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CASE REPORT

Left main and triple vessel dissection 2 months postpartum

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Abstract

Spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome. The majority of cases reported in the literature involve a single vessel; multivessel and left main (LM) coronary artery involvement is rare. We present a case of triple vessel and LM SCAD in a postpartum patient and review the literature regarding percutaneous coronary intervention in the setting of SCAD.

KEYWORDS

complications, coronary aneurysm/dissection/perforation, imaging, intravascular ultrasound, left main coronary disease, percutaneous coronary intervention, pregnancy, cardiovascular complications

1 | INTRODUCTION

Although spontaneous coronary artery dissection (SCAD) is a rare cause of acute coronary syndrome, it should be considered in young women presenting with chest pain. In women less than 50 years old presenting with acute coronary syndrome, up to 35% have been found to have SCAD, and many are initially misdiagnosed.[1] The majority of cases reported involve a single vessel and can be treated medically. Multivessel and left main (LM) coronary artery dissection is rare, but often requires invasive management.[2–4] We present a case of triple vessel SCAD involving the LM coronary artery, and review the diagnostic and management challenges of SCAD.

2 | CASE REPORT

A 41-year-old woman presented with sudden onset chest and back pain 10 weeks after delivery of twins via Caesarean-section. An electrocardiogram demonstrated sinus rhythm, with nonspecific repolarization abnormalities. She had been in good health and denied substance abuse. The initial troponin-I was 3.85 ng/mL (normal ≤ 0.05 ng/mL) with elevation of CK-MB (11.0 ng/mL, normal ≤ 3.6 ng/mL). Physical examination was notable for a tachycardia of 111/min. A chest X-ray was unremarkable.

Cardiac catheterization demonstrated discrete segments of narrowing in the mid- and distal left circumflex coronary artery (Figure 1). The left anterior descending (LAD) coronary artery had a relatively

small diameter, without discrete stenosis. The right coronary artery (RCA) was notable for two diffusely small caliber posterior left ventric- ular branches, with an irregular luminal appearance (Figure 2) with TIMI grade 3 flow.

Since the patient was a young woman without atherosclerosis risk factors, the findings were interpreted as consistent with SCAD, although it was unusual that multiple arteries were involved. The patient was treated medically with aspirin, clopidogrel and beta- blocker therapy. A transthoracic echocardiogram did not show wall motion abnormalities. A renal magnetic resonance angiogram was negative for findings of fibromuscular dysplasia (FMD). The cardiac enzymes trended downward.

On hospital day #3, she developed recurrent chest pain, accompanied by an increase in troponin-I to 9.49 ng/mL. A transthoracic echocardiogram demonstrated apical hypokinesis. The addition of oral calcium channel blocker and intravenous nitroglycerin therapy did not relieve her chest pain. She was transferred to a tertiary care center, where coronary CT angiography (CCTA) demonstrated dissection of the LM coronary artery, extending into the LAD and left circumflex coronary arteries (Figure 3). Despite the high-risk anatomical involvement, another attempt was made at medical therapy given resolution of chest pain and clinical stability at the time of these imaging findings. The patient initially responded and preparations for hospital discharge were initiated.

On hospital day #8, she developed severe chest pain and ventricular tachycardia. She was intubated and underwent emergent coronary angiography, which demonstrated dissection of the LM

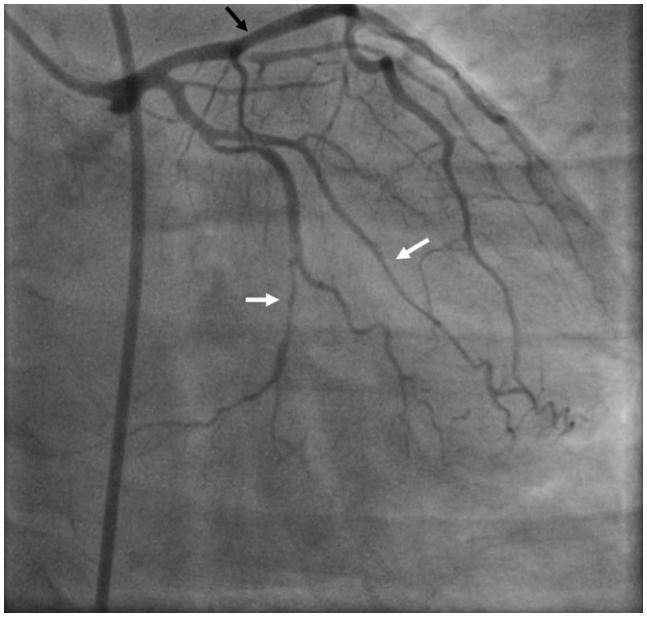

FIGURE 1 RAO caudal view of selective left coronary angiography. The white arrows designate diffuse narrowing of the distal left circumflex artery branches. The black arrow points to the left anterior descending artery, which does not appear to be affected by dissection in the initial angiogram

coronary artery from the ostium, extending the entire length of the LAD (TIMI grade 2 distal flow) and the left circumflex. The appearance of the RCA was unchanged. A non-selective ascending aortogram was used for guidance of a coronary guidewire into the LAD without selective injection of contrast into the LM artery (Figures 4 and 5).

Prior to selective coronary injections, intravascular ultrasound (IVUS) was used to confirm guidewire positioning in the true lumen

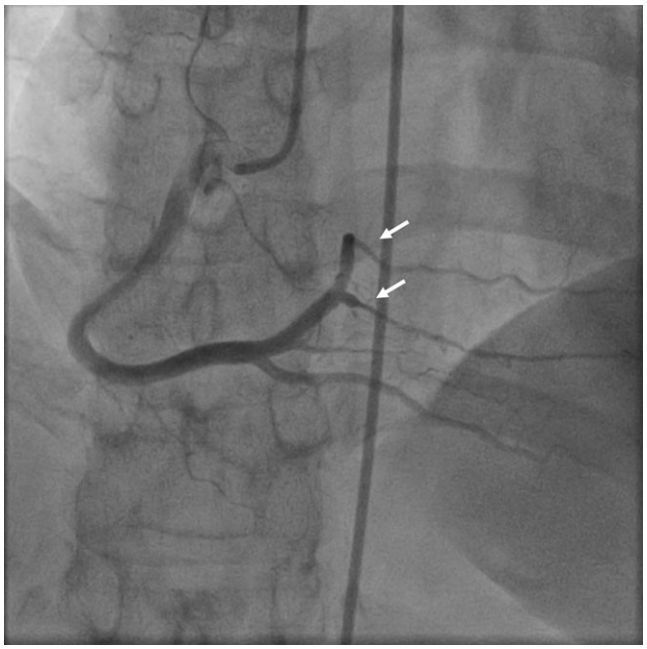

FIGURE 2 PA cranial view of selective right coronary angiography. White arrows denote two posterolateral branches with abrupt tapering of the vessel caliber and extensive thinning consistent with dissection

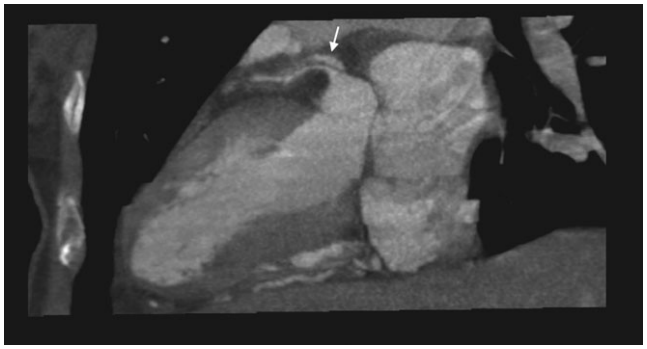

FIGURE 3 Coronary CT angiogram shows dissection of the left main artery as it branches into the proximal left anterior descending artery (white arrow)

(Figure 6). Five overlapping drug-eluting stents were placed, extending from the LM ostium into the distal LAD (Figure 7). An echocardiogram demonstrated a left ventricular ejection fraction of 35%. The troponin-I peaked at only 8.6 ng/mL, suggesting the possibility of myocardial stunning.

She was discharged on hospital day #16 on aspirin, clopidogrel, beta-blocker, statin, and a long-acting nitrate. She was placed on angiotensin receptor and aldosterone blockade. An echocardiogram 6 weeks later revealed an ejection fraction of 43% and 50% at 6 months.

3 | DISCUSSION

Spontaneous coronary artery dissection should be considered in young women presenting with chest pain. Risk factors associated with SCAD include postpartum state, multiparity, FMD, hormonal therapy, extreme emotional stress, use of cocaine or amphetamines, and other

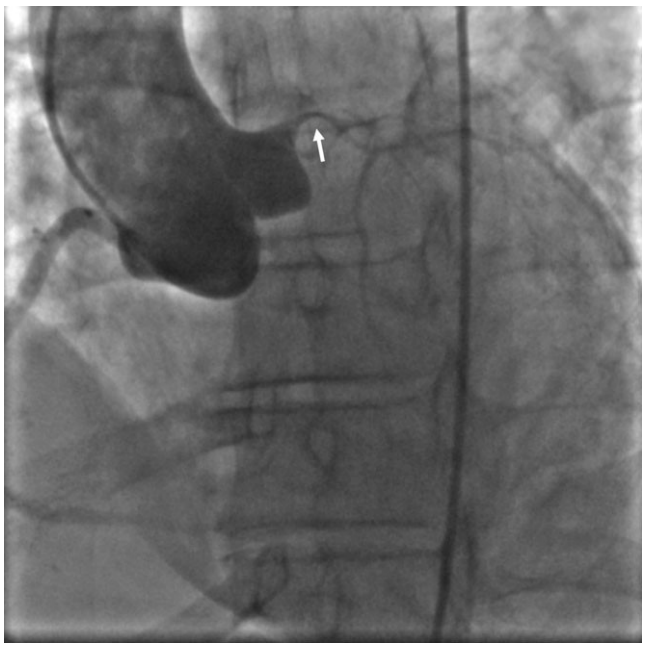

FIGURE 4 Non-selective aortic root injection with a pigtail catheter demonstrates an abrupt change in the caliber of the left main coronary artery

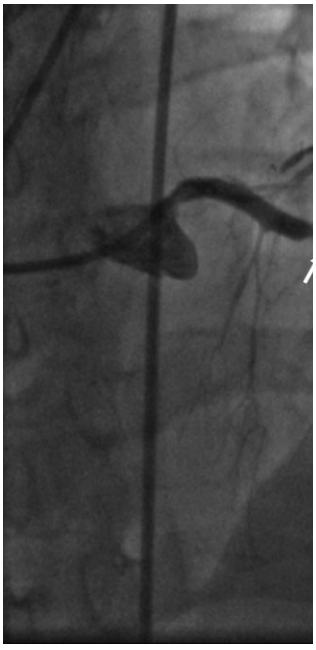

FIGURE 5 PA cranial view after placing the first stent into the left main artery. Selective left coronary artery injection. White arrow indicates diffuse narrowing of the mid segment of the left anterior descending artery where the true lumen is compressed as seen on IVUS. The distal guidewire is positioned in a small diagonal branch

vasculitides.[1,5] Our patient presented relatively late in the postpartum period, although SCAD has been reported from 5 weeks gestation to 1 year postpartum.[1]

Due to the relatively low incidence of SCAD, there are no guidelines for management. An algorithm to guide the decision of invasive versus conservative management was developed based on observational data and expert consensus.[1] The majority of patients with SCAD recover and show angiographic resolution with medical therapy. The relatively high complication rate associated with PCI treatment also favors a noninvasive approach. Although medical therapy remains reasonable in the clinically stable patient, if the patient is unstable or has refractory symptoms, revascularization is warranted.[1]

3.1 | Diagnosis

Coronary angiography remains the primary modality for diagnosing SCAD. There are three angiographic patterns of SCAD: type 1 is pathognomonic with contrast staining of the arterial wall and multiple lumens; type 2 includes a diffuse and smooth stenosis >20 mm long, and is the most frequently observed pattern, with an abrupt transition from normal arterial caliber to diffuse stenosis; type 3 is a focal or tubular stenosis <20 mm that is the most difficult to differentiate from atherosclerotic lesions and often requires intracoronary imaging to confirm the diagnosis.[6] While this classification has increased the recognition of SCAD in recent years, diagnostic uncertainty can persist after conventional coronary angiography. In cases with clinical suspicion for SCAD where the diagnosis remains uncertain, intracoronary imaging such as IVUS or optical coherence tomography (OCT) should be considered. Intracoronary imaging is also useful during PCI to mitigate wiring of the false lumen and unintended propagation of dissection flaps. Coronary CT angiography is an imaging modality with the advantage of evaluating the coronary arteries without the risk of extending dissections with the force of injection during direct cannulation of the coronary ostia. Despite this, CCTA is not recommended as a first-line diagnostic modality due to its limitations in evaluating the small arteries and distal segments where SCAD frequently occurs (high false negative rate). There may be a role for CCTA as an alternative to coronary angiography for follow-up in assessing resolution of SCAD.[5–7] In our patient, SCAD was strongly suspected after initial coronary angiography, but as the multivessel involvement was unusual, the diagnosis was confirmed with CCTA and IVUS.

3.2 | Medical therapy

Medical therapy for SCAD is predominantly composed of antianginal therapy. Beta-blockers should be considered for patients with arrhythmia,

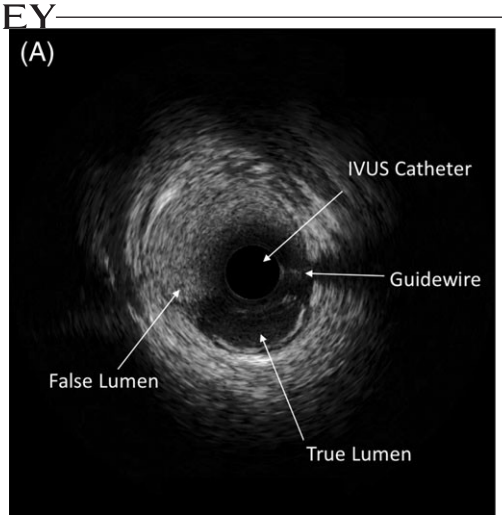

FIGURE 6 IVUS of the left anterior descending artery prior to selective angiography. Panel A (distal LAD) demonstrates the guidewire outside of the true lumen. The true lumen is compressed by the false lumen, which contains blood product representing hematoma or thrombus. Panel B (proximal LAD) demonstrates appropriate position of the guidewire in the true lumen. The guidewire had traveled from the proximal true lumen, into the false lumen, and back into the true lumen

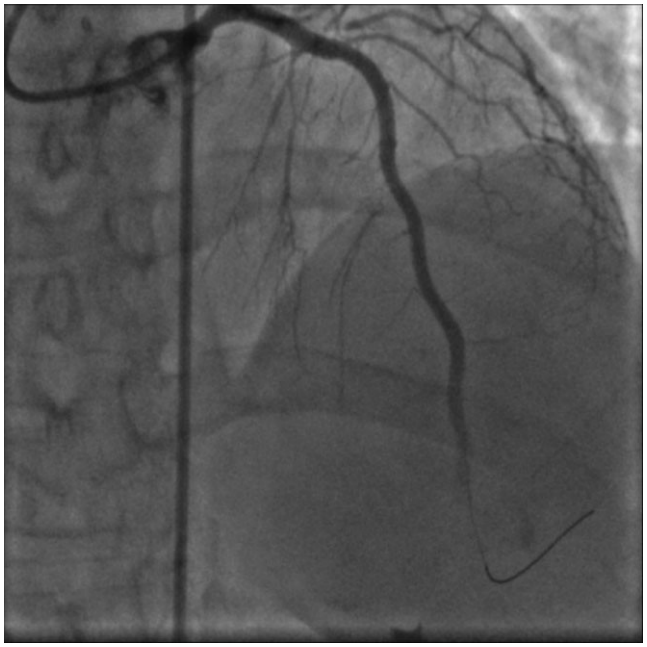

FIGURE 7PA cranial view of selective left coronary artery angiography post-stenting of the left main and left anterior descending arteries. Note the lack of septal perforator branches at the distal LAD, most likely due to occlusion of the vessels after PCI

hypertension, or left ventricular dysfunction. Angiotensin-converting-enzyme inhibitors and angiotensin receptor blockers should be used in the setting of myocardial infarction with left ventricular systolic dysfunction. Routine statin use is not recommended as observational data has not shown benefit in reducing recurrent SCAD. There is no evidence that anticoagulation or antiplatelet therapy is beneficial in SCAD, and there are theoretical concerns about the risk of bleeding into an intramural hematoma with extension of dissection with these therapies. Therefore, routine antiplatelet and anticoagulation is not recommended. Patients who require angioplasty should receive dual antiplatelet therapy as per guidelines for PCI.[1]

3.3 | Revascularization

The majority of SCAD cases involve a single vessel, predominantly the LAD, with the site of dissection most frequently in the mid to distal artery. Left main coronary artery involvement is rare, and even fewer cases of triple vessel involvement are reported (Table 1).[2–4] Patients with multivessel and/or LM involvement are often symptomatic or unstable, requiring invasive management. When feasible, PCI should be attempted. Although coronary artery bypass grafting could be considered in these cases, it may be difficult to identify the true lumen and maintain patency.

SCAD involving multiple vessels, particularly the LM coronary artery, poses several challenges for interventional cardiologists. Care must be taken when wiring across lesions to minimize the risk of entering the false lumen and propagating dissection flaps. The presence of intramural hematoma can extend proximally or distally when compressed during PCI. The intramural hematoma, which resorbs over time, may also produce late stent malapposition and stent thrombosis.[1,8] Finally, extensive involvement of distal, small caliber vessels often requires long stents with high rates of in-stent restenosis.[1]

Although there are no randomized studies of revascularization in SCAD, observational studies have shown a high risk of complications associated with PCI. PCI failure rates range between 27 and 36% in the three largest case series (Table 1). Even in cases of successful PCI, complication rates remain high. Saw et al reported that greater than 50% of case sof successful PCI were complicated by extension of dissections, including three patients with extension into the LM coronary artery.[3] Technical failure was attributed to wiring of the false lumen, extension of dissection resulting in reduced TIMI grade flow, loss or reduction in flow after stent placement, and residual stenosis >30%.[2–4] These findings are present across multiple smaller case series as well, highlighting the technically challenging aspects of PCI in patients with SCAD.

Several strategies for invasive management are associated with lower complication rates. When possible, a femoral approach is recommended, as radial access has been associated with increased risk of iatrogenic coronary dissection in retrospective studies. This may be due to non-coaxial engagement of the coronary ostium and tortuosity of the upper extremity vessels resulting in the need for more aggressive catheter manipulation.[1,3,9] Another potential benefit of femoral access is the ability to screen for associated arteriopathies with iliofemoral and renal angiography, which are common sites of extracoronary vascular involvement in FMD. Given the strong association of FMD and other arteriopathies with SCAD, screening with vascular imaging from the brain to the pelvis should be considered. CT angiography is the preferred noninvasive screening method; however, invasive angiography remains the gold standard due to superior resolution.[1] Once the decision to treat with PCI is made, stents long enough to cover both the proximal and distal edges of a lesion should be selected to minimize the risk of intramural hematoma extension. For long lesions requiring multiple stents, one strategy to prevent intramural hematoma extension is stenting of the distal edge first, followed by the proximal edge and ending with stenting of the middle of the lesion. Placement of stents can be confirmed by intracoronary imaging to ensure satisfactory coverage.[1,10] Bioresorbable vascular scaffolds (BVSs) may re-emerge as an alternative to metallic stents with the advantage of complete resorption over 2–3 years. This is an

TABLE 1PCI failure rates in large case series

	Left main	Multi-vessel	PCI as initial	PCI technical

Reference	Year	Patients, n	involvement, %	involvement, %	therapy, n (%)	failure rate, %
Saw et al	2014	168	<1	10	28 (16)	36
Tweet et al	2014	189	4	15	89 (47)	30[a]
Lettieri et al	2015	134	3	13.4	51 (38)	27.5

[a]Technical failure rate was calculated using SCAD-specific criteria (flow-based). By conventional criteria for failure (residual stenosis >30%), failure rate was 53%.

attractive option in SCAD, as patients areoftenyoung without atherosclerosis,andhavelonglesions requiringmultiplestents.Onelimitation of BVS is the observation that scaffold thrombosis ismorelikelyinsmallarteries,whichiswhereSCADtypicallyoccurs.[11]

Despite attempts to manage our patient conservatively,theextensive multivessel involvement resulted in severehemodynamicandelectricalinstability.Therefore,emergentrevascularizationwasperformed,whichwassuccessfullyguidedbyintracoronaryimaging.

4 |CONCLUSION

SCAD is an uncommon but increasingly recognized cause of chest painandacutecoronarysyndrome,particularlyinyoungwomenwithminimalriskfactorsforcoronaryarterydisease.Thediagnosisoftenrequiresahighindexofsuspicionandtheuseofintracoronaryimaging in addition to conventional coronary angiography. In uncompli- cated cases without high-risk anatomy, a conservative approach shouldbeattemptedduetothehighrisksoftechnicalfailureassociatedwithPCI.Surgicalevaluationisappropriatein patients with high-riskanatomy(LMorproximaltwo-vesseldissection).Inpatientswithhemodynamic instability or active ischemia, urgent revascularizationiswarranted.Intracoronaryimagingmayfacilitateidentificationofthetrue lumen duringPCI.

CONFLICT OF INTEREST

Nothing to report.

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