Acute myocardial infarction due to spontaneous coronary artery dissection in a twin pregnancy
Disseção espontânea das artérias coronárias numa gravidez gemelar: descrição de caso clínico

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Abstract

Spontaneous coronary artery dissection is a rare event during pregnancy, resulting in significant acute maternal and foetal mortality rates. We report a case of a mid-left anterior descending artery type I dissection in a 34-year-old primigesta with a 24-weeks monochorionic diamniotic twin pregnancy. She was treated medically with favourable outcome. Although quite rare in obstetric practice, spontaneous coronary artery dissection should be considered in the differential diagnosis of chest pain in pregnant women.

Keywords: Chest pain; Myocardial infarction; Spontaneous coronary artery dissection; Twin pregnancy; Pregnancy complications.

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) represents an important but underrecognized cause of acute coronary syndrome (ACS) and myocardial infarction (MI) in pregnant and post-partum patients¹. Up to 1 in 16,000 pregnancies are complicated by an acute MI² ³. It is a potentially catastrophic condition, with a fatality rate reaching 4.5%⁴.

Pregnancy-related SCAD commonly occurs during the late third trimester or the first month after delivery⁵ ⁶. Clinical presentation can vary widely, posing a diagnostic challenge⁴. Prompt recognition of cardiac symptoms and coronary angiography tends to improve maternal and foetal survival rates⁶. Given the potential for severe outcomes and a recurrence risk of up to 12.5%⁷, pregnancy is not recommended after a SCAD-event⁸ ⁹. Thus, proper contraceptive counselling must be provided⁹.

CASE REPORT

We present the case of a 34-year-old primigesta with a spontaneous monochorionic diamniotic twin pregnancy, complicated by selective intrauterine growth restriction (sIUGR) after the 15th week. Subsequent twin-twin transfusion syndrome (TTTS) was diagnosed by oligohydramnios/hydramnios sequence along with critically abnormal Doppler of the donor twin. She was admitted at 24 weeks and 3 days in order to provide maternal and foetal surveillance. Two days after admission, the patient developed sudden epigastric discomfort along with nausea and vomiting, and sharp, severe and constant chest pain radiating to the right arm. The patient had no cardiovascular risk factors, and no tobacco, alcohol or drug abuse. Physical examination revealed: arterial blood pressure 135/86mmHg on the left

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and 141/90 mmHg on the right arm, ample and regular pulse (82 beats.min⁻¹) and normal cardiac and pulmonary auscultation. Symptomatic treatment with acetaminophen, diazepam and metoclopramide was administered. An electrocardiogram (ECG) showed nonspecific repolarization changes and obstetric ultrasound revealed donor foetal demise. Laboratory assessment demonstrated normal blood count and coagulation, lactate dehydrogenase (LDH) 850 U/L, aspartate aminotransferase (AST) 506 U/L, alanine transaminase (ALT) 166 U/L and normal creatine kinase (CK), creatine kinase muscle/brain (CK-MB) and myoglobin. First troponin T test was slightly elevated – 0.065 ng/mL [normal < 0.057 ng/mL].

She underwent an emergency caesarean section for atypical preeclampsia, with birth of a 580g girl (5/5/8 Apgar score) and a 425g stillbirth. Surgical inspection showed no hemoperitoneum and a normal hepatic examination and ECG monitoring revealed no ST segment/T wave changes.

She remained drowsy during the postoperative period. Analytical control at 8 hours postpartum showed a troponin 1 level of 187.9 ng/mL, along with ECG T wave inversion in anterolateral leads. In the Emergency Room, handheld echocardiography revealed apical, anterior and lateral wall hypokinesia, with a mildly depressed left ventricular function, (left ventricular ejection fraction of 43%). A high-risk non-ST elevation ACS (NSTEMI) was suspected and the patient underwent emergent transradial coronary angiography for risk stratification. Angiography revealed a mid-left anterior descending (LAD) artery type I dissection (Figure 1). Due to haemodynamic stability and patent artery flow, a conservative strategy without percutaneous coronary intervention (PCI) was adopted.

The patient was admitted to the Coronary Care Unit asymptomatic and with normal hemodynamic parameters (arterial blood pressure 135/70mmHg, resting heart rate 75beats.min⁻¹). She was given dual antiplatelet therapy (aspirin 100mg qd and clopidogrel 75mg qd), low molecular weight heparin 1mg.kg⁻¹ bid, carvedilol 6.25mg bid and atorvastatin 40mg qd, and was discharged 8 days later without events. The discharge echocardiogram revealed a mildly depressed LVEF (45%).

Neonatal death of the second twin occurred at day 4 of life due to septic shock. Five months after the event, the patient remained asymptomatic and progestin-based contraception was initiated. Screening for an underlying autoimmune disorder with antinuclear antibodies (ANA), anti-neutrophil cytoplasmic antibodies (ANCA) and antiphospholipid (APL) antibodies was negative.

**DISCUSSION**

SCAD is an under-diagnosed cause of AMI during pregnancy, with an incidence for 1.81 in 100000 pregnancies⁵. Current use of high sensitivity troponin assays, early angiography in ACS and widespread availability of intracoronary imaging has led to a greater awareness of this clinical entity, with incidences of 0.07 to 0.2% of all angiograms and 2 to 4% of ACS-driven angiograms¹⁻⁸⁻¹₀. SCAD has been associated with fibromuscular dysplasia, connective tissue disorders and autoimmune diseases¹⁻³⁻¹¹. The predilection for female patients and the association with pregnancy suggests an etiological role for sex hormones⁵. Increased cardiac output during pregnancy had also been implicated as an etiological factor⁵. Additionally, emotional stressors have been reported in a high proportion of SCAD cases¹. In our case, as the postpartum screening for an underlying cause was negative, we might speculate that the pregnancy-related emotional stress associated with severe TTTS at the fetal viability threshold, along with a particularly exacerbated cardiac output in a twin preg-
Elevation of liver function tests (LFTs) was due to myocardial necrosis\(^{13,14}\). Not verified under surgical exploration. In this case, the diagnosis was made in the diagnosis of atypical preeclampsia and eventual subcapsular liver hematoma. That diagnosis was not verified under surgical exploration. In this case, the elevation of LFTs was due to myocardial necrosis\(^{13,14}\).

Our case poses an additional diagnostic challenge, as it presented early in pregnancy. Although described from as early as 5 weeks\(^3,5\), most cases occur late in the third trimester or in the peri- and postpartum period, with report rates ranging from 26 to 72.5\(^7,8,11\).

Due to the haemodynamic stability and patent artery flow, medical management was adopted in our case. This strategy is supported by multiple reports, main dissection, ongoing ischemia, ventricular arrhythmias or haemodynamic instability\(^3,5\).

Finally, our case raises an additional point of discussion regarding the patient’s reproductive future.

Given its potential for major complications, such as 10-year major adverse cardiac events and recurrence rates of 47.4\(^{11}\) and 29\(^%\)\(^{13}\) respectively, pregnancy after a SCAD-event is not currently recommended\(^13,37\). However, since it was a primigesta, a subsequent high-risk pregnancy is very likely; deferring it for at least 1 year is recommended\(^2\).

This case deserves to be highlighted by its rare presentation in a primigesta with a twin pregnancy complicated by the death of both foetuses. In the absence of reliable prevention strategies, it is necessary to establish guidelines for the surveillance of high-risk pregnancies after a PSCAD episode.

**CONFLICT OF INTEREST**

The authors declare that they have no conflict of interests.

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251

Inês Coutinho et al.