

Case Report

Spontaneous dissection of the left main coronary artery in a puerperal woman treated with percutaneous coronary intervention

Júlio Cesar Machado Andréa*, Priscilla Costa Mendonça, Hélio Roque Figueira, Jacob Atié, Luiz Paulo Machado, Luis Felipe Camillis

Clínica São Vicente, Rio de Janeiro, RJ, Brasil

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ABSTRACT

Spontaneous coronary dissection is an uncommon cause of acute coronary syndrome that may manifest as unstable angina, acute myocardial infarction, or sudden death. The condition occurs predominantly in women, with approximately one-third of cases reported during or after pregnancy. The etiology is unknown; however, associations with fibromuscular dysplasia and connective tissue diseases have been reported. Here, we describe a case of ST-segment elevation acute myocardial infarction in a puerperal woman due to spontaneous dissection of the left main coronary artery, successfully treated with percutaneous coronary intervention.

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Dissecção espontânea do tronco da coronária esquerda em puérpera tratada com intervenção coronária percutânea

RESUMO

A dissecção coronária espontânea é uma causa incomum de síndrome coronária aguda. Pode se manifestar como angina instável, infarto agudo do miocárdio ou morte súbita. Ocorre predominantemente em mulheres e, em cerca de um terço dos casos, durante ou após a gravidez. A etiologia é desconhecida, embora a associação com displasia fibromuscular e doenças do tecido conjuntivo tenha sido relatada. Os autores descrevem um caso de infarto agudo do miocárdio com supradesnivelamento do segmento ST em puérpera por dissecção espontânea do tronco da artéria coronária esquerda tratada com sucesso por intervenção coronária percutânea.

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Palavras-chave:

Dissecção
Infarto do miocárdio
Angioplastia
Período periparto

Introduction

Acute myocardial infarction (AMI) is a rare complication of pregnancy or puerperium that affects six out of every 100,000 pregnancies based on the US Nationwide Inpatient Sample. The risk is increased three- to four-fold compared with that for non-pregnant women of a similar age.¹ Spontaneous coronary artery dissection (SCAD) related to pregnancy is the most frequent cause of AMI among women in the fertile period, responsible for approximately 40% of the cases, making it more common than atherosclerosis, thromboembolism, vasospasm or Takotsubo syndrome.²

The term "peripartum spontaneous coronary artery dissection" has been the most widely adopted term for this condition because it

reflects a more comprehensive period, i.e., from conception to months after delivery. However, the condition predominately occurs between the last trimester of pregnancy and up to 6 weeks postpartum. Its mechanism is not fully understood but is likely attributed to the interaction between hemodynamic and hormonal changes induced by pregnancy.

Case report

A 35-year-old patient (7-day postpartum, cesarean delivery, G2P2) was admitted to the emergency department reporting severe abdominal pain with shoulder irradiation lasting 6 hours and evolving

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Corresponding author: Clínica São Vicente, Rua João Borges, 204 parte Hemodinâmica, Gávea, CEP 22451-100, Rio de Janeiro, RJ, Brasil.

E-mail: julioandrea@gmail.com (J.C.M. Andréa).

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with general malaise, sweating, and dyspnea. She had no pathological history or comorbidities and no history of smoking or illicit drug use. The patient had a family history of Takotsubo syndrome. On admission, her blood pressure was 150/90 mmHg, her heart rate was 96 bpm and her peripheral arterial oxygen saturation was 94%. Cardiac and pulmonary auscultations were normal. An electrocardiogram revealed ST-segment elevation in the anterolateral leads with mirror-image change in the inferior leads (Figure 1). The first high-sensitivity troponin T was 57 pg/mL (upper reference limit: up to 15.6 pg/mL). An emergency two-dimensional echocardiogram was ordered, which revealed segmental wall-motion abnormalities due to anterolateral hypokinesia and anteroapical akinesia with a left ventricular ejection fraction of 48%.

Once the diagnostic hypothesis of AMI was confirmed, the patient was referred to the cath lab, and pharmacological measures were taken (loading dose of 200 mg of acetylsalicylic acid and intravenous nitroglycerin). During this interval, the patient presented with ventricular fibrillation, which was reversed, and the patient was put on mechanical ventilation. Selective coronary angiography performed via right radial access with a 6 Fr introducer revealed a right coronary artery with no obstructive lesions, imaging compatible with dissection involving the left main coronary artery (LMCA), acute occlusion of proximal left anterior descending artery (LAD) and slowed flow (Thrombolysis in Myocardial Infarction – TIMI 2) in the left circumflex artery (LCx) (Figure 2).

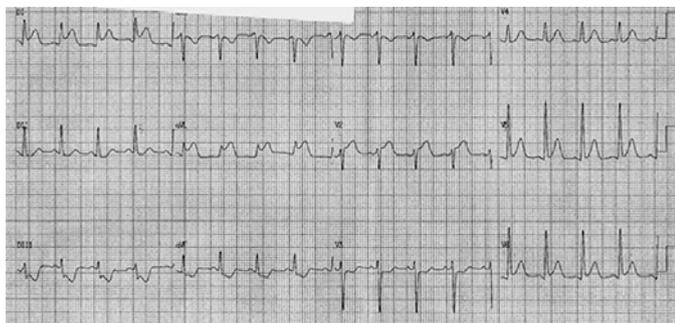


Figure 1. Electrocardiogram depicting ST-segment elevation in the anterolateral leads.

After systemic heparinization (100 U/kg), percutaneous coronary intervention (PCI) was performed using a JR4 guiding catheter. Next, 0.014" BMW™ guidewires (Abbott Vascular, Santa Clara, USA) were positioned for the true lumens of the LAD and LCx. Then, implantation of two overlapping everolimus-eluting stents (Abbott Vascular) was performed (3.0 x 38 mm and 3.0 x 28 mm in the LMCA and LAD). This approach resulted in normalization of blood flow, but progression of an intramural hematoma and progressive narrowing of the LCx ostium was noted (Figures 3A to 3D). Opting for a lateral branch approach (LCx) and using the T and protrusion (TAP) technique, a third everolimus-eluting stent (2.75 x 15 mm) (Figures 3E to 3I) was implanted. The procedure was finalized with kissing balloons and proximal optimization with non-compliant balloons, resulting in endoluminal reconstruction of the affected segments (Figures 3J to 3M). At the end of the procedure, intravascular ultrasound was performed, revealing stent struts with good apposition and in-stent luminal areas ranging from 10.9 to 13.8 mm² (Figure 4).

The patient was transferred to the intensive care unit on mechanical ventilation with intravenous infusion of noradrenaline and abciximab. The in-hospital evolution was satisfactory with resolution of the ST-segment elevation, removal of the ventilatory support 12 hours after PCI, normalization of myocardial necrosis markers and echocardiographic reassessment with anteroapical hypokinesia and left ventricular ejection fraction at 58%. She was discharged on the seventh day on acetylsalicylic acid, ticagrelor and bisoprolol.

One year after the procedure, the electrocardiogram showed a small Q wave in D₁ and aVL (Figure 5), and the echocardiogram revealed normal ventricular dimensions and thickness with no contractile deficit and preserved ventricular function. Coronary computed tomography angiography after 15 months revealed no signs of in-stent restenosis (Figure 6). At the 18-month clinical follow-up, the patient remained asymptomatic using dual antiplatelet therapy and following a cardiac rehabilitation program.

Discussion

Pregnancy-associated SCAD is the most common cause of AMI in pregnant women, occurring predominantly in the puerperium and in multiparous women. Other associated risk factors include black

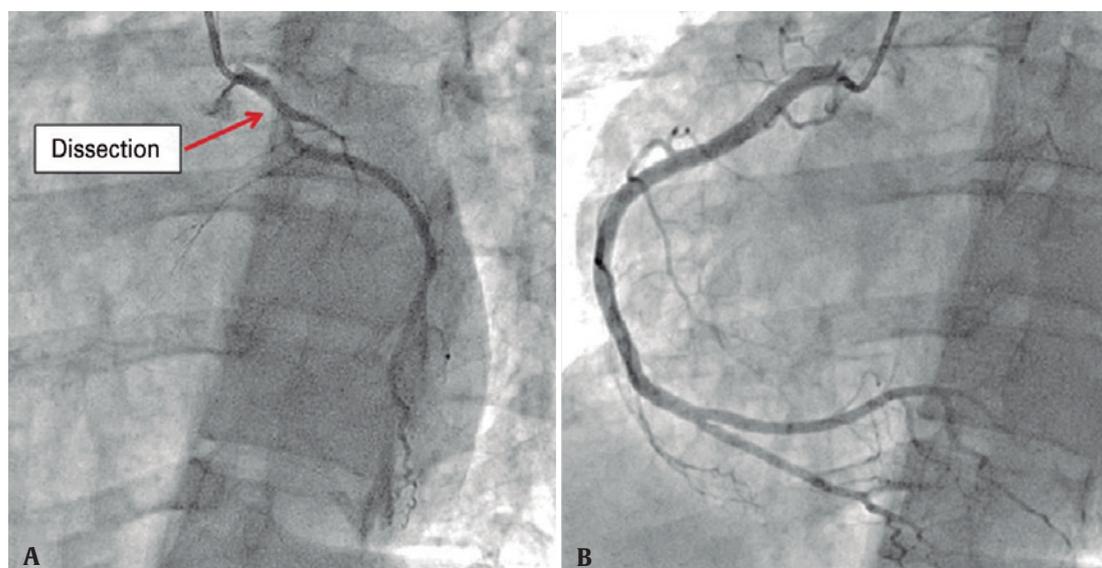


Figure 2. (A) Left coronary artery in left anterior oblique projection: left main coronary artery dissection (arrow) with proximal occlusion of the left anterior descending artery. (B) Right coronary artery in left anterior oblique projection: no obstructive lesions.

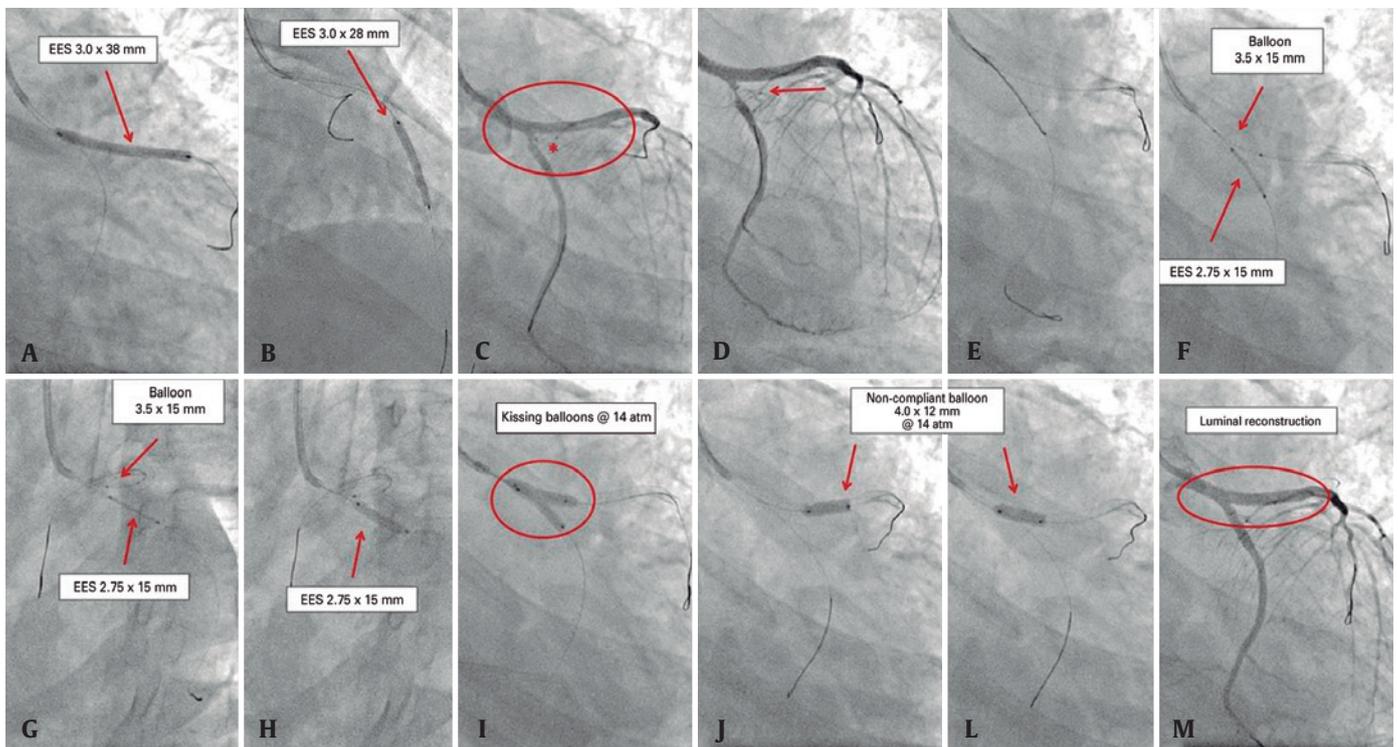


Figure 3. (A, B, and C) Implantation of two everolimus-eluting stents (EES) (3.0 x 38 and 3.0 x 28 mm) overlapping in the left anterior descending artery and left main coronary artery. (D) Narrowing of the left circumflex artery ostium due to progression of a hematoma. (E) Approach to the left circumflex artery. (F, G, H, and I) with the T and protrusion technique. (J and L) Proximal optimization with non-compliant balloons (NC). (M) Final result with endoluminal reconstruction.

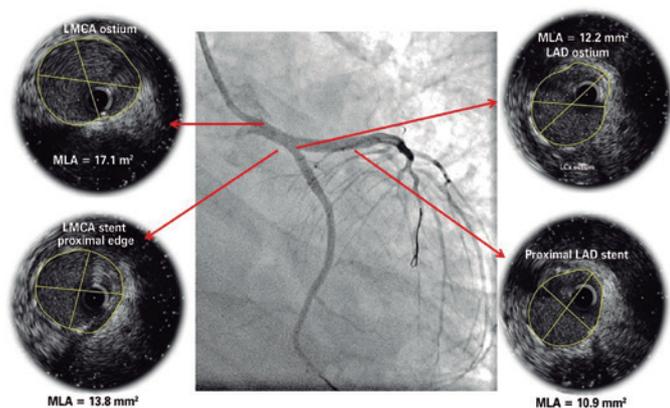


Figure 4. Intravascular ultrasound depicting well-placed struts and intrastent minimal luminal area (MLA) ranging from 10.9 to 17.1 mm². LMCA: left main coronary artery; LAD: left anterior descending artery; LCx: left circumflex artery.

race, systemic arterial hypertension, dyslipidemia, depression, migraine, advanced maternal age, and infertility treatment.³ Clinical manifestations include dyspnea, chest pain, congestive heart failure, acute coronary syndrome, ventricular arrhythmia, sudden death, and cardiogenic shock.⁴

Its pathophysiology has still not been fully elucidated. However, the condition is influenced by the interaction among hemodynamic, hormonal and vascular factors during pregnancy when there is a physiological increase in cardiac output and estrogen and progesterone levels, thus favoring histopathological changes in the tunica media of the arteries with disorganization of collagen and elastic fibers, cystic necrosis and inflammatory infiltrates.⁵ Labor may induce an increase in shear forces, precipitating dissections in coronary arteries. These

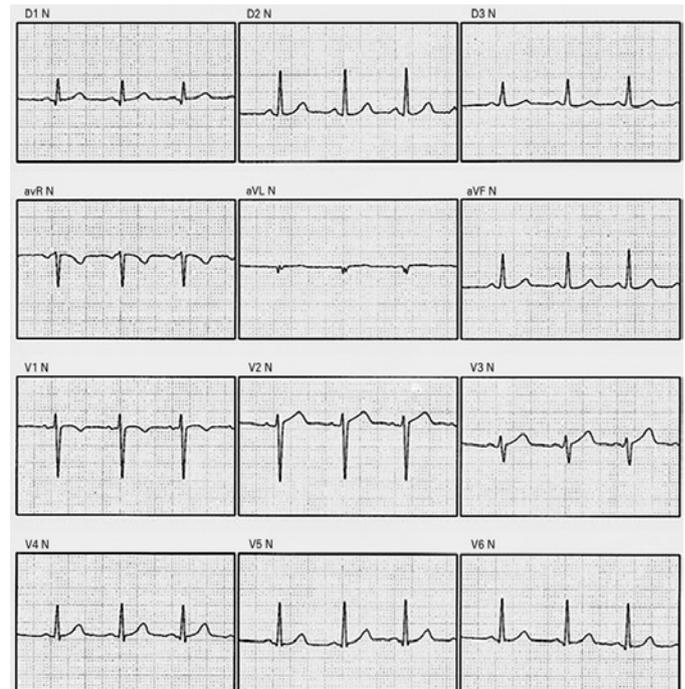


Figure 5. Electrocardiogram 1 year after the procedure depicting a small Q wave in D₁ and aVL.

changes may persist for more than 6 months, which would explain the occurrence of late-onset SCAD during breastfeeding.⁶ Other systemic conditions, such as fibromuscular dysplasia, connective tissue diseases and autoimmune diseases, may eventually develop.⁷⁻⁹

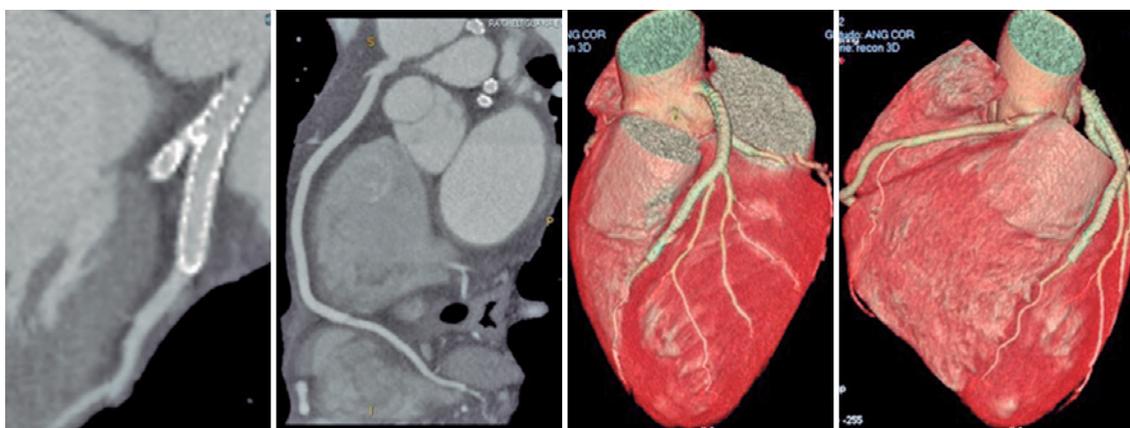


Figure 6. Control coronary computed tomography angiography 15 months after the procedure without signs of in-stent restenosis.

In a series recently published by Cade et al., including 13 patients in 11 centers in Brazil, the authors analyzed the findings of multimodal imaging (angiography, computed tomography, intravascular ultrasound and optical coherence tomography), acute-phase treatment and in-hospital outcomes.¹⁰ Mortality was observed in 7.7% of the cases. Clinical presentation of AMI was noted in 46% of cases. Approximately one-third of the women had cardiogenic shock. In total, 46% had involvement of the LMCA, and 69% had multiple dissections.

The management of pregnancy-associated SCAD depends on the clinical presentation, the number of vessels involved and the amount of myocardium at risk. In stable situations with single-vessel involvement, conservative treatment is an acceptable option.¹¹ In the presence of hemodynamic instability or a large area of myocardium at risk, the option for revascularization is imperative, as noted in the case reported here. PCI can be the initial strategy, however access to the true lumen can be challenging, especially in cases of intimal rupture. Complementary imaging methods, such as intracoronary ultrasound or optical coherence tomography, can help in confirming the correct location, sizing and implantation of the stents.^{12,13} Spontaneous reabsorption of intramural hematoma occurs over time, and the possibility of acute or late malposition with a consequent risk of stent thrombosis would make the use of bioabsorbable platforms attractive, although this technology has shown little encouraging results lately.¹⁴ Surgical treatment applies to circumstances in which PCI is not possible.

Questions regarding adjunctive antithrombotic therapy, such as heparin and glycoprotein IIb/IIIa inhibitors, are relevant given the risk of intramural hematoma propagation. In the present report, we made our determinations based on the exacerbated pro-thrombotic state and extensive reconstructed arterial segment with multiple stent implantations. Maintenance of dual antiplatelet therapy for 18 months following the procedure in individual cases is supported by current guidelines.¹⁵

Conclusions

Spontaneous coronary dissection is the main cause of acute coronary syndrome during the peripartum period and should be consid-

ered in the differential diagnosis of chest pain in young women. The early recognition of this clinical entity allows for better risk stratification and therapeutic approaches.

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