CASE REPORT

Pregnancy-Associated Spontaneous Coronary Artery Dissection: A Case Report and the Lessons from Practice

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ABSTRACT

Pregnancy-associated spontaneous coronary artery dissection (P-SCAD) is one of the fatal conditions that most commonly causes pregnancy-associated myocardial infarction. We herein report a case of P-SCAD in a 26-year-old multigravida woman, who complained of angina postpartum with raised cardiac biomarkers and transient ST-segment elevation on electrocardiograms. We emphasize the importance of a high degree of suspicion in the diagnosis of P-SCAD, together with the common presentation, the management approach, and its psychological impacts on the patients.
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INTRODUCTION

P-SCAD is the development of non-iatrogenic, non-traumatic and non-atherosclerotic coronary artery dissection during pregnancy or most commonly within the first week postpartum (1,2). It is estimated to happen in 1.81 per 100,000 pregnancies and contribute to almost 15% to 43% of pregnancy-associated acute myocardial infarction (2). Most of the P-SCAD cases occur in young women with few traditional cardiovascular risk factors. They usually present more severely with left ventricular failure, cardiogenic shock and multivessel dissection vis-a-vis those with SCAD not related to pregnancy (1).

CASE REPORT

A 26-year-old G3P2 woman at 37 weeks of gestation with no known medical illness antenatally, presented to our hospital after nine hours of contraction pain. She had an uncomplicated spontaneous vaginal delivery with a short second stage of the labour. An hour after the delivery, she complained of central crushing chest pain with a pain score of 5/10. She was slightly hypotensive with a blood pressure of 96/72 mmHg, which normalised shortly. The chest pain resolved spontaneously after ten minutes. Serial electrocardiograms (ECGs) revealed right bundle branch blocks with the progression of ST-segment elevation to T wave inversion and Q wave of the anterior chest leads (Fig. 1).

No family history of sudden cardiac arrest, cardiac disease or connective tissue disease was identified. She was initially deemed to have coronary artery vasospasm. However, her immediate highly sensitive troponin-I level was 32308 ng/L (reference interval [RI], < 54 ng/L) with a creatine kinase of 2946 U/L (RI, < 171 U/L) and an aspartate transaminase of 467 U/L (RI, < 33 U/L). Her troponin level increased to >50000 ng/L twelve hours later. Both her low-density lipoprotein and triglyceride levels were raised to 4.05 mmol/L and 3.61 mmol/L respectively. Urgent echocardiography demonstrated a reduced ejection fraction of 47% with left ventricular anterior and septal wall hypokinesis.

Coronary angiography with the aid of intravascular ultrasound (IVUS) revealed a dissection flap of about 1.5 cm in length extending from the ostial left anterior descending artery to the first large diagonal with a Thrombolysis in Myocardial Infarction (TIMI) grade three flow distally (Fig. 2 and 3). The other coronary arteries were normal. She was diagnosed with pregnancy-associated spontaneous coronary artery dissection

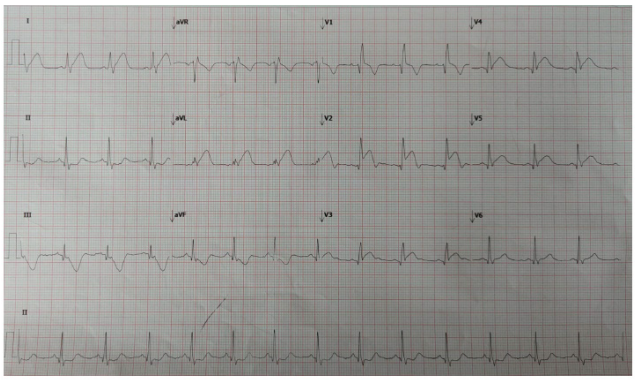

Figure 1: Serial Electrocardiograms showed right bundle branch blocks with the progression of transient ST-segment elevation to T wave inversion and Q wave of the anterior chest leads.

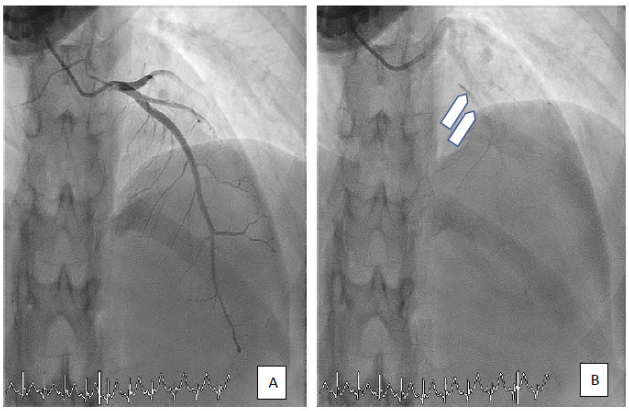

Figure 2: Left coronary angiogram in anteroposterior cranial view showed (A) normal TIMI grade three coronary blood flow in the left anterior descending artery and (B) type one spontaneous coronary artery dissection as evidenced by the contrast staining of the left anterior descending arterial wall (pointed by the white arrows).

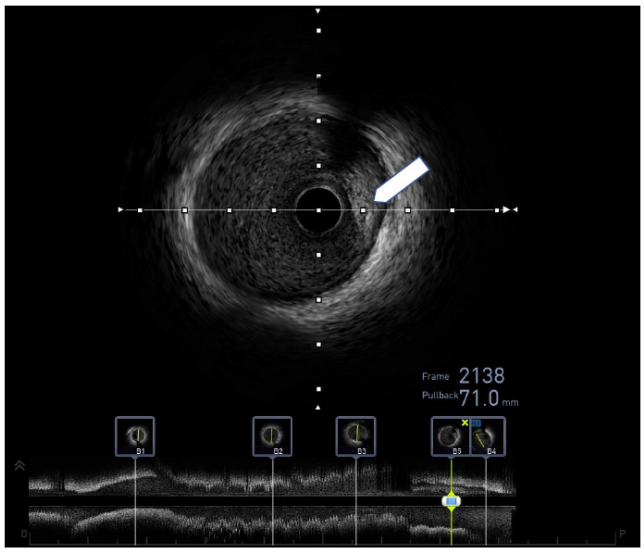

Figure 3: Intravascular ultrasound (IVUS) of the dissected left anterior descending artery showed hyperechogenic intramural haematoma in the false lumen (pointed by the white arrow).

(P-SCAD). Conservative management was opted for in this case. After being observed for another five days on the ward, she was discharged with atorvastatin 40 mg every night for her dyslipidaemia and outpatient cardiac magnetic resonance imaging as well as echocardiography appointments. She also consented to the insertion of an intrauterine contraceptive device.

DISCUSSION

The exact aetiology of SCAD is still unknown, albeit it has been almost a century since it was first described in 1931. The increased plasma volume and raised cardiac output during pregnancy were thought to precipitate P-SCAD by enhancing coronary artery demands together with the active Valsalva efforts during labour (3). The sudden increment in systemic blood volume due to swift post-delivery uterine contraction might explain the peak timing of P-SCAD within the first postpartum week (2,3). The surge of oestrogen during pregnancy and the rise in the oxytocin level toward the end of pregnancy have also been implicated in the pathophysiology of P-SCAD (3).

The patients typically present with angina with raised cardiac enzymes and ST-segment elevation on ECG as reported in this case, albeit complications such as sudden cardiac death, arrhythmias or cardiogenic shock can be the patients' first clinical presentation (2). The initial assumption of coronary artery vasopasm in this case might be lethal due to the fatal consequences of the clinical entity if the patient is not closely monitored in a coronary care unit. Coronary angiography remains the gold standard diagnostic tool with the cautious use of IVUS and optical coherence tomography if necessary due to the concern of increased risk of iatrogenic dissection (4).

Hence, a conservative approach is the preferred acute management (1,2). Percutaneous coronary intervention (PCI) and coronary artery bypass grafting which are commonly practised in atherosclerotic lesions, are generally not advocated in SCAD unless in the cases with haemodynamic instability, evidence of ongoing ischaemia and disrupted distal flow in the culprit vessel as they have been associated with a higher risk of complications (1,2). Similarly, thrombolysis is contraindicated as cases of dissection extension and rupture of coronary arteries have been reported because of the therapy (1). In addition, majority of the case series reported across the globe showed 68-100% dissection healing in conservatively managed patients who underwent repeat angiography (1). Beta-blockers and angiotensin-converting enzyme inhibitors are mostly reserved for patients with reduced ejection fraction, as in the case of statins for patients with dyslipidaemia (1).

Dual antiplatelet therapy (DAPT) in the case of SCAD, remains controversial, but is recommended by some

authors for at least two to four weeks after the coronary event, followed by low-dose aspirin alone for a total of three to twelve months (2). Conversely, others might be hesitant on the use of DAPT, considering its non-atherosclerotic pathophysiological mechanism, the uncommon intracoronary thrombus and the augmented bleeding risks even with aspirin monotherapy (2). According to a recent observational study by Cerrato et al., 2022 carried out in Italy and Spain, conservatively managed patients with SCAD who were started on DAPT, were associated with higher major adverse cardiovascular events than those on single antiplatelet therapy (5). These were predominantly due to non-fatal myocardial infarction or unplanned PCI (5). Based on the data, antiplatelet therapy may be particularly detrimental to the conservatively managed SCAD patients with intramural haematoma (5). In the context of our patient, her symptom resolved with the left main coronary artery being unaffected and the non-obstructive coronary arteries being found on coronary angiogram. Conservative management without antiplatelet therapy was thus adopted.

Subsequent follow-up is equally pivotal in the care of the patients. Echocardiography should be repeated at three months for patients with heart failure to reassess cardiac function (2). Besides, long-acting progesterone-only methods are the favoured contraceptive option as oestrogen-containing alternatives may simulate the hormonal surge in pregnancy, leading to an increased theoretical risk of P-SCAD (2). In terms of the physical activity after P-SCAD, it is not uncommon that the patients will be worried about its association with the recurrence of P-SCAD. Vigorous exercises and physical activities that require straining are generally not recommended (2). However, many studies have proven that a tailored cardiac rehabilitation program not only is safe for patients with a history of SCAD but improves their physical and mental well-being (2,4).

Last but not least, the uncertainty in the aetiologies and the lack of secondary prevention options often leave tremendous psychological impacts on the vulnerable patients (4). They should not be overlooked as they can result in post-traumatic stress disorder, depression and anxiety (4). The under-recognised psychological repercussions should be actively screened and tackled by a multidisciplinary team and social support groups.

CONCLUSION

This case highlights the importance of recognising P-SCAD as an important cause of myocardial infarctions in the gravid and postpartum patients. The initial management approach could have been detrimental to the patients if thrombolytic therapy were to be given for the deemed atherosclerotic aetiology. Hence, a high degree of suspicion is required to avert the diagnosis from being missed. It is hoped that more research initiatives will be carried out to shed some light on the disease and to enlighten the physicians in managing such a fatal and challenging clinical entity.

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