5-2020

Spontaneous Coronary Artery Dissection

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Case Presentation

A 36 y.o. female with past medical history of type 1 diabetes and recent cesarean section 10 days ago with delivery of a healthy male infant presents for chest pain. She reports chest pain for three days that has been constant, located in her left substernal region, sharp in nature and without radiation. She reports that the pain is worse with deep breathing and palpation. She has attempted to take Norco and Tylenol without relief. She states that she developed some mid thoracic back pain today as well. She admits to headache for one week. She denies lightheadedness, dizziness, nausea, vomiting, sore throat, shortness of breath, abdominal pain, fever. Prior to her arrival in the ED today, she presented to Urgent Care. She was given aspirin because of her chief complaint of chest pain and sent to the emergency department. Upon my initial evaluation in the Emergency Department she appeared uncomfortable. She was pale and tachycardic. She was not diaphoretic. Initial vital signs included a heart rate of 110, BP 115/80, RR 18 with oxygen saturation of 88% on room air. She was afebrile with a temperature 98.4° F.

The patient was placed on supplemental oxygen by nasal cannula. An ECG was ordered. Initial imaging orders included a portable 1 view chest radiograph and CT with IV contrast to evaluate for PE. Initial laboratory studies included a CBC, BMP, liver profile and a troponin with a repeat in 3 hours. ECG revealed sinus tachycardia at a rate of 116, t-wave inversions in leads V2 and V3, a right bundle branch block and no ST segment elevation. There were no prior ECG's to compare to. CXR demonstrated mild cardiomegaly, pulmonary vascular congestion with edema and bilateral small pleural effusions. Given the radiology findings, a BNP was added on. At this time the CBC, BMP and liver profile studies resulted. Her WBC was elevated to 15.6, hemoglobin 8.4, creatinine 1.71, BUN 33, AST 170 and the remainder of labs within normal limits. Additional labs were added, including coagulation studies, blood cultures, lactic acid. Due to the acute kidney injury and decreased GFR the CT PE study was withheld.

The laboratory called with a critical troponin value of 39.96 (normal < 0.04). The patient had already received aspirin at urgent care prior to arrival. She reported her chest pain as present but tolerable at this time. Cardiology was immediately contacted and requested that the patient be placed on heparin. Cardiology ordered a routine echocardiogram and planned to see the patient in the morning.

Additional labs resulted, revealing a BNP of 2,437 and repeat troponin was 58.80. ICU was consulted for evaluation given the rising troponin and overall clinical picture. Cardiology was contacted again given the rising troponin and persistent chest pain. Cardiology stated that they would see patient in the morning as she was low risk for acute coronary syndrome. ICU accepted the patient and she was transferred to intensive care shortly after in hemodynamically stable condition.

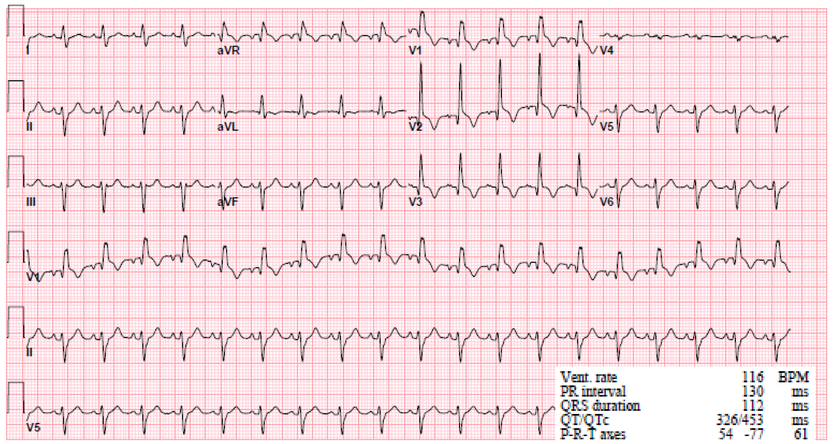
Overnight in the ICU the troponin peaked at 68.89 and began to trend downward. On day 2, Cardiology saw the patient in the morning and took her to preform a cardiac catheterization procedure. The cardiac catheterization revealed a coronary artery dissection of the LAD and an ejection fraction of 25 %. Cardiology recommended conservative medical management of her CHF which included aspirin, plavix, lipitor, digoxin, metoprolol, lasix, lisinopril and milrinone. An echocardiogram completed after the catheterization demonstrated an ejection fraction of 35% and akinesis of the apical and anteroseptal walls.

On hospital stay days 3 through 5 the patient continued to improve and was weaned of milrinone and oxygen. She was transferred to the general medical floor and remained chest pain free . On day 6 she was taken back to the cath lab for a repeat catheterization to re-assess the LAD. The study showed a 90% lesion of her LAD so the lesion was ballooned open and a stent placed. The patient tolerated to the procedure well and was observed overnight. She was discharged home on day 7 symptom free.

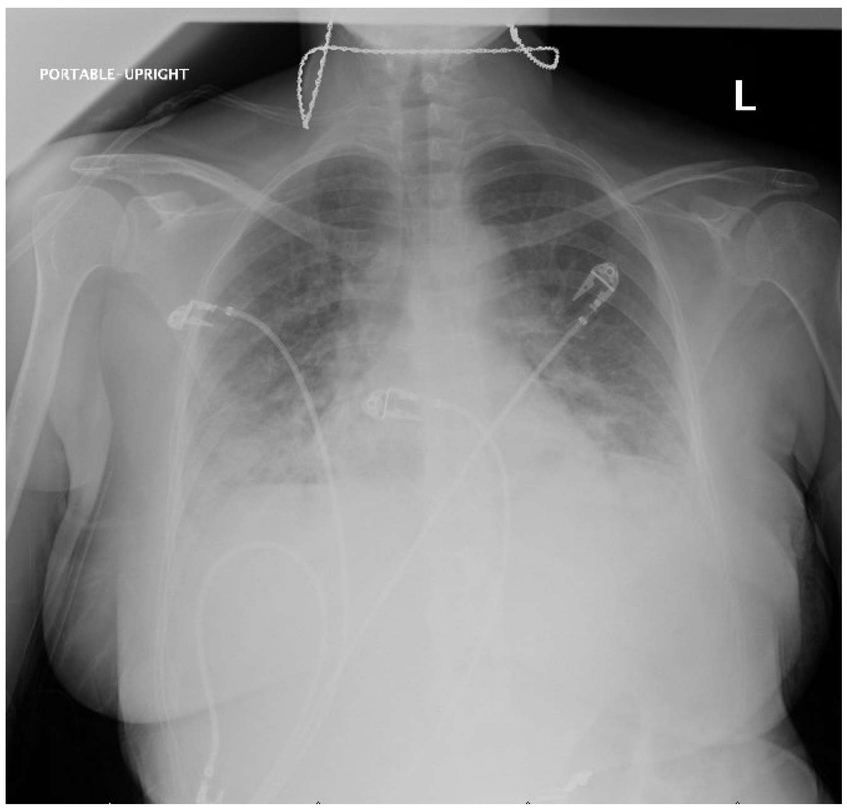
Introduction

Spontaneous carotid artery dissection (SCAD) is a rare cause of myocardial infarction. It occurs more commonly in younger, female patients. SCAD is non-traumatic and non-iatrogenic. Most patients that experience SCAD do not have typical risk factors associated with CAD. Conditions that pre-dispose patients to SCAD includes postpartum status, multiparty, connective tissue disorders (Marfan, Ehlers-Danlos), systemic inflammatory conditions or hormonal therapy. Although rare, labor and delivery can be a cause of SCAD.

Spontaneous coronary artery dissection is a cause of acute coronary syndrome in approximately 0.1 to 4% of cases. SCAD represents about 25% of ACS cases in women under 50.



Initial ECG and CXR in the ED



Presentation and Pathogenesis

Patients typically present with symptoms consistent with acute coronary syndrome, such as chest pain, dyspnea, nausea, back pain, neck pain and arm pain. History of extreme exertion just prior to onset of symptoms was reported in about half of men with SCAD. Emotional stress was reported by about 40% of women with SCAD. In one study, 18% of patients reported post-partum status. ECG imaging upon presentation may or may not show ST segment elevation. Troponin is usually elevated in these patients. There have been some reports of patients with SCAD who present with multiple dissections including the vertebral and carotid arteries, leading to very different clinical presentations. The underlying mechanism is not fully understood but includes an initial tear in the vessel lumen, followed by enlargement of a hematoma in that lumen that compresses the true lumen leading to ischemia and infarction. In postpartum women, it is believed that hormonal effects and hemodynamic stresses weaken the coronary artery wall.

Diagnosis and Treatment

Diagnosis begins with clinical suspicion. SCAD should be considered in young females without risk factors for coronary artery disease. It is important to recognize ACS and the need for coronary catheterization as this will ultimately make the specific diagnosis of spontaneous coronary artery dissection. Coronary angiography will show a dissection plane and the absence of coronary atherosclerosis.

Management for SCAD is usually conservative, medical management. Other treatment approaches have utilized percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG), and even cardiac transplant. However, studies have shown increased failure rates of PCI when compared to conservative management. This is due to the fragility of the vessel wall when it is dissected as well as difficulty with avoiding the false lumen when advancing the guidewire. Patients are often managed medically with aspirin, beta blockers and one year of clopidogrel. Following SCAD, recurrent cardiac events occur in about 17% of patients within 3 years, with 10.5% of those events being recurrent SCAD.

Conclusion

In conclusion, SCAD should be suspected in young female patients with no cardiac risk factors who present with ACS, especially if they are post-partum. The most common chief complaint is chest pain although other symptoms of ACS may be reported. ECG in the emergency setting may or may not show ST elevation however troponin is usually elevated. The official diagnosis of SCAD is made by coronary angiography and therefore will not be made in the emergency setting. Conservative management is recommended over PCI. Patients are often started on long term aspirin and a beta-blocker and given one year of clopidogrel. Approximately 17% of SCAD patients will have another coronary event within 3 years.

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