Spontaneous Coronary Artery Dissection: A Quite Unexpected Pregnancy Complication

Asad Mussarat, MD; Ahad Mussarat, MD; Ahmad Kasabali, MD; Shane Sanne, DO

Case: A 44-year-old female $G_{11}P_{11}$ with no prior medical history presented to the emergency department (ED) with left upper quadrant chest pain and shortness of breath that started at home while lying in bed. It lasted 45 minutes and was resolved on its own. She described her pain as substernal, radiating from her chest to her left arm and neck, and reproducible when lying supine. She denied alleviating factors, nausea, vomiting, weakness, numbness in her extremities, diaphoresis, or lower extremity edema. One week prior, she delivered her 11th child vaginally without complications. In the ED, she was afebrile and hemodynamically stable. Pertinent labs include elevated troponin at 2.79 ng/L (reference 0-0.04 ng/L) and D-dimer at 744 ng/mL. No ST-changes or T-wave inversions were appreciated on initial EKG. Concern for non-ST Elevation myocardial infarction (NSTEMI) prompted an aspirin load and coronary angiography which detected 40% luminal narrowing of the mid-vessel left anterior descending artery, consistent with a Type 2 coronary artery dissection. No additional coronary intervention was performed, and the patient was started on dual antiplatelet therapy (DAPT). CTA Head and Neck showed further non occlusive vertebral artery dissection. Patient's troponins had trended down since admission. She was discharged on DAPT and lifestyle recommendations, including avoiding subsequent pregnancy.

Discussion: Spontaneous coronary artery dissection (SCAD) is a rare cause of ACS that clinically mimics a myocardial infarction. Case studies calculate an incidence of 0.1 to 0.4% of patients in the general population, with a vast majority of cases occurring in women younger than 50 years old. In fact, pregnancy-associated spontaneous coronary artery dissection (P-SCAD) is considered the most common cause of acute coronary syndrome in childbearing women. Predisposing factors to pregnancy-related SCAD are those that increase coronary artery wall stress including multiparity, early postpartum state, and connective tissue disorders. Most P-SCAD events occur within the first month after delivery. Although P-SCAD's etiology is not fully understood, it is theorized that the increased level of estrogen during the third trimester enhances release of metalloproteinases. These matrix metalloproteinases are thought to weaken the vaso vasorum leading to the development of an intramural hematoma with or without intimal disruption.That hematoma can lead to compression of the coronary arteries and subsequent ischemia. Most SCAD patients heal spontaneously over time with conservative treatment, while coronary intervention would be reserved for cases with hemodynamic instability, involvement of large areas of the myocardium, and refractory ischemia. Unfortunately, pregnancy-associated SCAD carries a modest recurrence risk. Therefore, a multidisciplinary approach is imperative between a patient's cardiologist and obstetrics-gynecologist regarding management and counseling regarding subsequent pregnancy.