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Spontaneous coronary artery dissection as a cause of ST-segment elevation myocardial infarction

Dissecção espontânea de artéria coronária como causa de infarto agudo do miocárdio com supradesnívelamento do segmento ST

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ABSTRACT – Spontaneous coronary artery dissection is an uncommon condition, and the patients' clinical presentation is often underestimated due to few risk factors for atherosclerotic disease. Treatment must be individualized, with conservative therapy as the first option, respecting the criteria for referral for interventional treatment. We report a case of spontaneous coronary dissection, initially manifested as a non-ST segment elevation acute coronary syndrome, progressing to transmural infarction, in a young patient, with few risk factors for coronary artery disease, and give examples of difficulties related to the percutaneous approach.

Keywords: Coronary artery disease; Myocardial infarction; ST segment elevation myocardial infarction

RESUMO – A dissecção espontânea de artéria coronária é uma patologia pouco comum, e, frequentemente, o quadro clínico dos pacientes é subestimado devido a poucos fatores de risco por doença aterosclerótica. O tratamento deve ser individualizado, sendo a terapia conservadora a primeira opção, respeitando-se os critérios de indicação para tratamento intervencionista. Reportamos um caso de dissecção coronária espontânea, manifestado inicialmente como síndrome coronária aguda sem supradesnívelamento do segmento ST, evoluindo para infarto transmural, em paciente jovem, com poucos fatores de risco para doença arterial coronariana, exemplificando as dificuldades relacionadas ao manejo percutâneo.

Descritores: Doença da artéria coronariana; Infarto do miocárdio; Infarto do miocárdio com supradesnível do segmento ST

INTRODUCTION

The first case of spontaneous coronary artery dissection (SCAD) was described by Pretty in 1931.¹⁻³ This condition is an uncommon cause of acute myocardial infarction (MI),^{4,5} accounts for 0.1% to 4% of acute coronary syndrome cases,^{3,4} and affects mainly young and/or female patients.^{3,6,7} Conservative treatment is the preferred approach, and revascularization is reserved for cases of ischemia that are refractory to medical treatment or for high-risk situations, such as hemodynamic instability or involvement of the left main coronary artery.^{3,5-7}

The aim of this study was to report a case of a female patient, middle-aged, initially diagnosed with non-ST segment elevation acute coronary syndrome, who progressed with dynamic electrocardiographic changes and ST segment elevation, in addition to uncontrolled pain, and underwent urgent percutaneous myocardial revascularization.

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CASE REPORT

A 51-year-old woman, previously hypertensive and former smoker, with anxiety disorder and with four previous abortions. She sought emergency service complaining of precordial pain, which had started 7 hours before and had worsened one hour before, a tightness sensation of moderate intensity, irradiating to the left upper limb, back and tongue, accompanied by dyspnea, vertigo and diaphoresis.

She was hemodynamically stable, and an initial electrocardiogram (ECG) showed no acute ischemic changes. Initially, an anxiety attack was suspected and she was medicated with diazepam, but laboratory tests showed positive troponin in a curve (0.595mg/dL, 0.932mg/dL and 0.508mg/dL – reference value <0.1mg/dL), and was diagnosed as a non-ST-segment elevation myocardial infarction.

Acetylsalicylic acid 300mg, clopidogrel 300mg, and enoxaparin 1mg/kg were prescribed, in addition to statin and beta-blocker.

On the second day of hospitalization, the ECG showed inversion of the T wave in the anterior wall, and on the third day, the patient had recurrence of severe chest pain, associated with electrocardiographic alteration with ST-segment elevation in the inferior wall, with inversion of the T wave in the anterior and inferior wall.

Intravenous therapy with nitroglycerin was started, and the patient progressed with ST-segment elevation also in the anterior wall (Figure 1). She was referred to urgent coronary angiography, which showed extensive spontaneous dissection of the left anterior descending artery (LAD) (Figure 2). After crossing a guidewire through the

Figure 1. Electrocardiogram with ST-segment elevation in the anterior and inferior wall.

Figure 2. Coronary angiography showing spontaneous dissection of the left anterior descending artery with diffuse narrowing of the vessel. (A) In a right anterior oblique cranial view, a type 2B dissection of the left anterior descending artery is observed. (B) Right anterior oblique caudal view, with no initial involvement of the left circumflex artery and its branches.

true lumen of the vessel, a percutaneous coronary intervention (PCI) was performed with the implantation of two drug-eluting stents.

The patient maintained severe precordial pain at the end of the procedure, and was immediately re-studied, and the results showed proximal retrograde dissection of LAD and the first marginal branch (Figure 3).

A new stent was implanted with an overlap in the proximal third of the LAD. In the angiographic control,

opacification of the distal LAD bed was not observed after the distal border of the last stent. We chose to maintain a conservative treatment, both of this vessel and of the dissection of the first marginal branch, due to the resolution of the precordialgia and the regression of the ST-segment elevation in the ECG (Figure 4).

The echocardiogram estimated a left ventricular ejection fraction of 68% using the Simpson method, with akinesia of the apical and inferoapical segments. The Doppler ul-

Figure 3. Control coronary angiography after initial angioplasty of the left anterior descending artery. In right anterior oblique caudal views (A) and left anterior oblique caudal views (B), a retrograde dissection of the left anterior descending artery and marginal branch is observed.

Figure 4. Coronary angiography showing the result of the procedure. (A) Right anterior oblique caudal view shows marginal branch dissection, maintained under conservative treatment. (B) In a right anterior oblique cranial projection, three stents are observed in the left anterior descending artery, with no opacification of the distal bed of this coronary vessel.

trasonography of the carotid and vertebral arteries showed no changes. During hospitalization, testing for connective tissue diseases and systemic inflammatory diseases was performed, with a negative result. The patient did not use hormone therapy.

She maintained hemodynamically stable, and was discharged after 16 days of hospitalization, asymptomatic and with optimized pharmacological therapy. She remains in outpatient follow-up at our organization.

DISCUSSION

Spontaneous coronary artery dissection is defined as a coronary artery dissection that is not associated with atherosclerosis, trauma or iatrogenesis. It is a relevant cause of acute coronary syndromes and sudden death in patients, especially in young women and patients with few conventional risk factors for atherosclerosis.^{3,7} Most studies on the topic are in the form of case reports or case series.^{1,7}

The actual prevalence of the disease is still uncertain due to underdiagnosis.^{3,7} These patients are at risk of receiving alternative diagnoses or being discharged after being evaluated in health services because they are relatively young and often without risk factors for atherosclerosis, which is not the expected phenotype for patients with acute MI.³ Initially, SCAD was known as a rare and mostly fatal disease in women in the puerperium. Currently, evidence shows that this disease is more common than previously imagined.³ The most affected population is young women, in which SCAD is a possible cause in up to 24% to 35% of MI cases in female patients aged up to 50 years.^{3,7} Men are affected less frequently (less than 10% to 15% of cases).¹

According to the prevalence reported in cohort studies, the most frequently associated risk factors are fibromuscular dysplasia (25% to 86%), use of hormone therapy (10.7% to 12.6%), multiparity (8.9% to 10%), pregnancy (2% to 8%), systemic inflammatory disease (<1% to 8.9%), and known arteriopathy or connective tissue disease (1.2% to 3%).^{3,4,5,7} Most patients referred a precipitating factor to the clinical manifestation, such as intense exercise, Valsalva maneuvers, emotional stress, labor, use of illicit drugs, coughing or vomiting.³

Ischemia is generated, in these cases, by the formation of an intramural hematoma with consequent luminal obstruction, by the rupture of the intima layer or formation of an intraluminal thrombus.^{1,3} The pathophysiology is not clear, and two theories have been described. The first one proposes that the initial event is a rupture in the inner layer of the vascular wall, allowing the true lumen blood to flow through the false lumen. The second one proposes that the primary event would be a spontaneous hemorrhage from the *vasa vasorum* that infiltrates the vascular wall.^{1,3}

Although there is a wide spectrum of clinical presentations and severity of SCAD, patients who survive and seek medical attention almost always present with acute coronary syndrome with elevated myocardial necrosis markers,³

as in the case reported. According to available case series, 16% to 87% of patients with SCAD present with ST-segment elevation MI, and in 13% to 69% of cases, with non-ST-segment elevation MI.³ The clinical presentation of these two types of MI in the same patient during a single hospitalization is very rare, but possible, as reported by Kalamadasa et al.⁸ and in the present case.

The first line for diagnosis is coronary angiography. Intracoronary imaging methods, including intravascular ultrasound and optical coherence tomography, provide detailed visualization of the arterial wall, which helps in the diagnosis of SCAD. However, these tools have additional risks and costs, and are not widely available.^{1,3,5} Saw classification is commonly adopted for cases of SCAD. In type 1, there is the classic appearance of multiple radiolucent lumens or retention of contrast in the arterial wall. Type 2 refers to the presence of diffuse stenosis, which can vary in terms of severity and length. This is the most common angiographic manifestation according to several studies, and it was also evident in our clinical case. Variant 2A reflects diffuse arterial narrowing, preceded and followed by normal segments, and variant 2B shows diffuse narrowing up to the distal end of the artery. In type 3 cases, there is usually focal or tubular stenosis.^{3,7}

Observational studies have indicated that, in control angiographies, angiographic resolution of SCAD was observed in most cases undergoing conservative treatment.^{3,5,7} However, the time limit for spontaneous resolution is uncertain, and is more frequent after one month of the acute event. Early complications from recurrent MI can occur in 5% to 10% of individuals in this type of therapy. Most of these patients require emergency revascularization, and there are no predictive clinical or angiographic factors for the worsening of the condition. Therefore, monitoring for an extended period is recommended in cases of conservative therapy. This is not recommended in cases of high-risk patients with persistent ischemia, left main coronary artery dissection or hemodynamic instability. In these cases, urgent intervention with PCI or surgical revascularization should be considered.^{3,7} There is no consensus about the duration of dual antiplatelet therapy in these cases.³

Evidence suggests that PCI is associated with an increased risk of complications and suboptimal outcomes, with a risk of iatrogenic dissections or extension of spontaneous dissections or intramural hematomas induced by the procedure.^{3,5,6} After PCI, dual antiplatelet therapy should be administered, according to the type of stent implanted.³

There are few publications on surgical myocardial revascularization. Bypass grafting is described as a treatment strategy for SCAD in patients with left main coronary artery lesions or proximal coronary artery disease, after PCI failure or complications, or when there is refractory ischemia despite conservative treatment.^{3,8}

Hence, conservative treatment is generally preferred over other strategies, in clinically stable patients with

no evidence of recurrent ischemia. This strategy is also appropriate in patients with occlusion of distal vessels or branches, who would not routinely be submitted to PCI.³ In the clinical case described, PCI was chosen due to the symptoms and an electrocardiographic evidence of severe ischemia. The complication that occurred after the procedure was predictable, as described, and a new intervention was performed only in the main artery, which had greater territory at risk, and conservative treatment of the branch which was secondarily affected was maintained.

Drug therapy with a statin is not routinely recommended in these cases, and should be applied according to indications for primary prevention of atherosclerotic disease. The use of beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin receptor blockers is not consensual, and it is indicated in patients with ventricular dysfunction or for management of hypertension. Complementary antianginal therapy, with nitrates, calcium channel blockers and ranolazine, can be considered according to the symptoms and tolerance of patients.³

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DECLARATION OF CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: JAF; data collection: SOB and RRC; data interpretation: JAF and GGMM;

text writing: SOB and RRC; approval of the final version to be published: EGMJ and TON.

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