Recurrent spontaneous right coronary artery dissection in the postpartum period—Think twice before you revascularize

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Introduction

Spontaneous coronary artery dissection (SCAD) is defined as coronary artery dissection that is not associated with atherosclerosis, trauma, or iatrogenic injury. Herein, we present a case of recurrent SCAD of unknown etiology and also discuss indications for revascularization and optimal management.

Case Report

A 36-year-old postpartum woman presented with a 6-h burning chest pain. She had no history of chronic disease or classical cardiovascular risk factors. A 1-mm ST segment depression in V4–V6 was observed in the initial electrocardiogram. The serum troponin level was elevated; however, no left ventricular wall motion abnormality was present. On coronary angiogram, the left coronary system was unremarkable, however, a dissection in the distal right coronary artery (RCA) was present (Fig. 1a). Due to intermittent chest pain, the operator decided to proceed with ad-hoc percutaneous coronary intervention (PCI). Intracoronary imaging [i.e., intravascular ultrasound (IVUS) or optical coherence tomography] was not available; thus, the procedure was performed on angiographic basis. A drug-eluting stent (DES) was successfully implanted. Control angiography revealed no signs of residual dissection (Fig. 1b). After uncomplicated inpatient follow-up, the patient was discharged. The patient underwent a rheumatologic evaluation as an outpatient; however, no evidence of rheumatologic condition was found.

Three weeks following the discharge, the patient presented with intermittent chest pain. Serial ECGs revealed no signs of ischemia; however, serum troponin was elevated ×100 above 99% cutoff point. Hypokinesis of inferior left ventricular wall was observed. Coronary angiography revealed a dissection starting from RCA ostium extending to the proximal edge of the distally implanted stent (Fig. 2a, 2b). Distally, TIMI 1-2 flow was present. Ad-hoc PCI was performed: three overlapping DESs were implanted (starting from RCA ostium up to the distal stent). Post-PCI TIMI 3 flow was present, and the patient was discharged 2 days later. One month later, the patient was asymptomatic.

Discussion

Recent registries have demonstrated that in certain subgroups (such as women aged <50 years) of patients with ACS, SCAD may be the underlying condition in up to 24% of patients (1). Although SCAD was thought to mainly affect pregnant and peripartum woman, it is now clear that this population represents a minority (2). Multivessel involvement is present in up to 19% of patients (3). Recurrence of dissection was previously reported, at a median time of 45 days, similar to our case (4).

Management of SCAD is a controversial subject. It has been previously observed that up to 86.3% of lesions may display angiographic healing (5) after conservative management. Because the success rate is lower and complications are more common with PCI (6), techniques such as use of cutting balloons to depressurize false lumen and use of longer stents to reduce the chance of flap propagation have been proposed; however, no strong evidence exists. There are some reports on increased subacute stent thrombosis due to strut malapposition that becomes evident following hematoma resorption (7). While it is clear that conservative management is associated with good long-term prognosis, no randomized trial has tested the type of patients that will benefit from revascularization. Generally, revascularization...
is performed in clinically unstable patients, those who have ongoing ischemia, and stable patients with severe 2-vessel proximal or left main dissection.

The mechanism of reoccurring dissection in our case, due to the unavailability of intracoronary imaging, will remain speculative. First, it is possible that during first procedure, the proximal edge of the implanted stent may have landed on an intramural hematoma causing expansion of dissection, even though no evidence of this was present on control angiography. Alternatively, the second dissection may have been a de novo event. Intracoronary imaging would have certainly given insights into the mechanism of dissection (8).

During the first presentation, the patient was relatively low risk, with a calculated GRACE score of 67 points. The distal flow was well preserved, suggesting that conservative management approach could have been the strategy of choice. It is important to remember that in SCAD, ischemia is not the only cause of chest pain, vessel dissection itself may be the cause of pain; thus, ischemia as the cause of pain should be carefully considered. Similarly, using troponin elevation as a criterion for intervention is questionable as ACS was the main clinical presentation of SCAD in trials, which have found conservative management safe (5). On the second presentation, indication for revascularization was clearer as large myocardial area was at risk, and angiographically, distal flow was impaired.

Finally, when decision is made to proceed with revascularization via PCI, we believe that intracoronary imaging can be a valuable tool in guiding the procedure (8).

**Conclusion**

Decision for PCI in SCAD should be made after careful consideration of clinical presentation, myocardial area at risk, and preferably with aid of intracoronary imaging.

**Informed consent:** An informed consent was obtained from patient.

**References**


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DOI:10.14744/anatoljcardiol.2019.93962

A unique late complication of transcatheter atrial septal defect closure.

**Introduction**

Secundum type atrial septal defects (ASD) are the most common type of interatrial shunts. Transcatheter closure is widely used in suitable cases. Although transcatheter closure has many advantages over surgical correction, it also has many early and late complications. Here we present a unique late complication of transcatheter closure of secundum type ASD.

**Case Report**

A 32-year-old female was admitted with atypical chest pain. Her medical history revealed that the patient had undergone transcatheter closure of secundum type ASD 28 months ago. Operation notes were reviewed which stated that the defect size was confirmed with balloon sizing and stop-flow method during cardiac catheterization. The defect was of the dimen-