Post-Partum Spontaneous Coronary Artery Dissection Presenting as Congestive Cardiac Failure

A H Aaronson
North Devon District Hospital, UK

Corresponding author:
AH Aaronson,
North Devon District Hospital, UK
E-mail: amelia.aaronson@nhs.net

Case Presentation
A twenty-six year old woman presented on the medical take five months post-partum with a history of increasing shortness of breath for several days, minor pleuritic chest pain, and lower limb swelling.

Her past medical history included asthma, insulin-dependent diabetes mellitus, and HELLP syndrome during her recent pregnancy with significant gestational thrombocytopenia – although her platelet count had since returned to normal. No significant family history. Drug history included Lantus 18 units ON, Humalog 10 units TDS, and salmeterol 25mcg inhaler BD; no known drug allergies.

On examination she was tachycardic, no added heart sounds, had severe bilateral lower limb oedema extending to the thighs, was dyspnoeic at rest with orthopnea, and had reduced breath sounds bibasally with stony dullness to percussion, and bilateral wheeze. Due to the chest pain and signs of fluid overload, pericarditis and renal failure were respectively considered as differential diagnoses.

The patient had routine initial investigations: Urine dip was positive for nitrites, leucocytes, ketones, and haemoglobin; bloods showed slightly raised LFTs (ALT 63iU/L, ALP 238iU/L), reduced haemoglobin (97g/L), and a positive d-dimer (329mg/mL); other bloods were normal, including white cell count, electrolytes and renal function. Blood tests specific to the patient's history of HELLP, including platelets, lactate dehydrogenase, reticulocyte count and bilirubin, were also all normal.

Her ECG showed non-specific ischaemic changes but no ST elevation; chest radiography showed bilateral pleural effusions and pulmonary oedema, consistent with heart failure; an abdominal ultrasound scan to investigate possible renal failure showed a small left kidney, but nil else of note.

An echocardiogram revealed biventricular thrombi with an ejection fraction of 35%. The patient's care was then taken over by cardiology, and she was commenced on warfarin. There were concerns regarding a possible coronary artery thrombus, but she was too tachycardic to undergo a CT angiogram. Whilst in hospital she was investigated for anti-cardiolipin and lupus...
antibodies, but blood tests were all negative. The hospital’s heart failure team assessed her, and she was referred to the Community Cardiac Specialist Nurses for follow-up on discharge. The patient was discharged home after a six-day admission with warfarin, furosemide, ivabradine, ramipril, and spironolactone. A beta-blocker was not commenced due to her asthma. Further outpatient investigations were arranged for when her heart rate was more stable.

**Outcome and Follow-Up**

After discharge, the patient went on to have an outpatient cardiac MRI, which showed an infarct of the left anterior descending (LAD) artery territories, with a transmural enhancement, and associated hypokinesis of both ventricles.

She subsequently had a coronary angiogram, which showed diffuse LAD disease, focused around the origin of the principal diagonal branch. After discussion in the Multidisciplinary Team (MDT), this was thought to be secondary to spontaneous dissection of the coronary artery – however this was based on assumption and there were no other imaging modalities to confirm this.

Symptomatically the patient improved with medications for heart failure; her left ventricular function improved from 35% to 42%, her pleural effusions and peripheral oedema resolved, and dyspnoea improved to NYHA Grade One.

Currently this patient is not undergoing any further investigations, and her GP is to up-titrate her heart failure medications as necessary. The possibility remains for an intra-cardiac defibrillator in the future due to the risk of malignant ventricular arrhythmias. However this patient is likely to have a good prognosis; she responded well to medical management, and she has not experienced recurrent dissection six months on from the initial event, which has been shown to occur in approximately 50% of patients [1]. Previous published data shows that despite high mortality rates, the survival rate is now approximately 95% in patients who are diagnosed and treated early [1].

**Discussion**

There are several published cases and literature reviews into spontaneous coronary artery dissections. It is a rare cause of ACS – accounting for 0.1% of coronary angiograms [2]. The aetiology is broadly divided into atherosclerotic and non-atherosclerotic; although SCAD primarily affects young females, the atherosclerotic category is more common in males of older age [2]. Non-atherosclerotic SCAD is comprised of pregnancy-related, connective tissue disorders and autoimmune disease, including vasculitis [1].

The published cases are mainly of patients presenting with ACS despite having no risk factors. One such case appears to be the typical presentation – a young female presenting with chest pain, who had the diagnosis established with cardiac catheterisation, and consequently underwent coronary stenting [3]. The Netherlands Heart Journal describes a female four months post-partum, presenting with an acute inferolateral myocardial infarction secondary to SCAD who was treated with balloon angioplasty [2]; approximately one third of cases occur in the post-partum period, leading some studies to consider post-partum a separate aetiological category entirely [4].

As noted above, the majority of SCAD cases present as an acute myocardial infarction with typical cardiac chest pain. The presentation of the patient in this case report was unusual, and one particular case published in Heart and Vessels is noteworthy because of certain similarities. It describes a thirty-two year old woman who presented with symptoms of congestive cardiac failure and underwent similar investigations to this patient – chest radiography, echocardiogram and coronary angiography; she was found to have heart failure secondary to a spontaneous dissection of the LAD and right coronary arteries, and was treated with balloon angioplasty and stenting – however unlike this patient there was neither right ventricular failure nor biventricular thrombi [5].

The case reported here is not clear-cut; the diagnosis of SCAD is a difficult one to make due to its low prevalence, however in this patient it was made retrospectively and was a supposition rather than a confirmed diagnosis. The biventricular thrombi and cardiac failure do not classically fit with an LAD infarct, and coronary angiography showed diffuse disease and not a definite dissection as it was not done at the time of her initial presentation.

Given the retrospective nature of this patient’s diagnosis as well as its unusual presentation and findings – e.g. how LAD SCAD resulted in biventricular thrombi – it is important to consider the differential diagnoses. One of the major limitations of this case was the lack of investigations into some of these.

Despite the patient’s recent pregnancy and background of HELLP syndrome, conditions linked to these which were considered but not further investigated include thrombotic thrombocytopenic purpura (TTP), peripartum cardiomyopathy, and pulmonary embolism (PE).

TTP is an important differential due to the biventricular thrombi and history of HELLP syndrome, however it was not considered because her platelets had returned to normal levels soon after birth and her anaemia was not haemolytic. She also did not have other features of TTP such as renal or neurological signs and symptoms. Peripartum cardiomyopathy was not considered as she was five months postpartum – although this would still be considered within the time range of this condition, and the patient had diabetes mellitus – a known risk factor. As the diagnosis of SCAD was made retrospectively based on supposition, this should remain an important differential diagnosis.

A d-dimer was done on admission to rule out PE due to the pleuritic chest pain, tachycardia and signs of right-sided heart failure as well as risk factors such as pregnancy and history of thrombocytopenia. Yet despite a positive d-dimer, this was not further investigated because examination and ECHO showed biventricular cardiac failure rather than right-sided heart strain and there were no other signs to indicate thromboembolism such as haemoptysis or reduced oxygen saturations. Therefore it was not further investigated – although as with peripartum cardiomyopathy this should be considered as a differential.

Other important differentials include ACS secondary to a ruptured atherosclerotic plaque based on the angiography findings, vasculitis and other connective tissue disorders. This
patient did not have any investigations into any other possible causes of SCAD as it was thought likely to be post-partum – however as her case was atypical it might have been beneficial to investigate other aetiologies such as vasculitis and other autoimmune disorders.

Interestingly, although pregnancy and the post-partum period is a well-known risk factor for SCAD, its association with preeclampsia is less well reported on. The patient in this case report had a recent history of HELLP syndrome – a variant of preeclampsia. There is no firm causal association between pregnancy and SCAD, but it has been postulated that the haemodynamic and hormonal changes in pregnancy contribute to increased incidence of SCAD [6]. Physiological haemodynamic changes during pregnancy can increase stress on arterial walls, whilst hormonal changes are thought to alter their structure [2]. Preeclampsia is thought to be a further risk factor due to the increased vascular resistance and hypertension via vasoconstriction [7].

The management of SCAD is varied; one study showed that the majority of patients were medically managed, although some received either coronary artery stenting or coronary artery bypass grafting [1]. Another study showed that primary percutaneous coronary (PCI) intervention was the acute treatment of choice [2] – although that could not apply to this patient, as she was diagnosed retrospectively. Ultimately, SCAD is such a rare condition that there are no published guidelines on its management, and patients are assessed on a case-by-case basis. Generally patients with large areas of dissection or with haemodynamic instability are more likely to require intervention with PCI or bypass grafting, whilst smaller or stable lesions can be medically managed.

Learning Points

• Although uncommon, spontaneous coronary artery dissection is an important differential in patients presenting with acute coronary syndrome – especially younger female patients, patients without the usual risk factors for ACS, and pregnant or post-partum women.
• This case was not a definite diagnosis as detailed above, and it did not present typically. It is important to note that SCAD can occasionally present differently to ACS, such as this patient with symptoms of congestive cardiac failure.
• Although the mortality of SCAD is better than that of standard ACS, the complications are of a similar nature – such as myocardial infarction and congestive cardiac failure – and patients therefore require close follow-up [8]. If SCAD is suspected, it should be investigated and treated promptly due to the associated high morbidity and the risk of early recurrence in approximately 50% of patients [1].

Declarations of interest
The author declares no conflicts of interest.

Acknowledgements
The author states that she abides by the “Requirements for Ethical Publishing in Biomedical Journals” [9].

References