ESC European Society of Cardiology

European Heart Journal - Case Reports (2023) **7**, 1–5
https://doi.org/10.1093/ehjcr/ytad406

CASE REPORT
Coronary intervention

Clinical considerations during spontaneous coronary artery dissection in the post-partum period: a case report

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Received 3 April 2023; revised 3 August 2023; accepted 15 August 2023; online publish-ahead-of-print 18 August 2023

Background Spontaneous coronary artery dissection (SCAD) is a common and under-recognized cause of myocardial infarction during the post-partum period.

Case summary We report a case of a young women presenting with chest pain in the post-partum period. Her clinical appearance was that of a myocardial infarction, and angiography was indicative of a Type 2 SCAD. The patients had persistent chest pain, reduced left ventricular function, and critical left anterior descending artery stenosis. Percutaneous coronary intervention was done with caution. Shared decision-making with the patient helped guide the medical treatment plan and follow-up.

Discussion We discuss the clinical considerations surrounding the management of this patient.

Keywords SCAD • Peri-partum • Case report

ESC curriculum 3.1 Coronary artery disease • 3.2 Acute coronary syndrome • 3.4 Coronary angiography • 9.8 Pregnancy with cardiac symptoms or disease

Learning points

- To be aware of pregnancy-associated spontaneous coronary artery dissection as a potential diagnosis in patients with peri-partum myocardial infarction.
- To be aware of the complex management considerations, medical and interventional, in women with myocardial infarction in the peri-partum period.
- The decision to perform percutaneous coronary intervention should be individualized and considered in situations with high-risk clinical and angiographic features, as there is a risk of dissection propagation during PCI.

Introduction

Spontaneous coronary artery dissection (SCAD) results from the development of a false lumen in the coronary vessel wall, ensuring luminal narrowing and ischaemia. It is an increasingly recognized cause of myocardial infarction in younger women.[1] There is an association of SCAD and the peri-partum period.[2] This is hypothesized to be due to the hormonal effect on the vasculature that is heightened at the end of pregnancy as well as the increased blood volume in the maternal circulation following birth and uterine contraction.[2] Pregnancy-associated SCAD is associated with a

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Handling Editor: Grigoris Karamasis
Peer-reviewers: Josip Andelo Borovac
Compliance Editor: Ralph Mark Louis Neijenhuis

more severe critical clinical presentation and poorer outcomes.[3] We discuss the clinical considerations in the management of a patient with pregnancy-associated SCAD.

Summary figure

Baseline	Elective caesarean section and development of pre-eclampsia during admission
Two weeks later at 7 p.m.	Patient presents to the emergency room with chest pain.
7:20 p.m.	Patient undergoes cardiology assessment and echocardiography showing apical dyskinesia and reduced left ventricular function.
7:50 p.m.	Patient is taken for the cardiac catheterization laboratory, and angiography showed critical stenosis of 99% of the middle left anterior descending artery with imaging suggestive of spontaneous coronary artery dissection. Due to the clinical picture, percutaneous coronary intervention was performed.
Next day	Multidisciplinary team discussion with the patient regarding medication and breastfeeding implications
5 weeks later	Patient is doing well and repeat echocardiogram shows recovery of left ventricular function.

Case presentation

A woman in her 40s presented to the emergency department following 4 h of acute chest pain. The pain was described as severe pressure on the chest and radiated to the left arm.

The patient was 2 weeks following the birth of a healthy daughter (gravity 6, parity 3, caesarean section 3, spontaneous abortion 3).

The pregnancy was uncomplicated. She had an elective caesarean section due to previous caesarean section with her first pregnancy (breech presentation). This was performed at week 38 + 3. However, during admission following the caesarean section, the patient was noted to have elevated blood pressure and proteinuria and was treated with magnesium intravenously for pre-eclampsia. She was discharged with no further medication and was currently breastfeeding her child.

She was not on any other chronic medications, no past medical history, with no known allergies. She did not smoke, and her lipid profile 4 years prior revealed LDL blood level of 78 mg/dL.

Her initial vital signs were blood pressure 145/80 mmHg, heart rate 95 b.p.m., normal oxygen saturation, and no fever. She was not in haemodynamic compromise or respiratory distress. The heart and lung examination were without abnormal findings.

The electrocardiogram (*Figure 1*) showed sinus rhythm, normal axis, narrow QRS complex with T waves inversion in the anterior leads, and Type 2 Wellens sign in leads V4–V5 with QTC 462 ms (Bazett formula). There was no recording or symptoms of an arrhythmia on telemetry monitoring in the emergency room.

A focused echocardiogram in the emergency department showed reduced left ventricular dysfunction (ejection fraction estimated to be 40%) with hypokinesia of the mid and apical segments mostly of the anterior wall in the four-chamber and two-chamber views, with no valvular lesions and no pericardial effusion (see Supplementary material online, *Video S1*)

Blood results reported an elevated troponin T 1633 ng/L (normal value < 13 ng/L), NT ProBNP 1046 pg/mL (normal value < 125 pg/mL), creatinine kinase 1451 U/L, creatinine of 0.6 mg/dL (glomerular filtration rate according to MDRD 133.5 mL/min/1.73m^2) (normal value: 0.51–0.95 mg/dL), AST 116 U/L (normal values < 31 U/L), no electrolyte abnormalities, haemoglobin 14 g/dL (normal values: 12–16 g/dL), and platelets 431 K/mcL (normal values: 150–450 K/μL).

Due to ongoing chest pain, the patient was taken urgently to the catheterization laboratory. Angiography showed critical stenosis of 99% of the middle left anterior descending artery with TIMI flow score

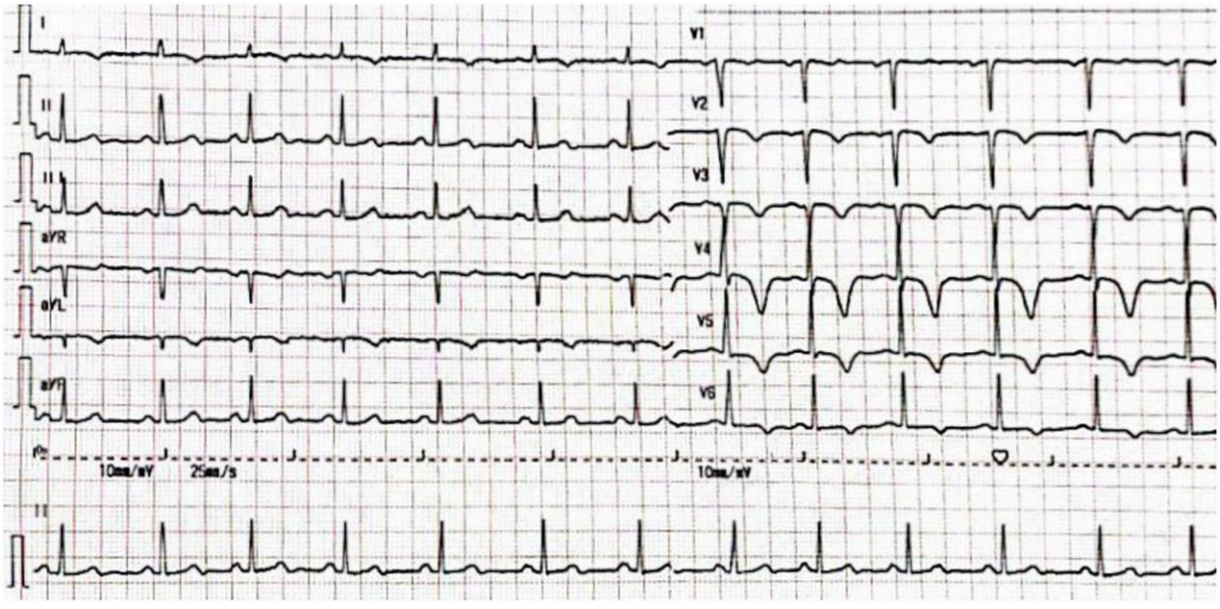

Figure 1 Electrocardiogram at presentation with sinus rhythm and Type 2 Wellens sign in leads V4–5 with QTC 462 ms (Bazett formula).

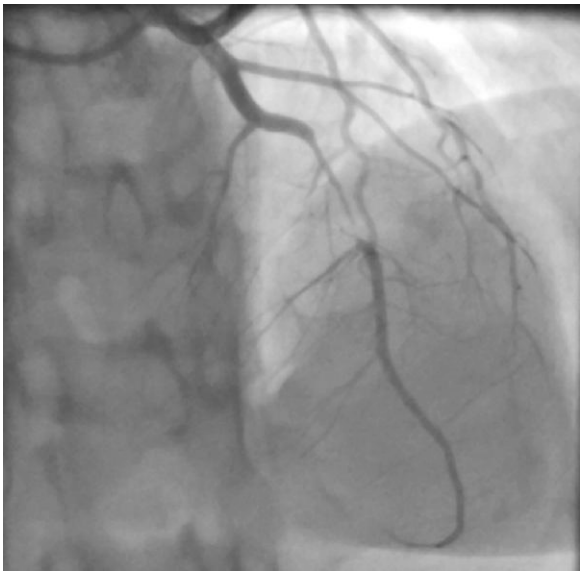

Figure 2 Angiography showed critical stenosis of 99% of the middle left anterior descending artery with TIMI flow score of 3 (left anterior oblique cranial view).

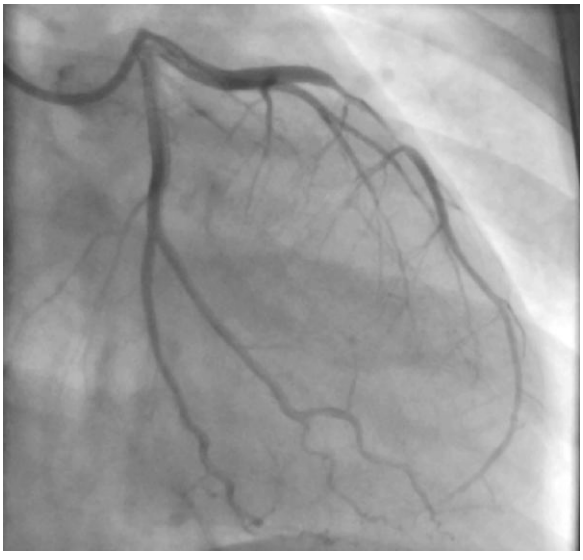

Figure 3 Angiography showed critical stenosis of 99% of the middle left anterior descending artery with TIMI flow score of 3 (right anterior oblique caudal view).

of 3 (*Figures 2* and *3* and Supplementary material online, *Video S2*). This lesion was tapered and indicative of spontaneous Type 2 coronary artery dissection (SCAD). No other coronary lesions were noted with smooth contour of the coronary arteries otherwise. Due to the clinical presentation of ongoing chest pain, abnormal electrocardiogram, and reduced left ventricular function, percutaneous coronary intervention was pursued. This was done with a 5 French 3.0 EBU engagement catheter, careful wiring of the coronary artery using a floppy wire (RUNTHROUGH NS Floppy, Terumo, Japan) and direct stenting

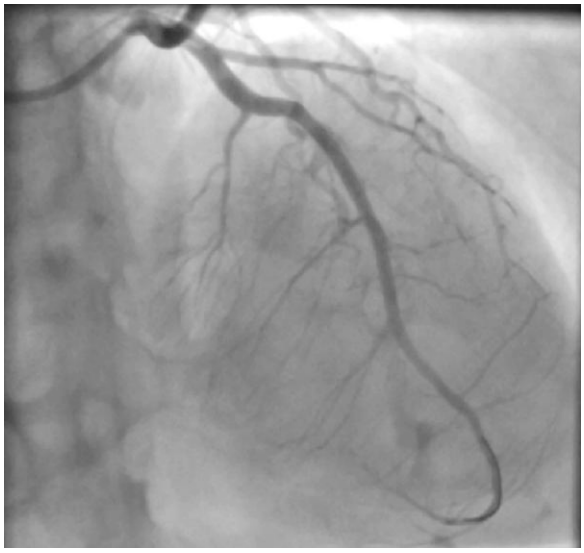

Figure 4 Good angiographic results following percutaneous intervention.

with a single long EluNIR™ ridaforolimus drug eluting stent (Medinol, Tel Aviv, Israel, 2.75 mm diameter × 28 mm length) with a good angiographic result and optimal distal angiographic flow (*Figure 4*). The patient had immediate clinical improvement and was transferred for observation in the coronary care unit. She was given a loading dose of aspirin (300 mg) and clopidogrel (600 mg).

Her echocardiogram the following day showed a reduced ejection fraction of 40% with anterior-apical dyskinesia and no valvular abnormalities. She was found to be hypertensive during her admission with 24 h albuminuria of 838 md/day and was started on Enalapril 5MG BD following a nephrology and gynaecology consultation. Bisoprolol 2.5MG QD was added due to her left ventricular dysfunction. LDL results were 144 mg/dL, and a statin (atorvastatin 80 mg) was initiated.

A shared decision-making discussion was done with the patient about the recommendations and relative lack of data on antiplatelet drugs and effects of drugs from drug eluting stents (DESs) in breastmilk. The patient decided to avoid the potential risk on her child from the drugs and to stop breastfeeding. Her antiplatelet regimen was then changed to dual antiplatelet with ticagrelor (90 mg twice daily) and aspirin (100 mg once daily).

Follow-up

One month following her admission, the patient continues to feel well. She was referred for magnetic resonance angiography of extra-coronary vasculature (renal and cervico-cephalic) for screening for fibromuscular dysplasia (FMD). The FMD is an arteriopathy with a high prevalence amongst patients with SCAD. Patients with SCAD should be screened for associated risk factors such as arteriopathies (including FMD), connective tissue diseases, and systemic inflammatory conditions.[2]

She was referred to our outpatient cardiac rehabilitation programme. Left ventricular function improved to an ejection fraction of 50% on repeat echocardiography at one month follow-up and further heart failure medications, such as SGLT2 inhibitors and mineralocorticoid receptor antagonists, were not initiated. At latest follow-up (3 months post-delivery), mother and baby are both doing well. We have discussed that our recommendation is a detailed pre-conception multidisciplinary discussion prior to any further pregnancy.

Discussion

This interesting case brings to light clinical considerations unique to acute coronary syndrome in the early post-partum period.

The first and most important is awareness of the clinical entity of SCAD, specifically in young women presenting with a myocardial infarction during the post-partum period. Pregnancy-associated SCAD most commonly occurs in the post-partum period but can occur at any time during the pregnancy. This entity comprises up to 43% of pregnancy-associated myocardial infarctions.[3] The peri-partum state is a known risk factor due to the haemodynamic changes that occur with an acute increase in blood volume with uterine contraction together with the hormonal changes that have occurred throughout the pregnancy causing softening of the vasculature.[4] Pregnancy-associated SCAD compared to SCAD not associated with pregnancy is associated with a more critical clinical presentation, worsened left ventricular dysfunction and haemodynamic instability, and an increased likelihood of multivessel dissections. Risk factors for pregnancy-associated SCAD include pre-eclampsia and multiparity, which were present in our patient.[3]

Another consideration is the treatment of these patients both in terms of catheterization and in terms of medical therapy. The nature of SCAD is that of weakened coronary arteries and the development of an endothelial tear and false lumen. In such, there is an increased risk of secondary iatrogenic dissection and propagation with percutaneous intervention. Conservative management is an acceptable and recommended management strategy in stable patients.[2,5] Coronary catheterization, if performed, should be performed by experienced interventionists with the utmost care to avoid further propagation of the dissection both distally and proximally. This included the use of the smallest coronary catheters available, minimizing unnecessary contact with the coronary vasculature during angiography and using long stents (relative to the dissected lesion length) to cover the weakened, narrowed vessel wall with a single intervention without causing further dissection. The European Society of Cardiology guidelines on acute coronary syndrome advocate percutaneous intervention only in selected case of high-risk anatomy or persistent symptomatology.[6] Thus, unlike 'conventional' ST elevation myocardial infarction, not every SCAD should be revascularized and conservative management should be considered as a reasonable management option.[2,5] Our patient was symptomatic and had evidence of substantial left ventricular dysfunction with a long-dissected segment of the left descending coronary artery and thus, intervention was performed. The guidelines do suggest the use of intracoronary imaging in the setting of obstructive SCAD with normal coronary flow, however, this is controversial and carries the risk of potentiating the dissection with further manipulations prior to definitive PCI. There is controversy as to the optimal management of patients presenting with SCAD, and patient management should be individualized based on the clinical and angiographic presentation of each case.

Due to the nature of SCAD, the use of dual antiplatelet therapy (DAPT) is debateable. There is limited data on the use of antiplatelet drugs during lactation with P2Y12 inhibitors. Low dose aspirin and clopidogrel should be used with caution, and the newer generation drugs of prasugrel and ticagrelor have conflicting/unknown data regarding their use during lactation.[7] The DISCO registry investigated retrospectively the use of antiplatelet therapy in medically managed patients with SCAD. They found that the use of DAPT compared to single antiplatelet therapy (SAPT) had a 2.6 times higher rate of major adverse cardiac events driven by increased non-fatal myocardial infarction or unplanned PCI. Most of these events occurred within the first month of follow-up.[8] These findings suggest that DAPT may prevent the absorption, or even enhance the intramural bleeding within the coronary wall and thus potentiate lumen narrowing and ischaemia. The SAPT can be an acceptable treatment strategy in those who are medical managed. However, more data is needed on the management strategy, and there is an ongoing randomized control trial on this topic.[9,10] We were also aware that the stent used was impregnated with a drug. The drugs used in DES target cell growth and neo-intimal proliferation to prevent in-stent restenosis and are also used for certain oncology and immunosuppression indications.[11,12] The effect of these potent long-acting drugs on the breastfeeding infant was a concern. The effects of antiproliferative drugs used in these stents have, to the best of our knowledge, not been studied in lactating mothers. However, we have checked with the DES manufacturer (Medinol, Tel Aviv, Israel) and learned that ridaforolimus used in the EluNIR DES is an analogue of rapamycin that has been studied in lactating rats.[13] This drug was detected in breastmilk in rats but at extremely low levels and found to have no effect on the offspring (data available on file at Medinol). Radaforolimus has been tested in clinical trials in humans and found to have a NOEL (no observable effect level) of 2.1 mg/kg, which is much lower than the amount of the drug found EluNIR stents (0.01 mg/kg in a 100 mm stent) (data available on file at Medinol). This data seems to suggest that the systemic exposure to the drugs in this specific DES is minimal, however, the clinical effects are difficult to examine and validate.

Spontaneous coronary artery dissection in the post-partum period can present as a clinical emergency. The management of these patients should be individualized, taking into consideration clinical and angiographic features. Shared decision-making with the patient regarding medical and follow-up treatment plans is important to ensure optimal outcomes.

Lead author biography

Nili Schamroth Pravda is a cardiologist and internal medicine specialist. She has a focus in the fields of cardio-obstetrics and adult congenital heart disease.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports*.

Acknowledgements

The authors would like to thank Dr Shir Tal, Dr Shahar Vig, and Prof. Katia Orvin that assisted in the management of this patient.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: None declared.

Funding: None declared.

Data availability

The data from this case study are available on request from the corresponding author.

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