

Coronary Embolism Causing ST Elevation Myocardial Infarction Complicated by Ventricular Septal Rupture

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Abstract

Introduction: Atherosclerosis is the most common etiology of myocardial infarction (MI), yet rarely, MI may be caused by an embolus to one of the epicardial arteries.

Case: A 66-year old female, with very few risk factors for coronary artery disease, presented clinically at Emergency Department with vomiting and ventricular fibrillation on cardiac monitor. After successful resuscitation, she was noted to have ST-elevation in the anterior and inferior walls of the heart. Cardiac catheterization showed total occlusion of the mid-left anterior descending artery with the rest of the coronary tree normal. Percutaneous coronary intervention was done however patient had no-reflow phenomenon, indicating that perfusion to the affected myocardium was still compromised. Follow-up examination upon admission at the coronary care unit showed new onset grade 3/6 holosystolic murmur at the left sternal border. Her 2D echo showed abnormal color flow across the muscular ventricular septum, indicative

of ventricular septal rupture. There was also note of calcifications in the aortic valve and calcified plaques along the walls of the aortic root, which were possibly the source of the coronary embolus. Serial monitoring of the ST elevation was then done and these showed resolution of the ST changes. Patient was also hemodynamically stable despite not undergoing intervention for the ventricular septal rupture because of financial constraints. She remained stable for the rest of her hospital stay and was eventually discharged and advised to consult with a cardiologist upon her return to her home country for definitive repair of ventricular septal rupture.

Conclusion: Coronary artery embolism should always be a consideration for patients who developed myocardial infarction in the setting of an angiographically normal coronary arteries.

Keywords: coronary embolism, myocardial infarction, ventricular septal rupture

Introduction

Myocardial infarction (MI) is most commonly caused by atherosclerotic plaque rupture that rapidly progresses to form an occluding thrombus in the affected coronary artery. Rarely, total occlusion of an epicardial artery may occur despite the absence of atherosclerosis. Coronary embolism is one of the etiologies of MI in non-atherosclerotic coronaries and the clinician should always have a high index of suspicion for this condition in order to identify the potential source of the clot. We report a case of an anterior wall ST-elevation myocardial infarction (STEMI) caused by coronary embolism from calcified plaques in the aortic root and complicated

by two other uncommon events: no-reflow phenomenon and ventricular septal rupture.

Case

Patient is a 66-year-old Filipino female, who consulted our emergency department due to vomiting associated with epigastric discomfort. These symptoms started one day prior to admission and was associated with epigastric discomfort. These symptoms persisted until the morning of the next day of admission. Hence, the patient was then brought to the emergency room (ER) of St. Luke's Medical Center for consult.

The patient denied any angina, exertional dyspnea, palpitations nor episodes of syncopal attack. Review of systems revealed intermittent back and knee pains which were relieved with non-steroidal anti-inflammatory drugs (NSAIDs) on a per need basis. She was also noted to have lapses in short-term memory for about a year and was told by her primary care physician that this may be due to beginning Alzheimer's disease; however patient had not been evaluated by a neurologist.

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Past medical history revealed osteoarthritis and hypertension for more than five years. Personal and social history is unremarkable. She is not diabetic and has no previous history of cerebrovascular disease or MI. She is a non-smoker and a non-alcoholic drinker.

She was seen awake, alert, ambulatory at the emergency room, with unremarkable vital signs. The rest of the physical examination was noted to be normal. She was initially managed as a case of gastroesophageal reflux disease but on arrival at ER she also started to complain of chest discomfort; hence a 12-lead ECG and Troponin T were also ordered by the ER physician. Before the diagnostics were completed, patient suddenly developed ventricular fibrillation. Advanced cardiac life support (ACLS) protocol was immediately started and return of spontaneous circulation was noted approximately 15 minutes after start of resuscitation. The ECG done after return of spontaneous circulation showed sinus rhythm with ST-elevation in the anterior and inferior leads (Figure 1).

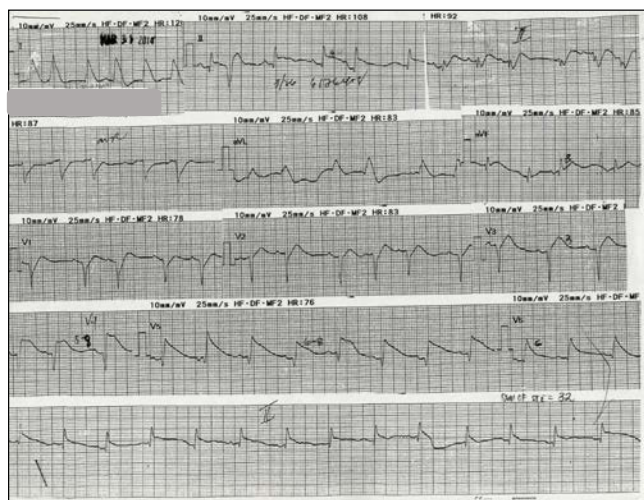


Figure 1. 2-Lead ECG after the return of spontaneous circulation

The patient underwent emergency coronary angiography which showed a type III LAD with total occlusion of the mid-left anterior descending (LAD) artery (Figure 2). The rest of the coronary tree was normal (Figure 3). A drug-eluting stent was successfully placed but there was note of no-reflow phenomenon after procedure (TIMI flow grade 0-1). Intracoronary verapamil 3.0 mg IV and Heparin 7000 units IV were given. Tirofiban bolus followed by continuous infusion was administered in addition to the regimen for acute coronary syndrome.

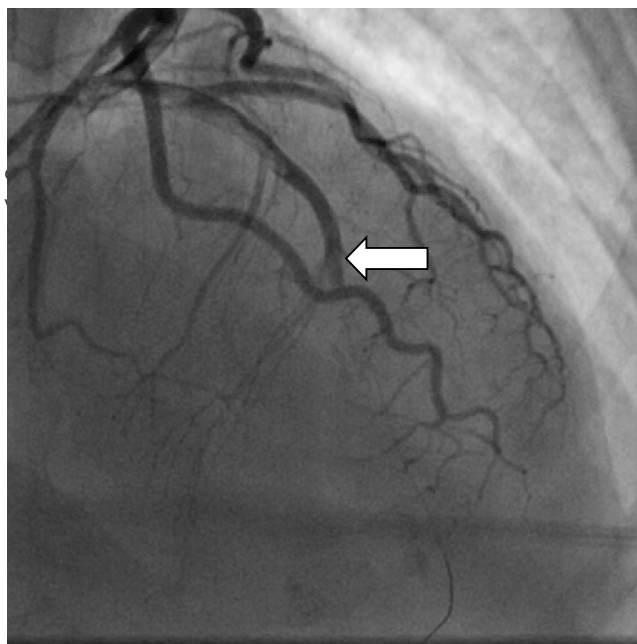


Figure 2. Angiogram showing an abrupt cut-off (arrow) in the mid-left anterior descending artery, indicating total occlusion

Upon transfer to the Coronary Care Unit (CCU), the patient remained hemodynamically stable but was noted to have a grade 3/6, non-radiating, holosystolic murmur on the left lower sternal border. A 2Decho done shortly after patient's arrival at the CCU showed a muscular type shunting (Figure 4). Since the examination during patient's initial presentation was normal, the new onset of this murmur was attributed to a ventricular septal rupture rather than a congenital defect. The ejection fraction was preserved at 56% (Simpson's method) with eccentric left ventricular hypertrophy and LVH, hypokinesia of the anterior interventricular septum (IVS) from base to apex and dyskinesia of the entire apical segment. Left atrial diameter was dilated with increased left atrial volume index (LAVI) of 32 ml/m² but no thrombus was noted on the left atrium. There were also calcifications in the aortic valve, as well as the anterior and posterior walls of the aorta. The possibility of a coronary emboli from these calcified plaques was entertained, likely from the calcified plaques in the aorta and the coronary sinus, because of the finding of an isolated occlusion in the mid-LAD while the rest of the coronary tree was normal ID. Coronary calcium score was also noted to be zero.

She was advised closure of the septal defect, however, patient was financially constrained for the procedure at the time. Medical management was then maximized. Repeat ECG post-intervention showed 68% decrease of the ST elevation in the anterior and inferior leads and STE-index was also less than 50%, indicating that patient may still had



Figure 3. Angiographically normal right coronary (white arrow) and left circumflex arteries (red arrow)

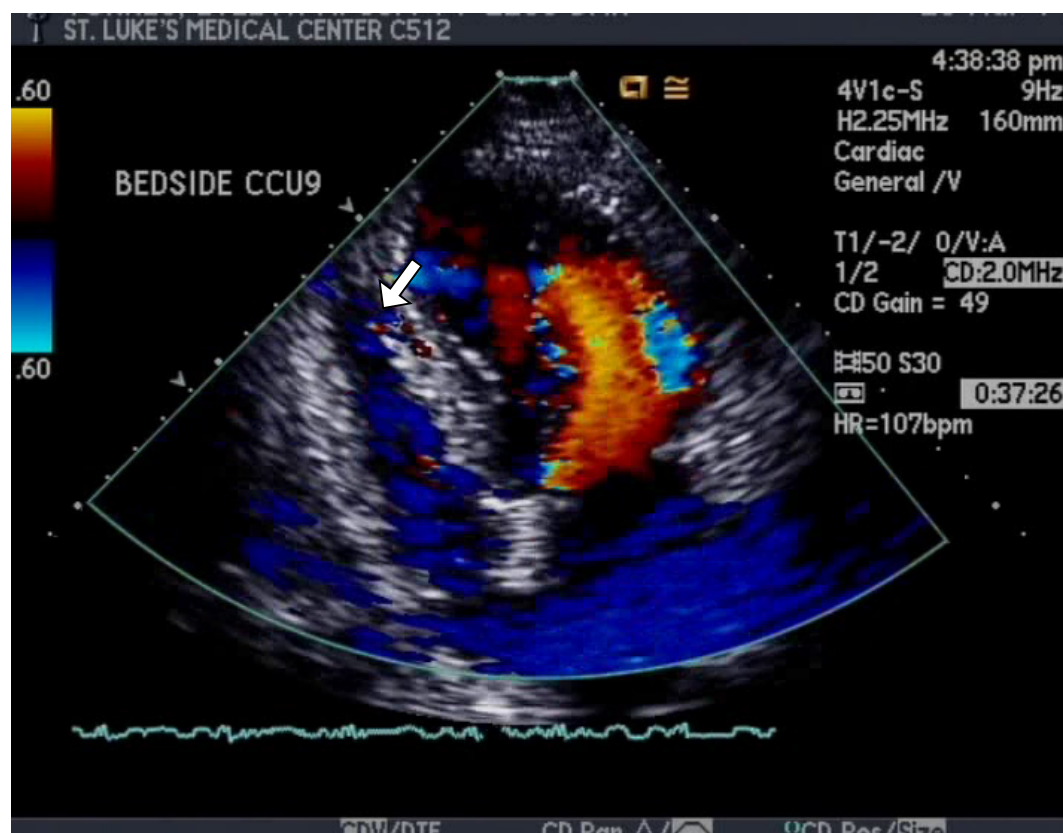


Figure 4. Abnormal color flow seen across the muscular portion of the interventricular septum (arrow)

partial microvascular obstruction during the first few hours after angiography. Serial ECGs, though, showed further decline in the ST elevation. She was transferred out of CCU 72 hours post-angiography. During the rest of her hospital stay, patient had episodes of dyspnea associated with bibasal rales but this was successfully managed with diuretics. She remained hemodynamically stable and was eventually discharged and advised to consult with a cardiologist upon her return to the United States for follow-up care and definitive management of the ventricular septal rupture (VSR).

Discussion

Coronary embolism is a rare cause of myocardial infarction; nevertheless, it remains as an important differential diagnosis, particularly in patients who present with MI with angiographically normal coronary arteries (MINCA).¹⁻³ The incidence of coronary artery embolism has been estimated to be about 13% and has a predilection for the left coronary system. Most of the case reports of coronary artery embolism were from patients with mechanical heart valves which developed thrombi due to suboptimal anticoagulation. Other causes were infective endocarditis, atrial fibrillation, paradoxical embolism and, in one report, thromboembolism during ablation for AF.⁴⁻⁷

The complications of coronary embolus depend on two major factors: 1) the size of the embolus and 2) the size of lumen of the artery in which it becomes impacted. There is greater chance that the embolus will lodge distally to a small coronary arterial segment if it is smaller. The more distal the occlusion, the less is the treatment and management of the coronary emboli.¹ In this patient, we surmised that the embolus is likely large in size since it lodged in the mid-LAD which had a nominal vessel size of 4.0 mm. It has also been observed from previous reports that most emboli involve the left coronary system which could be due to preferential flow of blood into the artery related to aortic valve morphology. Again, the angiographic findings in this patient is compatible with this observation.

Coronary embolism should always be suspected especially in the context of sudden chest pain in patients with valvular prosthesis, chronic AF, infective endocarditis, dilated cardiomyopathy, intracardiac shunts, mural thrombi and hypercoagulable states. In cases wherein patients presented with ST-elevation MI but coronary arteries are angiographically normal except for the occluded segment, coronary embolism may be one etiology of myocardial infarction though this is still controversial.²⁻³ In our patient, the whole coronary tree except for the mid-LAD was normal, making the diagnosis of coronary artery embolus very likely. A very likely source of the embolus could be

the calcifications in the aortic valve and walls of the aortic root since there was no LA thrombus noted, no history of infective endocarditis and no valve prosthesis.

There are no clear guidelines on the management of coronary embolism. In previous case reports, treatment of the embolism involved combination of aspiration followed by infusion of GpIIb/IIIa inhibitors with or without LMWH. In one case, PCI was attempted only after failed aspiration of the clot, however, due to the distal location of the occlusion, angioplasty was also unsuccessful.^{1,4-7} In this patient, atherosclerosis could still not be totally ruled out during the angiography and the calcifications in the aortic valve and aortic root were not visualized during the procedure since a left ventriculogram was not done. Hence PCI with drug eluting stent was the immediate management done. One of the limitations of angiography is that it only provides two-dimensional image of the lumen. In a patient who appears to have normal coronary arteries, intravascular ultrasound (IVUS) will often unveil extraluminal disease and can further reveal the specific etiology.² Unfortunately, IVUS was not available at our institute. However, the diagnosis of coronary artery embolism could be supported by the zero calcium score of the patient. In a review by Schmermund and Erbel⁸, coronary artery calcium (CAC) was measurable in patients who had acute coronary syndrome and those with a negative CAC had minimal or no atherosclerosis as confirmed by IVUS. The same was mentioned in a review by Greenland and Bonow who stated that a CAC score of zero is highly suggestive of the absence of atherosclerotic plaque or significant luminal obstructive disease.⁹

Percutaneous coronary intervention was successful in restoring patency to the mid-LAD however it was highly likely that the procedure itself caused distal embolization of thrombus fragments resulting to no-reflow phenomenon. Again, there are no clear cut guidelines in managing this phenomenon, though GpIIb/IIIa inhibitors have proven to be useful, as was the case in our patient.

The patient's course was complicated by ventricular septal rupture (VSR), which is another rare sequelae of myocardial infarction. Ventricular septal rupture is seen to be more common in women than in men, in single-vessel disease and in coronaries with TIMI flow 0-1 with absent collaterals. Other associated factors are older age, hypertension, non-smoking and LAD as the affected territory. In a study by Suzuki, et al,¹⁰ six patients who had early myocardial rupture complicating STEMI had initial TIMI 0-1 and persistence of ST segment elevation post-PCI despite TIMI 2-3. Though the number of patients in that study was small, nevertheless it suggested that failed myocardial reperfusion is an indicator of risk for developing early myocardial rupture. Hence, our patient had all the risk factors for VSR.

Conclusion

In summary, we presented a case of a 66-year-old female with very few risk factors for ischemic heart disease who developed myocardial infarction in the setting of an angiographically normal coronary arteries and zero calcium score. Coronary artery embolism should always be a consideration for these patients and a search for a source of the embolus should be carried out. This case also underscores the importance of being alert for mechanical complications of MI and those that are related to reperfusion of the culprit artery, especially if a patient presents with specific risk factors predictive of these sequelae.

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