



Case report

Saddle coronary embolism precipitates flash pulmonary edema

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ABSTRACT

Coronary artery embolism is an infrequent but important cause of acute myocardial infarction. We present a rare case of a large saddle embolism in the coronary vasculature which led to an acute myocardial infarction and consequently flash pulmonary edema. Despite intensive medical management, our patient did not survive.

1. Introduction

Coronary artery embolism is a relatively rare cause of acute myocardial infarction, however it has been associated with a higher risk of death [1–3]. It has been implicated in about 3% of acute coronary syndromes [4]. Studies reveal that patients may present with symptoms suggestive of atherosclerotic disease, however angiographic findings often reveal normal coronary arteries [5]. Between 1 and 12% of patients with a myocardial infarction have no significant stenosis (less than 30%) or abnormality of the coronary arteries [6]. Although flash pulmonary edema may present following acute coronary ischemia, it has not been previously reported in the setting of an acute myocardial infarction from a large saddle embolism [7]. To our knowledge, this is the first reported case in literature. (see Fig. 1, Image 1–3)

2. Case report

An 86-year-old Caucasian female presented to the Emergency Department (ED) in acute respiratory distress, shortly after being discharged from the inpatient unit. She had been hospitalized for a complicated urinary tract infection, which responded well to appropriate therapy. Her medical history was significant for hypothyroid disorder and mild dementia from traumatic brain injury.

Upon arrival to the ED, she complained of substernal chest pain and shortness of breath. Vital signs revealed a blood pressure of 105/74 mm Hg, heart rate 132, respiratory rate 26, oral temperature of 36.7 °C, with an oxygen saturation of 88% on room air. Physical examination demonstrated a rapid heart rate with bilateral crackles on auscultation of the lungs. A 12-lead electrocardiogram (EKG) showed sinus tachycardia with ST-segment depressions in the precordial leads V2 to V5. Arterial blood gas was significant for hypoxemia with mild metabolic acidosis and chest radiography demonstrated moderate pulmonary

edema and small pleural effusions bilaterally. Laboratory tests revealed a white blood cell count of $26.89 \times 10^3/\mu\text{L}$, and an initial troponin I of 0.38 ng/mL (normal 0.00–0.04) followed by a subsequent rise to 64.50 ng/mL.

Treatment was instituted for flash pulmonary edema secondary to non-ST segment elevation myocardial infarction. The patient was placed on non-invasive ventilation, and initiated on aspirin, clopidogrel, atorvastatin, antibiotics, furosemide, nitroglycerin, and heparin. She was admitted to the intensive care unit and underwent transthoracic echocardiography which showed moderate mitral valve regurgitation and stenosis. Subsequent left heart catheterization revealed a large saddle embolism involving two vessels of non-dominant circumflex coronary artery without associated stenosis. Intensive medical management was recommended per cardiology consultation. However, her respiratory status continued to deteriorate. She was not a candidate for invasive mechanical ventilation as she had previously expressed do-not-resuscitate or intubate instructions. Unfortunately, she sustained cardiopulmonary arrest which led to her death.

3. Discussion

Coronary artery embolism is an infrequent cause of myocardial infarction, and has an unknown prevalence, partially due to the difficulty of diagnosis in acute situations [8]. It is also associated with poor long-term survival and has an increased risk of death [3]. It has been proposed that the low frequency of coronary artery emboli may be explained by the size difference between the calibre of the aorta versus that of the coronary arteries, anatomical positioning of the coronary artery immediately distal to the aortic valve, increase blood flow in this area, and filling of the coronary artery during diastole [1].

Coronary artery emboli is most commonly attributed to underlying nonvalvular atrial fibrillation, but it may also be a consequence of

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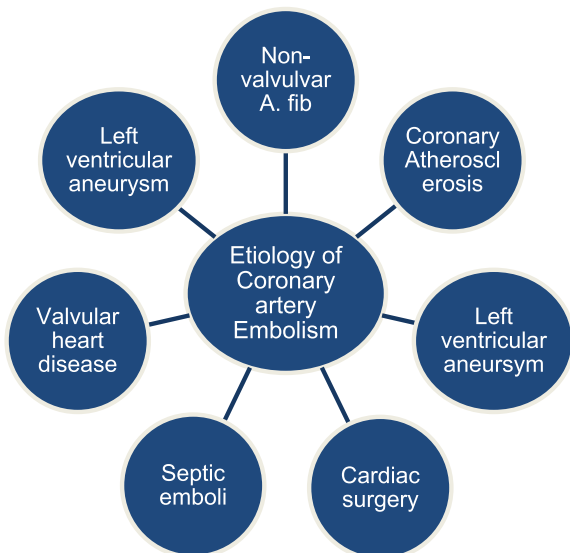


Fig. 1. Etiology of Coronary artery embolism.

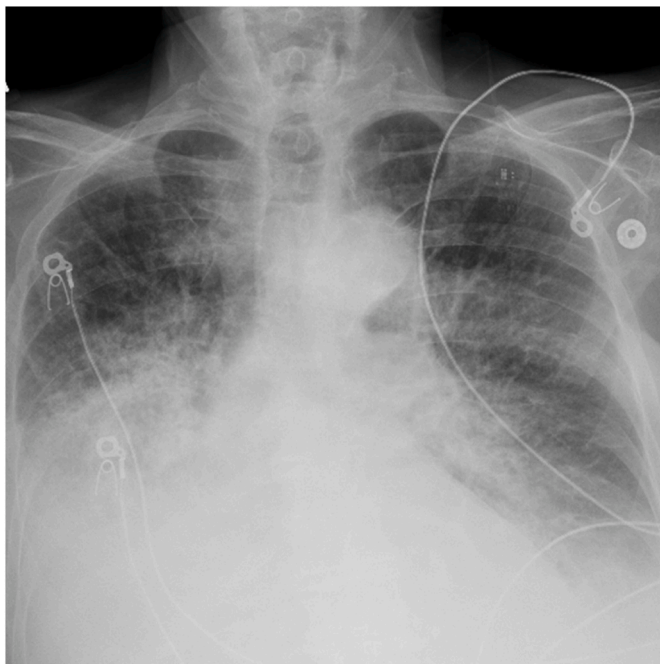


Image 1. Chest radiograph revealing bilateral pulmonary opacities, suggestive of flash pulmonary edema.

cardiac surgery, coronary atherosclerosis, left ventricular aneurysm, non-infected thrombi on prosthetic valves, septic emboli from infective endocarditis, cardiac tumors, and valvular heart disease [8–14]. Paradoxical emboli, arising from systemic circulation, may also be implicated in patients with a patent foramen ovale [1]. In most cases, emboli lodge in the left coronary artery circulation which may lead to transmural myocardial infarction [5].

Although there are a few case reports that attribute acute myocardial infarction to coronary artery embolism, flash pulmonary edema arising as a consequence has not been previously reported. Our case demonstrates a rare instance of flash pulmonary edema in the setting of an acute myocardial infarction, due to a large saddle embolism involving two coronary vessels. The underlying cause of coronary artery embolism in our patient may be attributed to mitral valvulopathy seen on the



Image 2. Image from cardiac catheterization demonstrating coronary artery embolism.

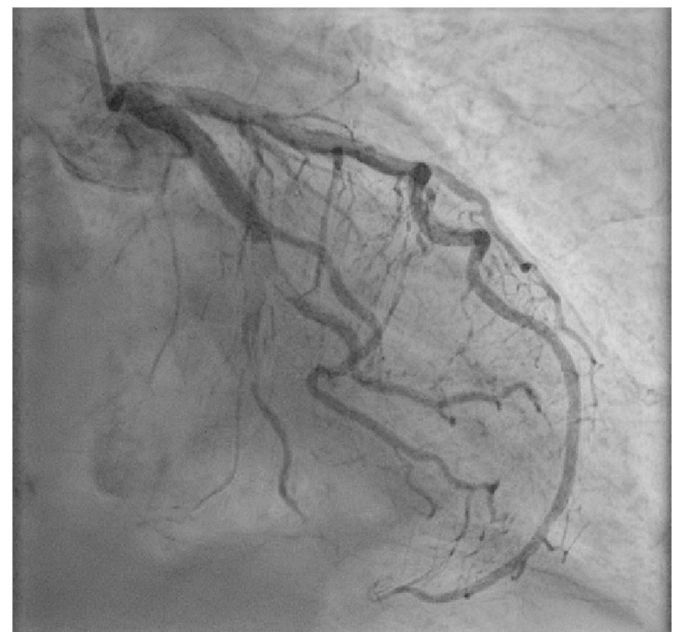


Image 3. Image from cardiac catheterization demonstrating coronary artery embolism.

transthoracic echocardiogram.

The treatment of coronary artery embolism lacks consensus, as both intravenous thrombolysis and percutaneous coronary intervention have been proposed [2]. In the presence of an acute myocardial infarction, emergent percutaneous intervention is imperative, and may necessitate both thrombus aspiration and stent placement. However, in patients with a simple emboli, removal of the thrombus without concurrent stent placement has been suggested [2]. Preventative measures in high risk patients include anticoagulation therapy to decrease recurrence [2].

4. Conclusion

The presentation of an acute myocardial infarction from a saddle coronary artery embolism is rare, however it can prove fatal as in our case report. Patients may present in acute respiratory distress secondary to flash pulmonary edema and concurrent circulatory shock. Diagnosis is corroborated by angiographic findings and thromboembolic risk factors in the presence of an acute myocardial infarction [10]. Treatment may include anticoagulation, percutaneous coronary intervention, and thrombus aspiration. Early recognition and appropriate therapy should be instituted early to prevent further decline.

Declaration of competing interest

No conflicts of interest to disclose.

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