Challenges in Managing Acute Myocardial Infarction Associated With Infective Endocarditis: A Case Report

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Abstract
Acute myocardial infarction (AMI) is a rare but recognized and potentially serious complication of infective endocarditis (IE). This case describes the challenges surrounding the management of AMI in the setting of septic coronary embolism, brain, spleen, and kidney infarcts due to septic emboli from native mitral valve IE.

Keywords
acute myocardial infarction, septic coronary embolism, CABG with simultaneous mitral valve replacement, infective endocarditis

Case Presentation
A 57-year-old female with family history of mitral valve prolapse presented to the hospital with altered mental status, preceded with 1 week of fatigue, generalized weakness, episodes of nonbloody vomiting, and diarrhea. Patient denied chest pressure, chest pain, fever, or history of intravenous drug use. On presentation, vitals were within normal limits except for heart rate of 135 beats per minute. Physical examination showed a thin, dry, lethargic female with Janeway lesions over palms and soles (white arrows, Figure 1A and B), Osler nodes (red arrow, Figure 1A), and splinter hemorrhages (Figure 1C). The heart rhythm was regular, and no murmurs were appreciated. Laboratory findings were significant for white blood cell count of 17,000/mm³ (normal 3700-11,000/mm³), hemoglobin of 11.7 g/dL (normal 12.0-16.7 g/dL), platelets 89,000/mm³ (normal 135,000-400,000/mm³), and point of care troponin of 0.28 ng/mL (normal 0.00-0.034 ng/mL). Electrocardiogram (EKG) showed sinus tachycardia with nonspecific ST-wave changes. Blood cultures were drawn, and patient was started on intravenous fluids and empiric intravenous antibiotics (ceftriaxone and vancomycin). Follow-up troponin returned at 0.85, and heparin drip was initiated. Patient was chest pain free. Repeat EKG showed new diffuse ST-segment elevation and PR depression consistent with pericarditis. Patient was treated with nonsteroidal anti-inflammatory drugs with symptomatic relief. Repeat TTE was requested and showed EF of 35% to 40% with severe diffuse hypokinesis of the apical wall and a possible vegetation on mitral valve. Transeosophageal echocardiogram (TEE) was scheduled on day 4, confirming EF of 30% to 35% with severe hypokinesis and medium-sized, 1.3 cm (L) × 1.0 cm (W), mobile vegetation on the atrial aspect of the tip of the anterior leaflet of mitral valve associated with moderate to severe mitral valve regurgitation (red arrow, Figure 2B). Cardiothoracic surgery was consulted.

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Figure 1. Multiple nontender and noncompressible macular spots of varying sizes over both hand (A) and foot (B) known as Janeway lesions (white arrows). Also a painful, red, raised lesion found on the hand (A) known as Osler nodule (red arrow). (C) Splinter hemorrhage underneath fingernail.

Figure 2. (A) Angiogram of left anterior descending coronary artery showing 100% occlusion without evidence of atherosclerotic coronary artery disease concerning for a septic embolus (blue arrow). (B) Transesophageal echo view showing medium-sized, 1.3 cm (L) × 1.0 cm (W), mobile vegetation on the atrial aspect of the tip of the anterior leaflet of mitral valve (red arrow). (C) Gross specimen showing vegetation involving the mitral valve leaflets.
Post TEE, patient developed shortness of breath and decompensated rapidly into cardiogenic shock. Patient was placed on vasopressor support. Troponin trended up to 49.50 ng/mL. An emergent cardiac catheterization revealed no signs of atherosclerotic coronary artery disease but a 100% occlusion of the proximal left anterior descending (LAD) artery suspicious for a septic embolus (blue arrow, Figure 2A). Patient was transferred to cardiac intensive care unit for further stabilization. The following day, patient underwent successful mitral valve replacement with coronary artery bypass grafting (CABG; left internal mammary artery to LAD). Gross specimen showed vegetations involving the mitral valve leaflets (Figure 2C). Patient did well in the postoperative period and was discharged to a rehabilitation center 2 weeks later in stable condition with 6 weeks of intravenous antibiotics (cefazolin 2 g intravenously every 8 hours) from the date of the surgery, metoprolol tartrate, furosemide, and potassium chloride.

Discussion

Infected endocarditis (IE) is a life-threatening multisystem disease that results from an infection of the endocardial surface of the heart, which may include heart valves, mural endocardium, or a septal defect. IE is almost inevitably fatal if left untreated, and often has long-lasting complications even in patients who survive. Acute myocardial infarction (AMI) secondary to septic coronary emboli is rare, and usually fatal. In a case series of 586 patients with IE, the incidence of acute coronary syndrome (ACS) was 2.9% (14 patients). Risk factors associated with increased coronary embolism include the presence of mobile vegetation, vegetation size greater than 10 mm, Staphylococcus aureus or non-viridans streptococcal infections, and previous embolism. ACS usually occurs within the first 15 days of disease onset. There have been different approaches used for the treatment of septic coronary emboli. These include balloon embolectomy, coronary stent placement, anticoagulation, fibrinolysis, surgical embolectomy, and CABG. Coronary stent placement or balloon embolectomy can be complicated by stent infection, emboli dislodgement, or mycotic aneurysm resulting in coronary rupture. Septic emboli consist of inflammatory cells and bacterial colonies with small proportion of fibrin and platelets; therefore, tissue plasminogen activator and anticoagulation seem ineffective, and increase risk of hemorrhage, especially in the setting of acute embolic stroke. Surgical embolectomy has been cited with good results; however, a direct incision into the coronary artery would increase the risk of contamination resulting in bacterial myocarditis that can lead to rupture of a ventricle. In contrast, emergent CABG presents a viable option for reperfusion of the myocardium while avoiding the complications seen in other interventions. In this case, the combined benefit of mitral valve replacement and bypass grafting outweighed the risk of surgical intervention.

Management dilemmas include the decisions to proceed with coronary angiography, surgical timing, and risk of renal failure. Previously, it was believed that coronary angiography in patients with IE was safe if no vegetation was observed on the aortic valve. Because of this, preoperative coronary angiography was only reserved for sets of patients who presented with ST-elevation. However, there are cases such as ours where the ST-elevations may not be obvious, and this could result in a missed diagnosis of ACS. Overall, guidelines do not recommend coronary angiography for non-ST-segment elevation myocardial infarction (NSTEMI), but do recommend diagnostic coronary angiography for STEMI. Preoperative coronary angiography can be carried out safely, with regard to the risk of complications of cerebral embolization or acute kidney injury. Another challenge in management is timing of potential surgery, especially in patients with concomitant cerebral infarcts, and risk of hemorrhagic transformation, but there is no evidence of worse outcomes with earlier cardiac bypass surgery. Although an average delay of less than 5 days between preoperative angiogram was previously described as a risk factor for postoperative renal failure and mortality, in the setting of total LAD artery occlusion, decreasing EF, and cardiogenic shock, we had no choice but to proceed with surgery. The decision to delay surgical intervention would increase risk for morbidity and mortality, or ultimate end-stage heart failure requiring cardiac replacement therapies.

Follow-up and Outcome

Repeat TTE on postoperative day 1 showed EF of 10%. EF improved to 20% on postoperative day 5. Unfortunately, 3 weeks after discharge, the patient had witnessed a seizure in the rehabilitation facility and Emergency Medical Services was called. Patient went into pulseless electrical activity (PEA) en route to hospital, cardiopulmonary resuscitation (CPR) was performed with return of spontaneous circulation. Patient was found to have potassium of 8, elevated troponins, and lactate acid. CT scan of brain showed evolution of the previously known ischemic infarctions with no acute or hemorrhagic components. Stat TTE postcardiac arrest (postoperative day 34) was unchanged from previous echocardiogram prior to discharge with EF of 20%. Cardiac arrest was likely secondary to seizure causing hyperkalemia followed by PEA arrest. There were no structural or dynamic changes in the TTE. Patient did not regain consciousness with resuscitative methods and was terminally extubated on postoperative day 35.

Conclusion

There are no clear guidelines for management of AMI due to IE. Diagnostic angiography of coronary arteries should be obtained in patients with active IE presenting with elevated troponins or acute left ventricular dysfunction seen on echocardiography. Coronary artery occlusion should
prompt immediate cardiothoracic surgery evaluation for consideration of emergent CABG with simultaneous valve replacement.

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**Author Contributions**
Mohammed Ali Faluk, Steven Vuu, Kiran Kathi, Ramy Abdelamaseih, and Christian Cignoni wrote the original draft of the paper. Mohammed Ali Faluk participated in gathering the data for the case and also compiling the draft. All authors read and approved the final manuscript.

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**Ethics Approval**
Our institution does not require ethical approval for reporting individual cases or case series.

**Informed Consent**
Written and verbal informed consent was obtained from the patient for their anonymized information to be published in this article. Special thanks to our patient.

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