Spontaneous coronary artery dissection (SCAD): a rare cause of acute myocardial infarction in a young post-partum woman

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Abstract

Spontaneous coronary artery dissection (SCAD) is a non-traumatic and non-iatrogenic separation of the coronary arterial wall. It is an uncommon cause of acute coronary syndrome especially in young women.

We report a case of 32-year-old post-partum women who presented with out of hospital cardiac arrest and subsequently diagnosed to have massive myocardial infarction likely secondary to SCAD with incidental pulmonary embolism.

Case presentation:

A 36-year-old lady presented to hospital following an out of hospital cardiac arrest. She successfully had return of spontaneous circulation (ROSC) after thirty minutes of cardiopulmonary resuscitation (CPR). Her cardiac rhythm was a combination of pulseless electrical activity and asystole throughout the CPR. Post ROSC electrocardiogram (ECG) showed complete heart block with HR of 30. Atropine 500 micrograms IV boluses to a total dose of 3 milligrams were administered with no response to heart rate.

External pacing was initiated for complete heart block.

She did not have significant past medical history; however, she was 10 days post-partum following emergency caesarean section at 37 weeks of pregnancy due to foetal distress and breech presentation (4TH child). Further collateral history revealed that she reported left arm pain for the last 3 days and three fainting episodes prior to the cardiac arrest. No chest pain was reported. Did not have any history of thromboembolism and no family history of sudden cardiac death. She was a smoker of about 5 packs year. No history of alcohol intake and recreational drug use.

She was haemodynamically unstable needing vasopressor support. Lungs were clear on auscultation, no cardiac murmurs. Abdomen was soft with clean Caesarean scar. No per-vaginal bleeding was noted. She was subsequently transferred to ITU.

Bloods showed significantly raised D-dimer of 43997 ng/ml (normal (230ng/ml) and Troponin of 936ng/L (normal <14ng/L) with repeat Troponin of 6747ng/L. ABG showed type 1 respiratory failure with metabolic acidosis. Differentials of massive pulmonary embolism and myocardial infarction were considered. Treatment for both acute coronary syndrome and pulmonary embolism were started.

CT pulmonary angiogram (CTPA) was done which showed pulmonary embolism in the right lower segmental and subsegmental branches of pulmonary artery. No cardiac thrombus or right heart strain. It also showed soft tissue in the left lower lobe bronchus with near collapse of the left lower lobe likely due to aspiration. However, it is unlikely that pulmonary embolism was the likely cause of cardiac arrest in this patient as it was only a segmental pulmonary embolism.

Transthoracic echocardiogram showed normal left ventricular (LV) size but significant LV impairment with ejection fraction (EF) of <20%. Inferior and septal walls were akinetic. Apex appeared thin and rounded. She was then transferred to cardiothoracic centre and externalized dual chamber pacemaker was inserted.

Subsequent coronary angiogram demonstrated hazy appearance in the proximal LAD suggestive of thrombus and the likely cause was SCAD event or an acute plaque rupture. However, SCAD was the likely diagnosis as she was post-partum and multiparous and no convincing risk factors for atherosclerosis.

Plan for repeat angiogram with possible PCI was made should she recover from multiorgan failure. Unfortunately, she deteriorated due to multiorgan failure and was not deemed fit for ECMO and heart transplant. She subsequently passed away.

Discussion:

SCAD is the cause of acute coronary syndrome (ACS) in 0.1 to 4 percent of cases (1).

The underlying mechanism of non-atherosclerotic SCAD is not fully understood, but an intimal tear or bleeding of vasa vasorum with intra-medial haemorrhage has been proposed (2). Both result in creation of a false lumen filled with intramural hematoma. Pressure-driven expansion of the false lumen by an enlarging hematoma may lead to luminal encroachment and subsequent myocardial ischemia and infarction [3]. Atherosclerotic cause of coronary dissection is a mechanistically distinct variant and is typically limited in extent by medial atrophy and scarring (4).

In pregnant or early postpartum women, dissection may be a consequence of increased physiological hemodynamic stresses or from hormonal effects weakening the coronary arterial wall (5). The exposure to recurrent and chronic hormonal pregnancy changes can further increase SCAD risks in women with multiple previous births (multiparity), and dissection in all arterial beds are more common during pregnancy (6).

Potential predisposing factors include fibromuscular dysplasia (FMD), postpartum status, multiparity (≥4 births), connective tissue disorders, systemic inflammatory conditions, and hormonal therapy. However, up to 20 percent of cases are labelled as idiopathic (7).

In this case report, two significant risk factors (early post-partum and multiparity) for SCAD were present.

Patients with SCAD present with signs and symptoms of acute myocardial infarction, the most common being chest pain. Less common symptoms include arm pain, back pain, neck pain and sweating.ST-elevation MI (STEMI) was present in 25 to 50 percent of patients, with the remainder presenting with non-ST-elevation MI (8). Life-threatening ventricular arrhythmias occurred in 4 to 14 percent (8). Cardiogenic shock was reported in 2 to 19 percent of patients with SCAD, depending on the proportion of patients with STEMI (9).

Our case report reflects above presentation as she presented left arm pain, NSTEMI and cardiogenic shock.

The left anterior descending coronary artery (LAD) was the most frequently affected vessel (approximately 40 to 70 percent of cases) (8). In our case report, LAD was the affected coronary artery. Diagnosis of SCAD is made at the time of coronary angiography. Imaging such as OCT or IVUS and cardiac MRI will help in the diagnosis.

In most SCAD patients, conservative therapy is the preferred strategy after the diagnosis is secured (09). However, the optimal management is uncertain, in part due to the limited clinical experience. A wide range of approaches, including conservative management, emergency revascularization with percutaneous coronary intervention (PCI) or coronary artery bypass grafting (CABG), fibrinolytic therapy (with or without subsequent PCI), mechanical hemodynamic support, and cardiac transplantation have been reported (8). Patients presenting with acute myocardial infarction who have symptoms of ongoing ischemia or hemodynamic compromise should be considered for revascularization with PCI or coronary artery bypass grafting (8).

However, in our case, patient quickly deteriorated and was deemed not fit for any further interventions and subsequently passed away.

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