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Coronary Embolism Presenting as NSTEMI in a Patient with Splenectomy

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INTRODUCTION

The coronary arteries generally are protected from embolism as they arise at acute or right angles from the coronary cusps.[1] However, coronary embolism (CE) is a recognized and important cause of non-atherosclerotic acute coronary syndrome. The incidence of acute coronary syndrome (ACS) secondary to coronary embolism is reported to be around 3%.[1-3] The most common causes of CE include atrial fibrillation, infective endocarditis, iatrogenic causes, and prosthetic valve thrombi.[4] No consensus agreement has been established regarding treatment of ACS related to coronary artery embolism (CE). Irrespective of the treatment, clinical consideration of the source of embolism is very important for successful management and recurrence prevention.

Unclear causes of CE may prompt a hypercoagulable workup. Few cases of thromboembolism have been reported with a link to hereditary spherocytosis (HS), particularly in patients with a prior history of splenectomy.[5-9] We present a case of a patient who was transferred from an outside hospital showing non-ST elevation myocardial infarction (NSTEMI) and who subsequently was diagnosed with CE.

CASE REPORT

A 50-year-old Caucasian male with a past medical history of HS diagnosed in childhood presented to the emergency department as a transfer from an outside emergency department with worsening mid-sternal chest pain starting at rest, radiating to the left arm, and accompanied by nausea. He had a splenectomy performed for refractory hemolysis as a child. He had a prior history of tobacco abuse for 20 years, with smoking cessation noted for the past three years. At encounter, the patient had no signs of heart failure.

Physical examination was unremarkable. Blood pressure on presentation was 150/100 mmHg (equal in both arms) and his heart rate was 67 beats/min. Electrocardiogram (EKG) showed normal sinus rhythm with no acute ST changes (Figure 1). The troponin level at an outside hospital was 5.7 ng/ml and was repeated four hours later at initial presentation to the emergency department with a trended upward value of 12.5 ng/ml. Chest x-ray did not show any pulmonary edema or widening of the mediastinum. Bedside echo showed a preserved LV function and no pericardial effusion. A point of care cardiac ultrasound was not performed. The patient was loaded with 325 mg aspirin and was started on a heparin drip. Despite sublingual nitroglycerin and morphine, the patient's chest pain did not resolve. Repeated EKG showed normal sinus rhythm with no ST elevation.

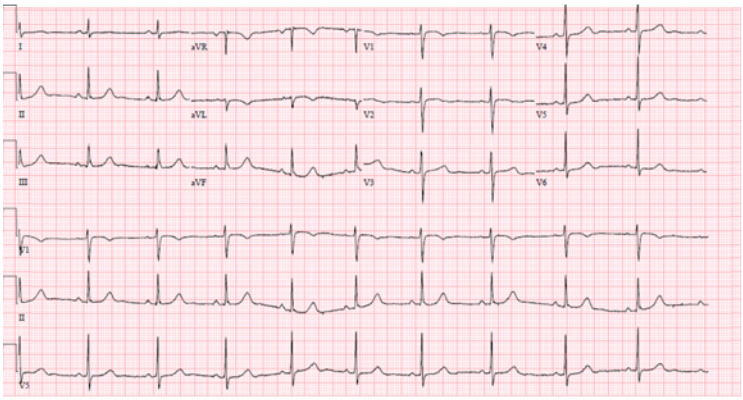

Figure 1. Electrocardiogram on patient presentation to the emergency department showed normal sinus rhythm with no acute S-ST changes.

The patient was taken to the cardiac catheterization lab. Angiography showed a 90% filling defect involving the distal left main coronary artery (LMCA) with an extension to the proximal left anterior descending (LAD) and proximal left circumflex (LCx; Figure 2). Distal LAD, LCx, and RCA showed no significant coronary artery disease. The described finding was most consistent with a coronary artery embolus rather than a ruptured coronary plaque.

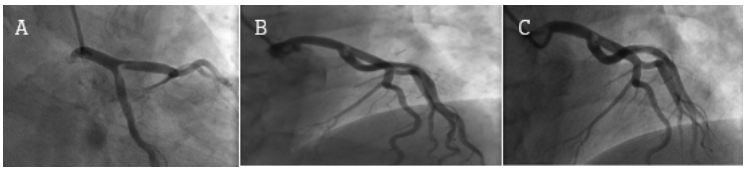

Figure 2. Multiple angiographic views of the distal left main filling defect involving the bifurcation extending into the left anterior descending (LAD) and proximal left circumflex arteries.

Given the location of the thrombus and the risk of distal extension, mechanical thrombectomy was avoided. The patient continued to be stable and reported an improvement in chest pain. The treatment plan was to continue medical management because the lesion was non-flow limiting, embolic in nature, and there was a lack of coronary artery disease.

The patient was admitted to the cardiac care unit and was started on an IV heparin drip, dual antiplatelet therapy with aspirin, and IV tirofiban drip. Over the next 24 hours, the patient's chest pain completely resolved and the troponin value trended downward. Tirofiban was discontinued after 24 hours. Telemetry showed no ventricular tachycardia (VT), atrial fibrillation, or heart block. The patient's lab work was unremarkable.

Transthoracic echocardiography (TTE) showed a normal ejection fraction of 55 - 60% without any wall motion abnormalities. Bubble study on TTE suggested an intra-atrial shunt. Transesophageal echo (TEE) revealed an atrial septal defect (ASD) measuring 0.9 x 0.6 cm and was confirmed using echo colored Doppler and an agitated saline bubble study (Figure 3). There was no left atrial appendage thrombus (LAA), valvular vegetations, or left ventricular thrombus.

Doppler ultrasound of lower extremities was negative for deep vein thrombosis (DVT). A repeat coronary angiogram done after 72 hours of medical management showed complete resolution of the thrombus without any residual stenosis (Figure 4). The patient was discharged on apixaban, aspirin, and atorvastatin.

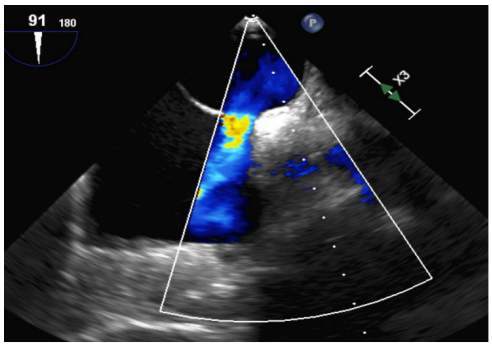

Figure 3. Transesophageal echocardiogram view showing the atrial septal defect.

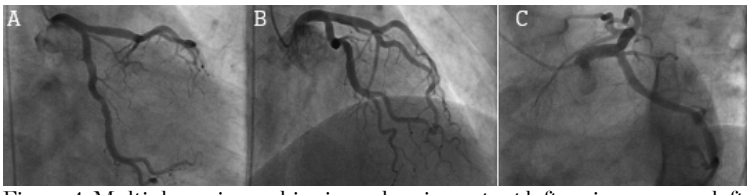

Figure 4. Multiple angiographic views showing patent left main coronary, left anterior descending, and left circumflex arteries.

The patient was seen in the interventional cardiology clinic a month after discharge and remained in good health. He was offered the option to pursue closure of the ASD but deferred the decision at that time.

DISCUSSION

The most common etiology of NSTEMI is an atherosclerotic plaque rupture.[10] However, non-atherosclerotic causes should be considered in absence of atherosclerotic risk factors. Differential diagnosis in this case would include CE secondary to paradoxical venous emboli in the setting of an ASD, paroxysmal atrial fibrillation, hypercoagulable condition, recreational drugs, septic embolism, and, less commonly, tumors such as myxomas. There are no guidelines for management of NSTEMI due to coronary embolism. In this case, mechanical thrombectomy had been deemed high risk for distal embolization. Additionally, the patient was clinically stable, and the lesion was embolic in nature and non-flow limiting. Therefore, the care plan was to proceed with medical management.

The causes of CE can be classified according to the location and mechanism of its origin.[1] CE can arise from a cardioembolic source or can be paradoxical emboli arising in the venous system (e.g., from DVT) and crossing into the left-sided cardiac chambers through a patent foramen ovale or ASD. Additionally, CE also can arise at the time of diagnostic or therapeutic interventions through iatrogenic means.

HS patients with subsequent splenectomy are at higher rates of arterial and venous thrombosis compared to their HS counterparts without splenectomy as reported by an observational study.[11] The previous study estimated an occurrence of five times the risk of arterial events in individuals with HS who had splenectomy compared to those who had an intact spleen. The pathogenesis of these thromboembolic events after splenectomy is likely multifactorial. Persistence of abnormally shaped red blood cells, disturbance and activation of the endothelium, platelet activation, endothelial alteration, abnormal lipid profiles, and a reduction in the removal of circulating procoagulant factors from the bloodstream after splenectomy are all potential risk factors.[12]

Aside from the need for anticoagulation, there are no standard guidelines in the treatment of CE patients presenting with ACS. Management options in CE were based on the patient's hemodynamic stability. Treatment options include thrombectomy, intracoronary thrombolysis, percutaneous coronary intervention, or medical management alone.[1]

Identifying the cause of CE is important as the results could determine clinical management and long-term treatment to prevent recurrence. TEE was used to confirm the presence of ASD and to rule out LAA thrombus. Atrial fibrillation also was ruled out. As CE patients presenting with ACS have acute thrombosis and are initiated on anticoagulation in an emergent manner, thrombophilia workup at presentation is not recommended due to flawed interpretation of the results because of the anticoagulant.[13]

This case represented educational value in its novelty in reporting an association between HS and CE in a patient with an ostium secundum ASD and in describing a step-wise approach to the case starting from initial management followed by investigational workup to identify the etiology of the embolus.

CONCLUSIONS

CE as a complication of splenectomy in hereditary spherocytosis patients should be recognized in patients with the correct clinical picture. However, complete workup should be pursued to identify any reversible causes of CE which might preclude the need for long-term anticoagulation. Emergent management of the CE cases should be similar to any ACS patient, but definitive management can differ based on the individual case.

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