segment elevation in the inferior and lateral precordial leads. (b and c) Angiography of the left and right coronary arteries without significant stenosis. d. Subcostal echocardiographic image showing a large pericardial effusion with coagulum in the pericardial space. Blue arrow -pericardial fluid. Yellow arrow - coagulum. RV - right ventricle. LV - left ventricle.

no significant plaque burden or evidence of recent plaque rupture to explain the myocardial infarction. Histology demonstrated thickened arterial media in the vessels adjacent to and overlying the myocardial infarction without evidence of atherosclerotic plaque (Fig. 2d).

3. Discussion

This case demonstrates the importance of suspecting myocardial rupture with tamponade in the setting of hemodynamic instability even in the absence of obstructive coronary disease. Two remarkable findings of this case are the lack of a culprit atherosclerotic plaque to cause an acute myocardial infarction, and the small size of the infarct that caused the myocardial rupture.

Possible etiologies of a transmural infarction in this case include spontaneous recanalization of plaque rupture, prolonged coronary vasospasm, or thrombus. Pathologic support for any of these etiologies was lacking. Spontaneous recanalization, previously defined as thrombolysis in myocardial infarction anterograde ≥2 flow on the preintervention angiogram, is known to occur in up to 20% of acute myocardial infarctions, however rarely without evidence of angiographic stenosis [4, 5]. There were no intracardiac masses to raise suspicion of a thromboembolic event. Coronary vasospasm was not seen on angiography and was not diagnosed during autopsy. Vasospasm would be less likely in an older individual with hypertension as a risk factor for atherosclerotic disease.

The correlation between acute myocardial infarction and rupture was initially described by Wessler et al. in 1952. Pathologic substrate for rupture included a fresh coronary artery occlusion, and a recent transmural myocardial infarction that was poorly supplied by collaterals and lacking in fibrosis [6].

Myocardial infarction with angiographically normal coronary arteries (MINOCA) has been described and thought of as rare [7, 8]. A recent literature review of MINOCA suggested a prevalence of 6%, but typical myocardial infarction by cardiac MRI accounted for only 24% of these. Alternative explanations for suspected myocardial infarctions with normal coronaries included structural myocardial dysfunction (i.e., myocarditis, Tako-tsubo cardiomyopathy), coronary artery spasm, and thrombophilia disorders [9]. The case described above demonstrated a true transmural infarction without angiographic or histologic evidence of structural disease, thrombosis, or plaque rupture. In the SHOCK Trial Registry of 1048 patients with acute MI and cardiogenic shock, 2.7% of patients had free wall rupture or tamponade, and of this group, all demonstrated a culprit vessel on diagnostic angiography [2]. In an older necropsy study by Mann et al., 107 of 108 examined patients with rupture had either obstructive atherosclerotic disease or occlusive thrombus, although those dying from rupture had less extensive atherosclerotic disease when compared to those dying from acute myocardial infarction without rupture [10]. Isolated case reports suggesting rupture without major vessel obstruction have been reported, but most have suggested vessel thrombus or branch vessel coronary artery disease [11–16].