

CLINICAL CASE OF THE MONTH

A 65-Year-Old Woman with Left Main Coronary Artery Thrombosis

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Cardiogenic shock due to left main coronary artery thrombosis is rare and associated with high mortality and poor prognosis. Urgent revascularization and hemodynamic support are vital to improving survival. We discuss a case of left main thrombosis as a culprit for myocardial infarction.

CASE PRESENTATION

A 65-year-old woman with past medical history of psoriasis, hypertension, hyperlipidemia and smoking presented to the emergency department (ED) with a five-day history of intermittent palpitations that became persistent and increased in intensity the morning of presentation and were associated with fatigue. She denied any other symptoms. She was not taking any medications and was allergic to codeine. She denied use of alcohol or illicit drugs. Her family history was noncontributory.

In the emergency department, vitals included a pulse of 107 beats per minute, blood pressure of 125/70 mmHg, temperature of 97.8° F, respiratory rate of 19 breaths per minute, and oxygen saturation of 100% on room air. Her physical examination was unremarkable. The presenting electrocardiogram revealed sinus tachycardia, ST elevation in aVR and diffuse ST-segment depression (Figure 1).

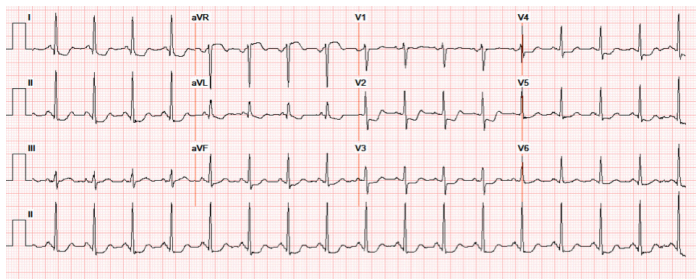


Figure 1: The presenting electrocardiogram in the emergency department, showing sinus tachycardia, diffuse ST-depression in the inferoposterior leads, with ST elevation in lead aVR.

The patient received a 325mg aspirin tablet and her palpitations resolved. D-dimer and urine toxicology screen were negative. Cardiac enzymes were mildly elevated. After evaluation by the cardiology team, the patient was taken urgently to the

catheterization laboratory for coronary arteriography. After right coronary artery (RCA) injections were completed, left coronary arteriography was conducted. Arteriography of the left coronary artery (LCA) demonstrated a thrombotic lesion in the left main artery (LM), and the patient began experiencing chest pain, diaphoresis, and nausea (Figure 2).

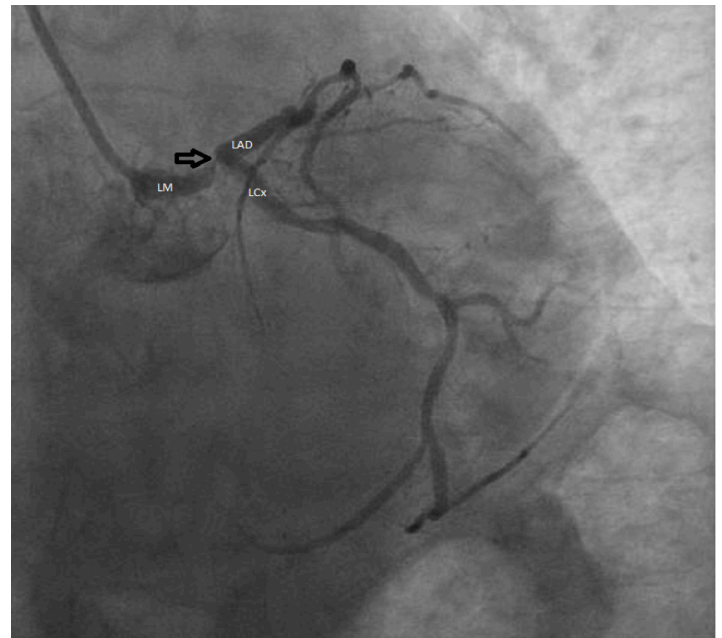


Figure 2: Antero-posterior/caudal image of the left coronary artery before intervention. Arrow points to the LM artery thrombosis. LM: left main artery; LAD: left anterior descending artery; LCx: left circumflex artery.

At this time, tachycardia worsened and the patient became hypotensive. Diagnosis of evolving cardiogenic shock and ongoing angina forced percutaneous coronary intervention (PCI) of the LM. An intra-aortic balloon pump (IABP) was advanced through the right femoral artery but shock persisted. Due to ongoing worsening hypotension and concern for pulmonary edema from cardiogenic shock, the patient was emergently intubated. A drug eluting stent was deployed in the LM artery extending into the left anterior descending (LAD) artery and balloon inflations in the ostial left circumflex (LCx) artery and subsequently kissing balloon inflations in the LM/LAD and LM/LCx arteries were performed using 3.0mm and 2.5 mm balloons (Figure 3).

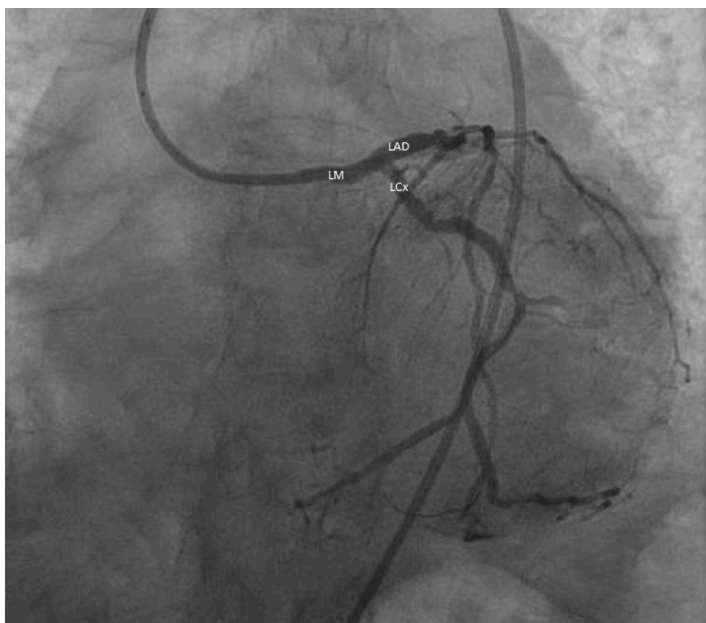


Figure 3: Antero-posterior/caudal image of the heart after intervention. LM: left main artery; LAD: left anterior descending artery; LCx: left circumflex artery.

The patient remained in shock in spite of successful PCI, IABP, and three inotropes. The decision was made to place an Impella CP heart pump device (Abiomed, Danvers, MA) for cardiopulmonary support. A transvenous temporary pacemaker was also placed for transient complete heart block. At this point, the patient was transported to the cardiac care unit on maximal doses of dopamine, norepinephrine, and epinephrine, along with Impella mechanical circulatory support.

Over the next 24 hours, the patient hemodynamics improved significantly and she was extubated. She was also successfully weaned off the inotropic and vasopressor agents as well as the mechanical circulatory support. A permanent pacemaker was placed due to recurrent atrioventricular block. The patient was discharged home on dual antiplatelet therapy and highest tolerated guideline-directed medical therapy. On follow up, the patient remains asymptomatic.

DISCUSSION

Significant LM coronary artery disease presents in about 3% of patient undergoing coronary angiography¹ and 4-10% of patients with unstable angina or NSTEMI.² Myocardial infarction, either non-ST-segment elevation (NSTEMI) or ST-segment elevation (STEMI) myocardial infarction, due to left main thrombosis is rare.³ Widimsky et al., concluded in their registry that the LM coronary artery was the culprit vessel in 97 out of 6742 patients with acute myocardial infarction (1.4%). Only 36% of these patients had a Thrombolysis in Myocardial Infarction (TIMI) 0-2 flow distal to the lesion.³

Acute myocardial infarction due to left main disease carries a very high mortality, especially if the patient presents with cardiogenic shock.^{4,5} In a meta-analysis conducted by Vis et al., 26% of patients who present with NSTEMI or STEMI due to left main disease presented with cardiogenic shock. This subgroup of patients had a 30-day mortality five-fold higher than those who did not present with cardiogenic shock (57% vs 11% respectively)⁴.

The management of unstable patients with acute myocardial infarctions due to LM artery disease is quite challenging, because they need hemodynamic support as well as rapid revascularization of an inherently complex lesion. National guidelines for management of patients with unstable angina/NSTEMI give coronary artery bypass surgery (CABG) a Class I indication if LM disease is the culprit and a IIa indication for PCI if the patient is not a CABG candidate. The same guidelines give PCI a class IIa recommendation in STEMI patients if it is feasible to perform quickly and safely.⁶ In patients with cardiogenic shock, ACA/AHA/SCAI guidelines for PCI give consideration of percutaneous mechanical circulatory support (MCS) a Class Ib indication.⁸ A number of percutaneously inserted devices are currently available; these include IABP, Impella (2.5, CP, or 5.0), Tandem Heart, extracorporeal membrane oxygenation (ECMO), and surgically placed left ventricular assist devices. The goals of MCS include the ability to prevent systemic shock syndrome by maintaining vital organ perfusion, reducing left ventricular filling volumes and myocardial oxygen consumption, augmenting coronary perfusion, limiting infarct size, and supporting circulation during complex intervention.⁸ In our case, we first placed an IABP which typically provides an extra 0.5-1 L/min of cardiac output, decreases myocardial oxygen consumption, and increases coronary artery perfusion.⁷ However, patients must have a modicum of left ventricular function and electrical stability for an IABP to be effective. The IABP, in addition to the pharmacologic support, proved to be insufficient hemodynamic support to bridge the patient over cardiogenic shock to recovery. As a result, cardiopulmonary support was escalated using an Impella CP. The Impella CP provides nonpulsatile flow up to an additional 3-4 L/min of cardiac output. The Impella 2.5 device (which we did not use) would have provided 2.5 L/min of additional cardiac output. Compared to IABP, Impella devices support cardiogenic shock by providing more cardiac output and systemic perfusion. In addition, these devices conceivably further reduce native left ventricular stroke work, filling volume,

and myocardial oxygen demand.⁸

CONCLUSIONS

Cardiogenic shock due to left main coronary artery thrombosis is rare and associated with high mortality and poor prognosis. Urgent revascularization and hemodynamic support are vital to improving survival. Intra-aortic balloon pumps, which until recently have been the only percutaneously deliverable mechanical support devices, have now been joined by a number of additional percutaneous mechanical circulatory support devices which can provide even greater assistance in cardiac function.

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