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Post-Partum Spontaneous Coronary Artery Dissection with Occluded Left Anterior Descending Artery

T. Davies[1], J. He[1,3], A. Hawley[2], B. Dyer[1], A. George[1]

1. Department of Cardiology, Royal Devon and Exeter Hospital NHS Foundation Trust, Barrack Road, Exeter EX2 5DW, UK.
2. Department of Cardiology, North Devon District Hospital, Barnstaple EX31 4JB.
3. Diabetes and Vascular Medicine Research Centre, University of Exeter, Barrack Road, Exeter, UK EX2 5AX.

Abstract

Presentation

We present a case of spontaneous coronary artery dissection (SCAD) in a 35-year-old lady who was 4 months post-partum. Despite a lack of typical risk factors, initial presentation and investigations were strongly suggestive of ACS.

Diagnosis

Coronary angiography revealed an occluded proximal left anterior descending (LAD) artery with TIMI 0 flow. Angiographic appearances and intravascular ultrasound (IVUS) were consistent with SCAD.

Treatment

A single drug-eluting stent was deployed, successfully restoring good flow, without extending the dissection flap.

Discussion

SCAD should be considered as an important and underdiagnosed cause of ACS, predominantly in young women without other apparent risk factors.

Introduction

SCAD is the spontaneous development of a false lumen within the coronary arterial wall (most commonly in the tunica media). This can lead to compression of the true lumen, and even occlusion of the artery, as demonstrated in this case. Risk factors include sex (female), the peripartum period, connective tissue disorders, hormonal therapies, and stress. In addition to increased rates of major adverse cardiovascular events (MACE), spontaneous coronary artery dissection may lead to sudden cardiac death[1]. With the use of high sensitivity biomarkers and early coronary angiography in suspected ACS, recent large cohort studies suggest that SCAD is more common than previously thought[2].

Case Report

A 35-year-old female who was 4 months post-partum presented with sudden onset, central chest pain at rest.

Her past medical history included previous abdominal surgery for bowel obstruction secondary to adhesions, and an ovarian haemorrhage. She was breastfeeding. She was taking the progesterone only pill and no other medication. Physical examination was unremarkable, and she was haemodynamically stable.

The initial ECG was unremarkable but she continued to have intermittent chest pain. ECGs were repeated, showing hyperacute T waves without ST-segment changes anteriorly. Initial high sensitivity troponins (Roche, Elecsys) were 57, 68, 44 ng/L (normal <14 ng/l). D-Dimer was <150 ng/mL (normal <500 ng/ml). Urgent trans-thoracic echocardiography (TTE) was performed showing moderate hypokinesia of the apex and apical segments, anterior, and anteroseptal wall. Simpson's biplane left ventricular ejection fraction was 43%.

Emergency coronary angiography demonstrated an occluded proximal LAD (Fig. 1). A high diagonal/intermediate vessel had an appearance suggestive of coronary dissection which had TIMI 3 flow and was therefore managed conservatively. The LAD was wired with a Sion Blue and the second diagonal branch (D2) with a BMW wire. Balloon dilatation using a 2.0x1.5 semi-compliant balloon was performed but did not restore flow. Since the exact mechanism of the vessel occlusion was not obvious at this point, Intravascular ultrasound (IVUS) was utilised, demonstrating changes consistent with SCAD in the mid LAD (fig. 2). Significant mismatch was noted between the LAD vessel size before and after the D2 bifurcation. A 3.5x16mm Synergy drug-eluting stent was hence deployed in the mid LAD, just before the origin of D2. This was then post dilated to 20atm with a 3.5x12mm non-compliant balloon, restoring TIMI 3 flow. Thrombus was noted at the LAD-D2 bifurcation and Tirofibran was given.

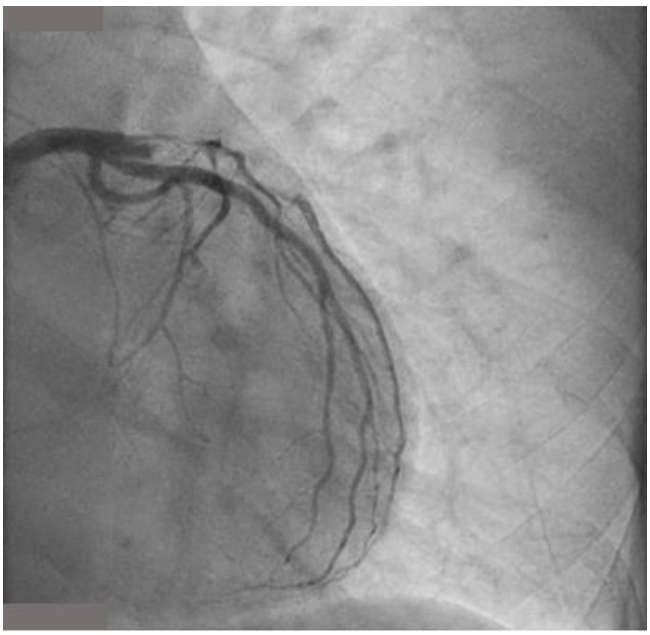

Figure 1: *(Occluded LAD)*

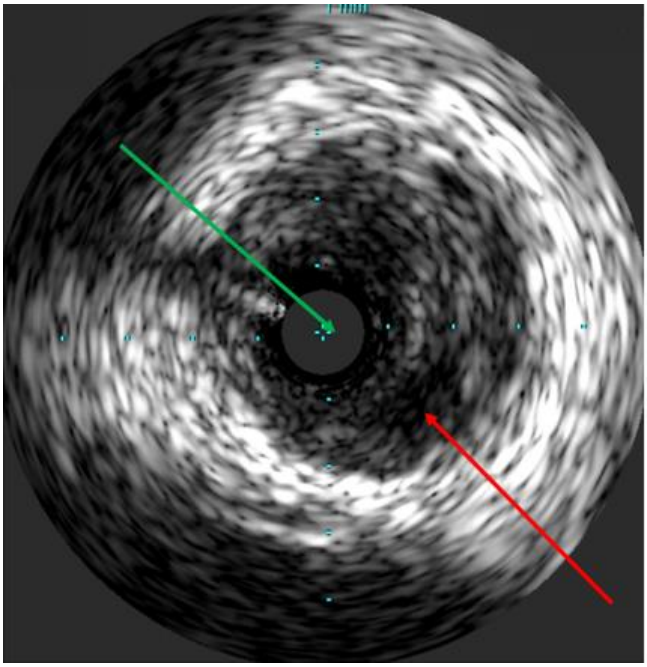

Figure 2: *(IVUS, green arrow demonstrating true lumen, red arrow demonstrating false lumen)*

The patient was transferred, pain-free, to the coronary care unit and later discussed at the "Heart Team" meeting, where ongoing optimal medical therapy was advised with interval TTEs. Further coronary angiography was felt to be high risk for further dissection and unlikely to influence management. A repeat TTE 6 months later showed normal left ventricular function with no regional wall motion abnormalities.

Discussion

The mean age of patient's presenting with SCAD is 44-53 years and 73% of cases are seen in women. Pregnancy or post-partum SCAD accounts for around 10% of cases and typically occurs in the early postpartum period. However, 50% of post-partum acute coronary syndromes are reportedly due to SCAD[1]. It is thought hormonal changes and haemodynamic stress associated with pregnancy weaken the coronary artery wall[3,4]. A retrospective review found that pregnancy was associated with more acute presentations of SCAD and higher risk features such as ST elevation, impaired left ventricular function and left main or multivessel SCAD[5].

The optimal management of SCAD remains uncertain. The European Society of Cardiology recommends conservative management unless there is haemodynamic instability or if the distal flow to the culprit coronary artery has been compromised[1]. There is an increased risk of coronary complications following percutaneous coronary intervention. If stenting is required, second-generation drug-eluting stents (DES) are advised.

There is an association between SCAD and fibromuscular dysplasia as well as other vascular abnormalities. The European Society of Cardiology therefore advises that SCAD-survivors undergo imaging of extra-coronary vascular beds to assess for arteriopathies[1].

Declaration of Conflicts of Interest:
The Author(s) declare(s) that there is no conflict of interest.

Corresponding Author:
Dr Timothy Davies
Clinical Fellow in Cardiology
Department of Cardiology
Royal Devon and Exeter Hospital Foundation Trust
Barrack Road
Exeter
EX2 5DW
E-Mail: timothy.davies2@nhs.net

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