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CASE REPORT
Coronary heart disease

Postpartum multi-vessel spontaneous coronary artery dissection in the setting of cocaine and amphetamine use: a case report

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Background Spontaneous coronary artery dissection (SCAD) is a recognized cause of acute coronary syndrome (ACS). Pregnancy, the postpartum period, and illicit drug use have all been reported as potential triggers.

Case summary We describe the case of a 41-year-old patient who presented to the emergency department with chest pain in the setting of recent cocaine and amphetamine use. The patient was 4 months postpartum following an uncomplicated pregnancy. Past medical history was non-contributory, with no known risk factors for ischaemic heart disease. Electrocardiogram was normal but high-sensitivity troponin T was significantly elevated. Coronary angiography revealed multi-vessel SCAD. This was managed conservatively as the patient remained clinically stable and pain free without high-risk anatomy (left main stem or proximal two-vessel coronary artery dissection).

Discussion Spontaneous coronary artery dissection must be considered in a postpartum patient presenting with ACS, particularly in the context of environmental stressors such as illicit drug use. Coronary angiography is key to determine diagnosis and guide management. Conservative therapy is favoured, except for patients with ongoing ischaemia, haemodynamic instability, and left main stem involvement. In this case, we suspect SCAD occurred due to the haemodynamic effects of cocaine and amphetamines in the context of structural arterial changes of the postpartum state.

Keywords Spontaneous coronary artery dissection • Acute coronary syndrome • Postpartum • Cocaine • Amphetamines • MDMA • Case report

Learning points

- Spontaneous coronary artery dissection (SCAD) must be considered in a postpartum patient presenting with acute coronary syndrome, particularly in the context of environmental stressors such as illicit drug use.
- Coronary angiography is key to determine diagnosis and guide management of SCAD.
- Conservative management of SCAD is favoured, except for patients with ongoing ischaemia, haemodynamic instability, and left main stem involvement.

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Introduction

Spontaneous coronary artery dissection (SCAD) is a recognized cause of acute coronary syndrome (ACS) and constitutes 2–4% of ACS presentations overall. However, the incidence is higher in women under 50 years presenting with ACS, estimated at 24–35%.

Pregnancy, the postpartum period, and illicit drug use have been reported as triggers. We present a case of SCAD in a 41-year-old postpartum female in the setting of both cocaine and 3,4-methylenedioxymethamphetamine (MDMA) use (an amphetamine derivative). To our knowledge, this is the first reported case of SCAD with all three of these potential precipitating factors coinciding in a single patient.

Timeline

Day	Event
0	Ingestion of cocaine and amphetamines Patient is 4 months postpartum
3	Presents to ED with chest pain ECG shows normal sinus rhythm with no ST changes. Initial Troponin T is elevated at 196ng/L Echocardiography shows preserved left ventricular function with normal valves and no regional wall motion abnormalities
4	Coronary angiogram demonstrates type 2a dissection of the mid to distal left anterior descending artery There is also type 2b dissection of the distal posterior descending artery of the right coronary artery
6	Magnetic resonance angiography of the renal and carotid/vertebral arteries is normal
7	Discharged home on medical therapy
40	Patient well at follow-up in clinic

Case presentation

A 41-year-old female presented to the emergency department (ED) with chest pain. It was described as substernal chest tightness, 5/10 in severity with radiation to the left arm and jaw. Past medical history was non-contributory, with no known risk factors for ischaemic heart disease. The patient was 4 months postpartum following an uncomplicated pregnancy. She admitted to using cocaine and MDMA 3 days prior to presentation. Physical examination was normal with a blood pressure of 130/80 and a heart rate of 76 b.p.m. The patient was pain free on arrival to ED.

An electrocardiogram (ECG) on presentation showed normal sinus rhythm with no ST changes. Initial Troponin T was elevated at 196 ng/L, peaking at 1116 ng/L at 12 h (normal range: 0–14 ng/L). Echocardiography showed preserved left ventricular (LV) function with normal valvular morphology and no regional wall motion abnormalities.

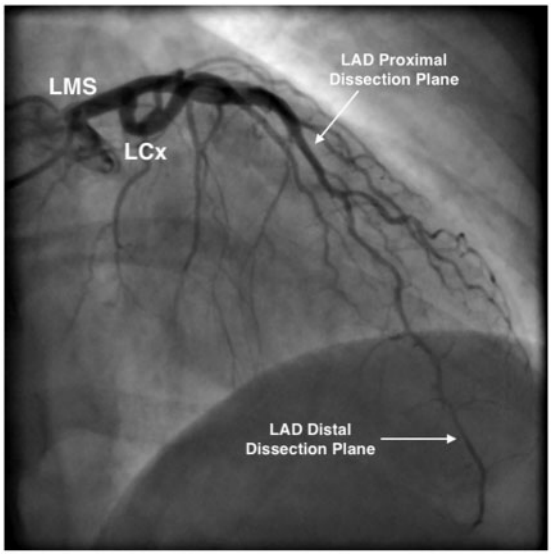

Figure 1 Coronary angiogram demonstrating type 2a dissection of the mid to distal left anterior descending (LAD) artery without extension to the apex.

Coronary angiography demonstrated type 2a dissection of the mid to distal left anterior descending artery without extension to the apex (*Figures 1* and *2*). There was also type 2b dissection of the distal posterior descending artery of the right coronary artery (*Figure 3*). Intracoronary glyceryl trinitrate (GTN) did not affect these findings. The left main stem and circumflex artery were angiographically normal. Intracoronary imaging was not performed in order to avoid the risk of propagation of dissection. This was managed conservatively as the patient was clinically stable and pain free without high-risk anatomy (left main stem or proximal two-vessel coronary artery dissection).

The patient was treated with aspirin and beta-blocker therapy. Magnetic resonance angiography of the renal and carotid/vertebral arteries was normal. The patient was discharged after 7 days and was well at 6 weeks follow-up in clinic. There were no further episodes of chest pain and excellent compliance with medications. The patient has been enrolled in cardiac rehab and counselled on the importance of illicit drug use cessation. We plan to see her in clinic in 3 months.

Discussion

Spontaneous coronary artery dissection was first described by Pretty at autopsy in 1931.[1] The predominant mechanism of myocardial injury is coronary artery obstruction by intramural haematoma or intimal disruption, rather than atherosclerotic plaque rupture or intraluminal thrombus.[2] Secondary causes (cardiac catheterization, chest trauma, aortic dissection, and cardiac surgery) must be excluded before a coronary artery dissection is deemed 'spontaneous'. The aetiology of SCAD is multifactorial. Potential contributors include underlying arteriopathy, genetics, hormonal influences, systemic inflammatory diseases, and environmental stressors. The most dominant association

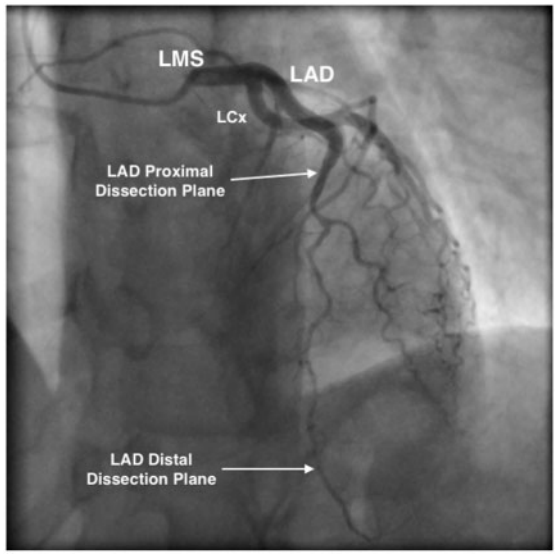

Figure 2 Coronary angiogram demonstrating type 2a dissection of the mid to distal left anterior descending (LAD) artery without extension to the apex.

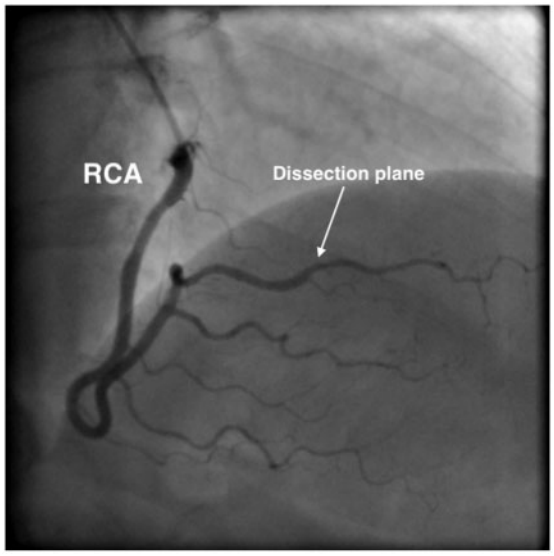

Figure 3 Coronary angiogram demonstrating type 2b dissection of the distal posterior descending artery (PDA) of the right coronary artery (RCA).

reported is fibromuscular dysplasia with a prevalence of up to 86% in SCAD cohorts who were routinely screened.[3]

Pregnancy-related SCAD (P-SCAD) accounts for <5% of SCAD cases.[3] However, P-SCAD is implicated in ~40% of pregnancy-related myocardial infarction.[4] It occurs most commonly in the 3rd trimester of pregnancy and the early postpartum period. High progesterone levels during pregnancy can weaken the tunica media through loss of normal corrugation of elastic fibres, a decrease in acid mucopolysaccharides, and impairment of collagen synthesis.[5] Haemodynamic changes during late pregnancy, such as augmented cardiac output and circulating volume, predispose to SCAD. These changes increase arterial wall shear stress, causing micro-structural changes in the aorta, which can extend to the coronary arteries.[6]

Cocaine is associated with a number of cardiovascular conditions, including SCAD.[7] Jaffe *et al.*[8] described the first case of cocaine-related SCAD in 1994. Cocaine acts as a sympathomimetic, resulting in increased inotropic and chronotropic response, and vasoconstriction via alpha-receptor stimulation. Consequent high arterial wall shear stress may lead to dissection. Cocaine also has a prothrombotic effect through increased platelet activity and aggregation.[9] Amphetamine-related SCAD has also been described.[10] Amphetamines have similar cardiovascular sequelae to cocaine, relating predominantly to coronary vasospasm and platelet activation-mediated thrombus formation.

In this case, we suspect SCAD occurred due to the haemodynamic effects of cocaine and MDMA in the context of structural arterial changes of the postpartum state. This 'second hit' phenomenon has been suggested previously, with environmental factors precipitating SCAD in vulnerable individuals. This is based on the concept that sequential insults, which are individually innocuous, can lead to overwhelming physiologic reactions.

Coronary angiography is the gold standard for the diagnosis of SCAD, regardless of aetiology. Minimal contrast injections must be used when performing angiography to mitigate the risk of propagation of dissection. Spontaneous coronary artery dissection is divided into three types using the Saw angiographic classification. Type 1 is characterized by multiple radiolucent lumens. Type 2 is described as intramural haematoma and diffuse stenosis. This diffuse narrowing may be bordered by normal artery segments proximally and distally (type 2a), or it may extend to the apical tip of the artery (type 2b). Type 3 mimics atherosclerosis.[11]

Intravascular imaging can be a useful adjunct to coronary angiography. It has been suggested that optical coherence tomography or intravascular ultrasound can be considered for type 2 SCAD and is required for the diagnosis of type 3 SCAD.[4] Intracoronary imaging may provide a definitive diagnosis; however, there are potential catastrophic risks, including extension of the dissection and catheter-induced vessel occlusion. It may be reserved for cases where there is diagnostic ambiguity and to aid percutaneous intervention.

The optimal management strategy for SCAD remains undetermined and there is a paucity of randomized controlled data in this area. Observational data have indicated angiographic healing of SCAD lesions in the majority of patients (70–97%) who were restudied weeks to months after a conservatively managed index episode.[11,12]

Medical therapy includes beta-blockers, antiplatelets, and angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Beta-blockers should be considered in patients with SCAD who have LV dysfunction or ventricular arrhythmias. Some advocate for their routine use on the basis of extrapolation from benefits in aortic dissection.[4] The exact role of antiplatelet therapy for SCAD is unknown. Most experts recommend aspirin for at least 1 year.[2] In

light of increased bleeding risks with antiplatelet agents, and uncertain benefits and risks, careful individual selection of suitability for dual-antiplatelet therapy in conservatively managed patients is recommended. Renin-angiotensin system antagonists should be used when MI is complicated by LV systolic dysfunction. Female patients of reproductive age must be warned of the teratogenicity of these medications.[2]

The value of follow-up angiography for SCAD is undefined. Computed tomography coronary angiography has been proposed as a potential alternative to conventional angiography although current data are limited to single case discussions and one small series.[13]

In terms of screening for potential predisposing conditions, the European Society of Cardiology (ESC) 2018 position paper advocates imaging of extra-coronary vascular beds in patients with SCAD.[14] Genetic screening and blood work to screen for inflammatory or connective tissue disease are of low yield and not routinely recommended.

Overall, cardiovascular outcomes for SCAD patients are relatively good with acute in-hospital mortality reported as <5%. Recurrent SCAD is frequent, occurring in ∼15% of patients at 2-year follow-up.[3] In patients with LV dysfunction, ejection fraction has been observed to improve after the acute presentation.[15] This may represent normalization of stunned myocardium following spontaneous resolution of SCAD.

Conclusion

This case highlights the importance of considering SCAD in a postpartum patient presenting with ACS, particularly in the context of environmental stressors such as illicit drug use. Coronary angiography is key to determine diagnosis and guide management. Conservative therapy is favoured, except for patients with ongoing ischaemia, haemodynamic instability, and left main stem involvement.

Lead author biography

Dr Laurna McGovern is a Specialist Registrar in Cardiology in Ireland.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

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

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