Case Report

Role of IVUS in Assessing Spontaneous Coronary Dissection: a Case Report

Amin Daoulah, MD[1*], Awad Al Qahtani, MD[2], Majed Mazen Malak, MD[3], Saud Al Ghamdi, CVT[1]

[1]*King Faisal Specialist Hospital & Research Center-Jeddah, Jeddah, Saudi Arabia.*
[2]*Hamad Medical Corporation, Doha, Qatar.*
[3]*King Abdulaziz University Hospital, Jeddah, Saudi Arabia.*

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare condition that can result in unstable angina, acute myocardial infarction, and sudden death. This condition may occur particularly in women during late pregnancy and in the postpartum period. We present the case of a 33-year-old African American woman, who had spontaneous left anterior descending coronary artery (LAD) dissection two weeks postpartum, resulting in acute ST-segment elevation myocardial infarction with severe left ventricular dysfunction. The use of the intravascular ultrasound (IVUS) in our case confirmed the diagnosis and helped with stent sizing and implantation. On subsequent follow-up, there was marked left ventricular function recovery and clinical improvement.

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Introduction

Primary spontaneous coronary artery dissection (SCAD) as a cause of acute coronary syndrome or sudden death is a rare entity with complex pathophysiology. It appears mostly in young women without traditional risk factors for coronary artery disease and a significant proportion of them present during the peripartum period. Early recognition of SCAD is important for initiation appropriate management.[1]

Case report

A 33-year-old African-American woman (Para = 2, Gravida = 0) was discharged home after an uneventful full-term vaginal delivery of a healthy baby. Two weeks postpartum, she presented to a regional hospital with severe chest pain of 24-hours' duration radiating to both arms. She did not have a history of hypertension, hypercholesterolemia, or diabetes mellitus or connective tissue disorder. She denied any history of medication that could have caused vasoconstriction or recreational drugs use, and she had had no intense physical activity prior to the onset of chest pain. There was no past history of spontaneous abortion. On physical examination, she had a pulse rate of 52 beats per min and a blood pressure of 150/85 mmHg. There was

[*]**Corresponding Author:** ***Amin Daoulah,*** *Consultant, Cardiovascular Department, King Faisal Specialist Hospital & Research Center-Jeddah, P.O. Box: 40047. Jeddah 21499, Saudi Arabia. Tel: +96 62 667 7777. Fax: +96 62 667 7777. E-mail: amindaoulah@yahoo.com.*

no clinical evidence of the Marfanoid feature or connective tissue disease, and her cardiovascular examination was essentially normal. The pelvic examination was significant for heavy vaginal bleeding. Her initial electrocardiogram (ECG) showed ST segment elevation throughout the antero-lateral leads (Figure 1). A bedside echocardiogram revealed severe hypokinesia of the antero-apical walls and lateral walls with left ventricular ejection fraction < 40%. Cardiac enzymes were elevated with peak total creatine kinase (CK) 500 IU/L (normal < 145 Iu/L), peak CK-MB 235 IU/L (normal < 16 Iu/L), and troponin 6 g/L (normal < 0.4 g/L).

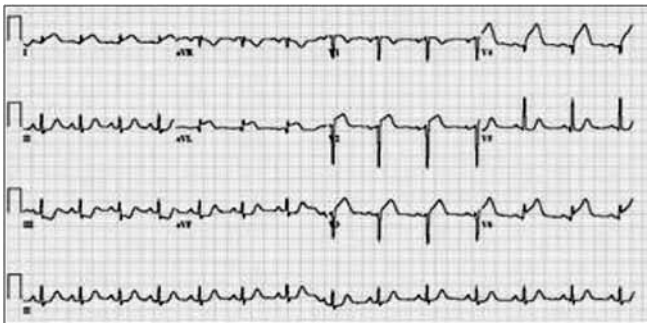

Figure 1. 12-lead electrocardiogram showing ST segment elevation through the anterolateral leads

Electrolytes and complete blood count test were normal. Erythrocyte sedimentation rate (ESR) was 3 and C-reactive protein (CRP) < 1. Thrombolytic therapy was contraindicated in her case; therefore, within one hour, the patient was transferred to the cardiac center for urgent coronary angiography and revascularization. The invasive coronary angiography showed mild ectasia with abnormal flow and 'hang-up' of contrast in the proximal part of the left anterior descending artery (LAD), which suggested a dissection flap. Also, there was a significant caliber reduction with thrombolysis in myocardial infarction (TIMI) Grade 2 flow in the distal part, and the rest of the study was unremarkable (Figure 2). An intravascular ultrasound (IVUS) was preformed to assess the size of the vessel and confirm the diagnosis (Figure 3). Thereafter, the patient underwent coronary artery angioplasty to the area of dissection, whereby two bare metal stents (4.0 x 16 mm, and 4.0 x 12 mm) were deployed at 16 atmospheres across the lesion in the proximal and mid –LAD. Post-stent IVUS showed good result with no further residual dissection detected and good stent wall opposition and expansion (Figure 4).

After revascularization, an intra-aortic balloon pump (IABP) was placed for hemodynamic support, and the patient was started on intravenous heparin. In addition, after consultation with her obstetrics/gynecologist, we initiated an eptifibatide infusion in order to reduce the risk of thrombosis and then she was transferred to the coronary care unit. The following day, the patient was asymptomatic, with stable vital signs and improved myocardial ischemia blood works, so the intravenous medication and IABP were discontinued and she was started on oral medications which included Aspirin, Clopidogrel, Ramipril, Metoprolol, and Simvastatin. Her hospital course was uneventful. On day six, she was discharged home. Her two dimensional echocardiography before discharge revealed ejection fraction (EF) > 50%. She remained symptom free during her follow-ups.

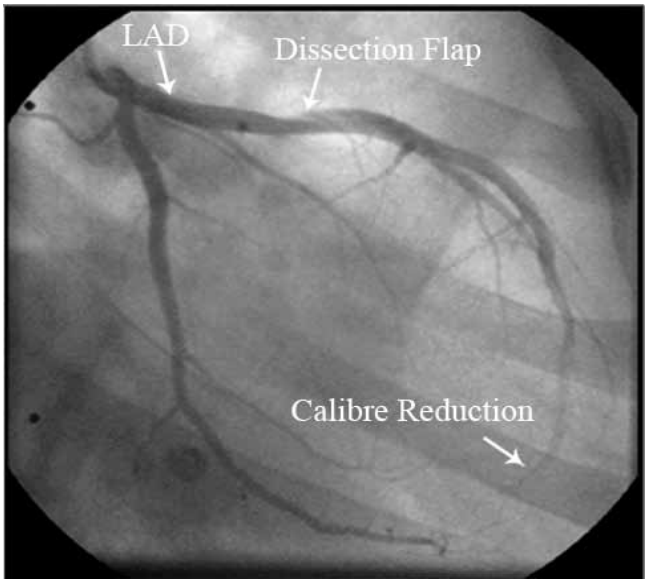

Figure 2. Right anterior oblique projection of the left anterior descending artery (LAD) demonstrating proximal ectasia and an intimal flap. There is significant caliber reduction distally with reduced flow

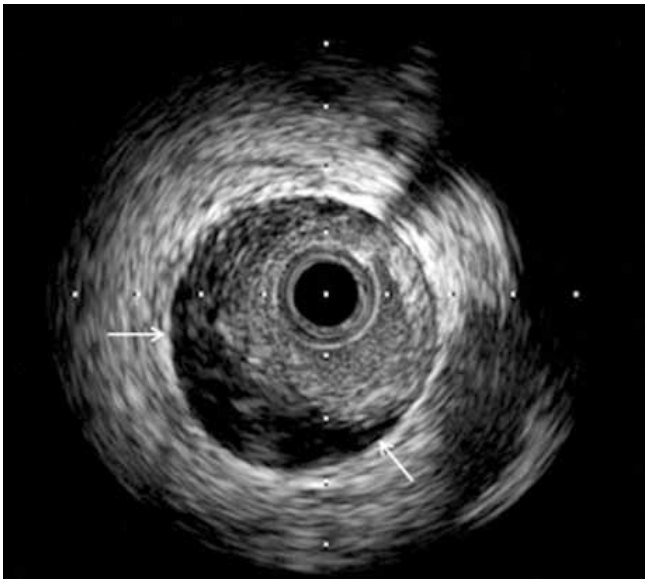

Figure 3. Cross-section of the mid-left anterior descending artery showing an intramural dissection between the media and adventitia, spanning from 5 to 9 o'clock (arrows)

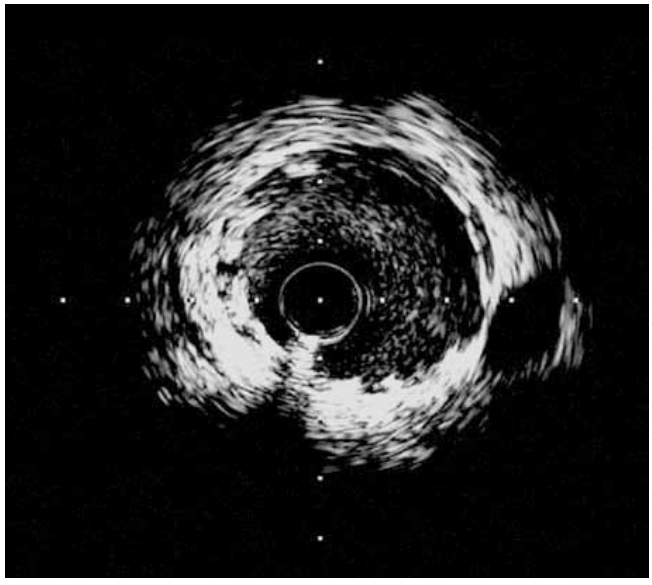

Figure 4. Post-stent intravascular ultrasound showing a good stent wall opposition with no further residual dissection detected

Discussion

Spontaneous coronary artery dissection (SCAD) is a rare well recognized cause of acute coronary syndrome. Seventy-five percent of cases occur in females with a mean age of 40 years and 33% are in the peripartum period.[1] It leads to sudden cardiac death in 50% of cases and a further 20% die within hours of symptom onset. Clearly, the mortality rate depends upon the extent and location of the dissection. SCAD is a result of a hematoma formation within the outer third of the tunica media, with subsequent expansion leading to compression of the true lumen and resultant myocardial ischemia.[2] A combination of hemodynamic factors and changes in the integrity of the coronary vessel lead to SCAD, which is often seen in the absence of atherosclerotic disease. SCAD is difficult to identify by conventional cardiac catheterization and may be an under recognized phenomenon. Dissection may be missed in the absence of an intimal tear or if the true lumen is severely narrowed. The classic angiographic appearance includes contrast media seen in two lumens separated by a radiolucent intimal flap, with persistence of contrast in the false lumen after washout from the remainder of the vessel. Also, hematoma filling the false lumen may simulate intracoronary thrombus. Consequently, the diagnosis of SCAD increasingly relies on IVUS, which can identify the presence and extent of medial hematoma.

The rarity and poor survival of SCAD limits data on the best available treatment options. Medical therapy of SCAD is appropriate for patients with resolution of symptoms and limited disease. Nitrates, beta blockers, and calcium channel blockers are thought to reduce coronary spasm and extent dissection.[3] Aspirin, Clopidogrel, and low-molecular weight heparin may limit hematoma formation and subsequent luminal compression.[4] The role of glycoprotein IIb/IIIa inhibitors is unclear.[5] Thrombolytic therapy may lyse the compressing hematoma and allow the native vessel to re-open. However, there have been several deaths following thrombolytic therapy in middle-aged females presenting with acute coronary syndrome (ACS) in the absence of atherosclerotic risk factors.[6] Clearly, lysis might allow expansion of the intramural hematoma and further myocardial ischemia from the native lumen compression. Surgical revascularization is best reserved for the left main, multi-vessel disease, or refractory ischemia despite aggressive medial therapy.[7] In the case of a well-localized symptomatic single coronary dissection not involving the left main, percutaneous coronary intervention with stenting is possible.[8]

Patients who have experienced peripartum dissection should be counseled against future pregnancies as the risk of dissection increases with multiparity and increasing age.[9] Patients should be followed clinically for symptoms of recurrent ischemia. Routine angiographic follow-up is not recommended. Stress testing with nuclear perfusion imaging is reasonable for surveillance, especially in patients with dissections of large vessels with large areas of myocardium at risk.

Conclusion

We conclude that the diagnosis of SCAD should be considered in the differential diagnosis of chest pain, especially in younger patients, peripartum women, and patients with underlying connective tissue disease. If the diagnosis of SCAD is suspected, then patients should be referred for primary percutaneous coronary intervention, where the diagnosis of coronary dissection can be made by coronary angiography and an IVUS may help in stent sizing and implant.

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