

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare but serious condition, constituting approximately 1% of acute coronary syndromes. It is predominantly seen in younger women, particularly postpartum and those on oral contraceptives. This non-atherosclerotic dissection involves the separation of coronary artery wall layers, leading to intramural hematomas and luminal tears, often triggered by physical or emotional stress. SCAD presents similarly to acute myocardial infarction, with chest pain as the hallmark symptom. The gold standard for diagnosis is coronary angiography. We present a 38-year-old postpartum female with SCAD.

CASE PRESENTATION

A 38-year-old female, six months postpartum and with a history of non-ST segment elevation myocardial infarction, presented to the emergency department with ongoing substernal chest pain radiating to the left shoulder. Previous coronary angiography had shown a 10-20% occlusion of the mid-left anterior descending artery. Physical examination was unremarkable. An electrocardiogram showed ST elevations in V5 and V6 (Figure A). Initial troponin was 0.07, peaking at 39.95. She received full-dose aspirin and was started on intravenous heparin drip. Subsequent coronary angiography revealed no significant occlusion but identified SCAD in the proximal obtuse marginal artery (OM1) with distal filling defects (Figure B). Echocardiography demonstrated left ventricular ejection fraction of 45% with lateral wall hypokinesis. She was initiated on aspirin, clopidogrel, metoprolol succinate, lisinopril and rosuvastatin. The patient was discharged with a plan to repeat coronary angiography in three months.

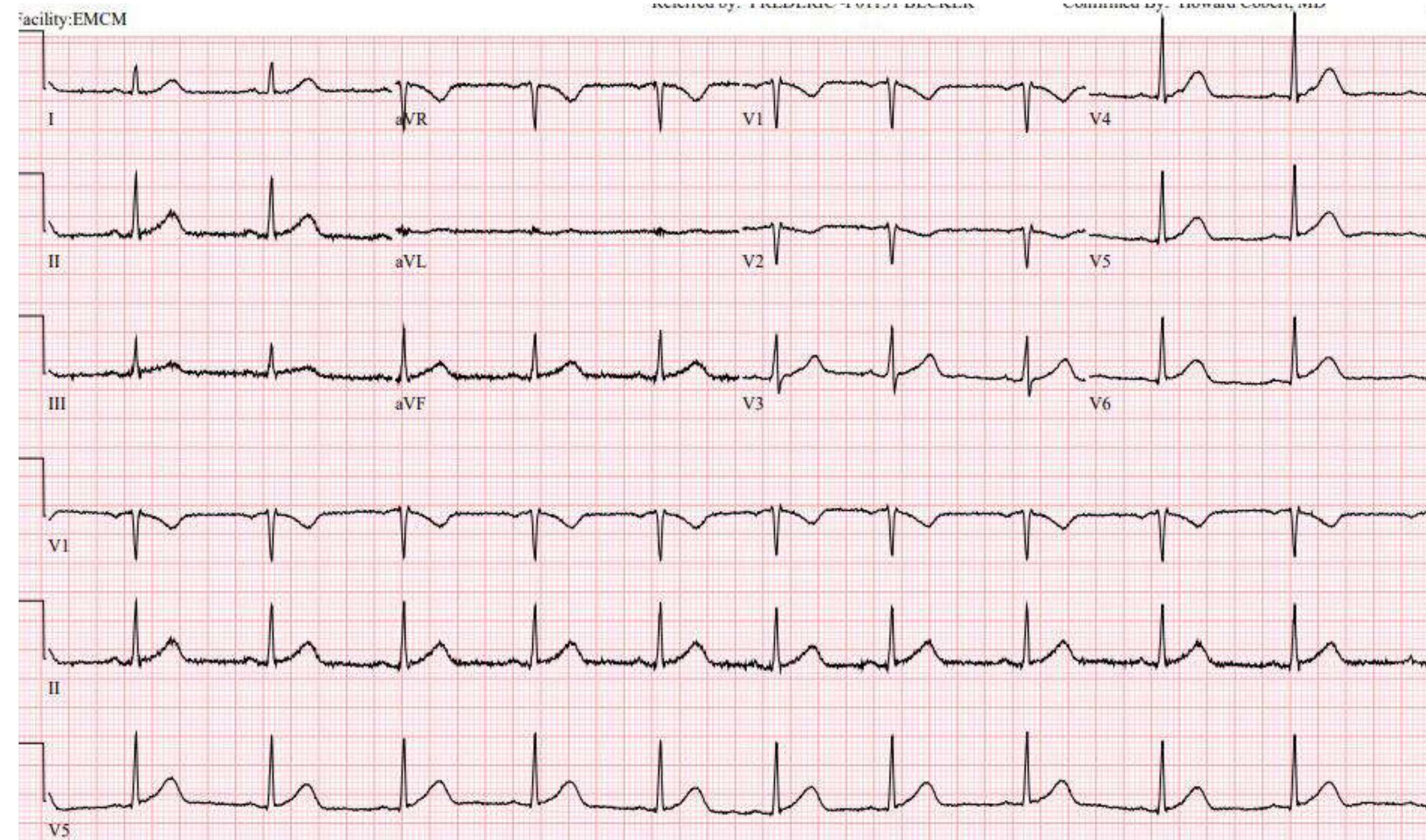


Figure A: EKG showing ST segment elevation in lead V5 and V6

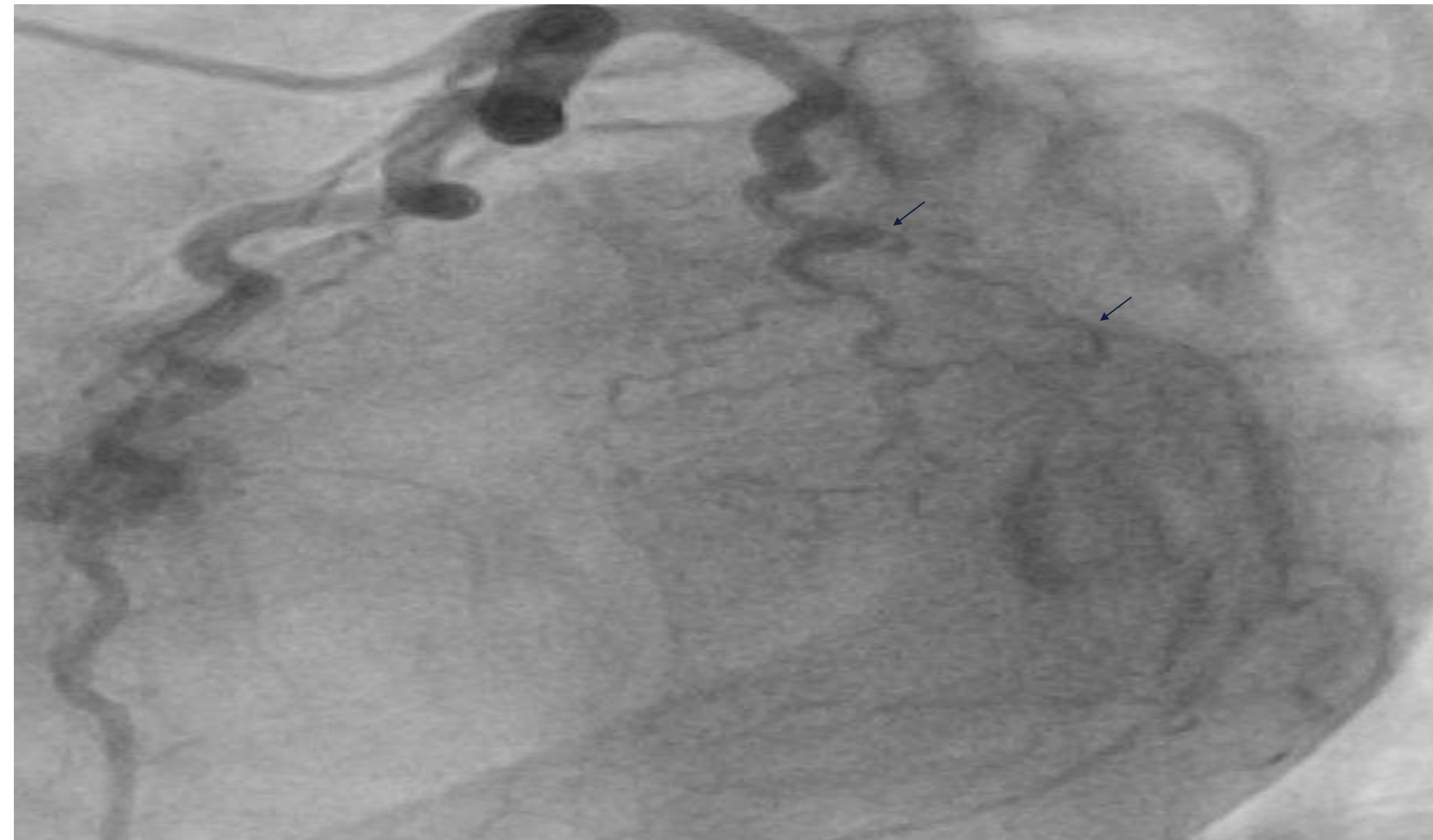


Figure B: Coronary angiography showing spontaneous coronary artery dissection in the proximal portion of OM1 with distal filling defects (arrows)

DISCUSSION

SCAD during pregnancy typically occurs within six weeks postpartum but can also appear during pregnancy and up to 18 months postpartum.

While not fully understood, elevated progesterone and hormonal changes may compromise vessel walls, with increased cardiac output, blood volume, and childbirth stress intensifying the risk. Unlike obstructive coronary artery disease, the management of SCAD prioritizes conservative treatment due to the fragility of the affected vessels. Percutaneous coronary intervention is generally avoided due to technical challenges and the heightened risk of procedural complications. Stable patients with low-risk lesions are managed with long-term aspirin, beta-blockers, and short-term clopidogrel, with statins added in cases of dyslipidemia. Beta-blockers are particularly pivotal in management, as their negative inotropic effects reduce blood pressure and arterial wall stress, thereby decreasing the risk of dissection propagation and recurrence. Coronary artery bypass grafting is reserved for severe cases involving multiple vessels, left main coronary artery, and hemodynamically unstable patients.

Our patient was managed conservatively with dual antiplatelet therapy, metoprolol, and rosuvastatin highlighting the tailored approach required for effective SCAD management.

CONCLUSION

SCAD is linked to high mortality and increased risk of heart failure and recurrence. Early diagnosis and proper management might improve outcomes and reduce long-term morbidities. Reproductive-aged women with a history of SCAD should receive comprehensive counseling on recurrence risks, especially if planning future pregnancies.

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