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Cardiorespiratory arrest after acute myocardial infarction due to coronary vasospasm. Case report and literature review

Parada cardiorrespiratória após infarto agudo do miocárdio por vasoespasmo coronariano. Relato de caso e revisão da literatura

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ABSTRACT - Vasospastic angina is an uncommon cause of cardiac arrest and ventricular arrhythmias. However, survivors of these complications are at an increased risk of recurrence, despite normal ventricular function and optimized medical therapy. We describe a case of a 50-year-old former smoker who developed cardiorespiratory arrest secondary to severe coronary vasospasm.

Keywords: Angina pectoris; Heart arrest; Arrhythmias, cardiac; Coronary vasospasm

RESUMO - A angina vasoespástica é uma causa incomum de parada cardíaca e arritmias ventriculares. No entanto, os sobreviventes dessas complicações têm um risco aumentado de recorrência, apesar da função ventricular normal e do tratamento médico otimizado. Descrevemos o caso de uma ex-tabagista de 50 anos que teve parada cardiorrespiratória secundária a vasoespasmo coronariano grave.

Descritores: Angina pectoris; Parada cardíaca; Arritmias cardíacas; Vasoespasmo coronário

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INTRODUCTION

Vasospastic angina, or Prinzmetal´s variant of angina, is a syndrome characterized by chest pain. It occurs at rest and usually relieves with administration of nitrates.[1] Coronary spasms play an important role in this pathophysiology, resulting in obstruction of the artery.[2]

Transient myocardial ischemia causes angina in many patients, and myocardial infarction may develop in cases of persistent spasm.[1] Despite optimal medical therapy with calcium channel blockers (CCB) and nitrates, symptoms may persist in 5 to 30% of patients.[3,4]

CASE REPORT

Patient M.L.C., 50-year-old, female, born in São Paulo (SP, Brazil), homemaker and former smoker (quitted 2 years ago; 30 pack-years). She was admitted to the emergency department of Hospital São Paulo due to cardiorespiratory arrest (CA), with a description of previous typical chest pain, sweating and malaise. Out-of-hospital cardiopulmonary resuscitation (CPR) was performed initially by family members for 20 minutes, and later by the emergency medical service team for another 20 minutes until arrival at the hospital, with a report of defibrillation applied by an automatic external defibrillator.

She was admitted after 40 minutes of CPR, intubated, and ventilated with a bag-valve-mask device. After two more cycles of CPR and administration of 1mEq/kg

intravenous (IV) bicarbonate, return of spontaneous circulation (ROSC) was observed. Focused cardiac ultrasound demonstrated satisfactory myocardial contractility, with no evident signs of segmental dysfunction. The patient developed hemodynamic instability and required increasing doses of vasopressors (noradrenaline).

An electrocardiogram (ECG) was performed and revealed T-wave inversion of V4-V6, D1, and aVL (Figure 1). A mild ST-segment depression was also observed. A few minutes later, the patient presented with another cardiac arrest with a non-shockable rhythm and ROSC after three cycles of CPR. Another round of ECG was performed, showing ST-segment elevation in aVR and diffuse ST-segment depression (Figure 2). Additionally, an elevation of cardiac biomarkers was observed; troponin level upon admission was 52pg/mL and rose to 833pg/mL (reference value: up to 14pg/mL).

After hemodynamic stabilization, the patient was referred to an emergency coronary cineangiography that revealed no significant obstructive lesions. The left anterior descending artery (LAD) had 30% stenosis, left circumflex artery (LCX) had 30% stenosis in the middle third, and right coronary artery (RCA) had no stenosis (Figure 3). Left ventriculography revealed basal hyperkinesia with hypokinesia in other segments, in addition to moderate systolic dysfunction (Figure 4). The suggested diagnosis was CA secondary to coronary vasospasm. The patient was transferred to the intensive care unit (ICU), where drug therapy with CCB and high-potency statins was maintained, and eventually had ventricular dysfunction, therefore requiring dobutamine. After 5 days, brain death was diagnosed.

It was revealed by family members that in a previous hospitalization 2 years ago, the patient underwent coronary cineangiography, while still a smoker, due to an obstructive lesion in the left main coronary artery (LMCA) of 70%, which was reduced to 30% after nitroglycerin administration, with a final diagnosis of coronary vasospasm (Figure 5). Regular clinical treatment was initiated with diltiazem 180mg per day rosuvastatin 10mg per day, acetylsalicylic acid (ASA) 100mg per day, and isosorbide mononitrate 60mg per day.

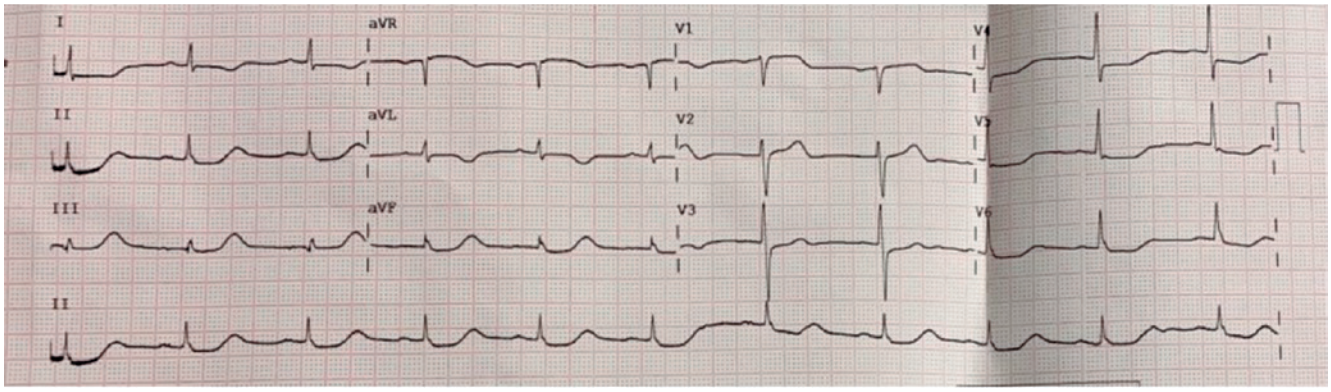


Figure 1. Electrocardiogram upon admission showing T-wave inversion of V4-V6, D1 and aVL, and mild ST-segment depression of descending pattern.

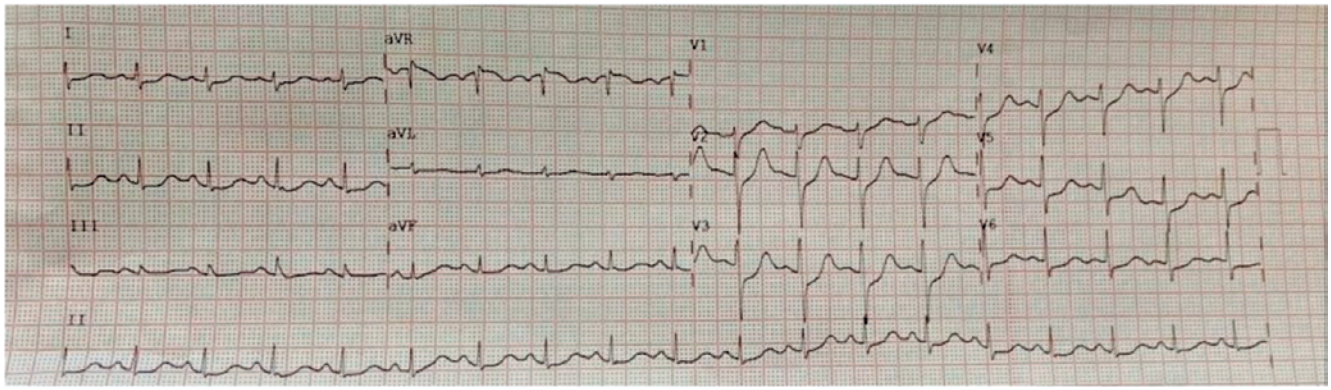


Figure 2. Electrocardiogram performed after the second cardiac arrest demonstrating ST-segment elevation in aVR, and diffuse ST-segment depression.

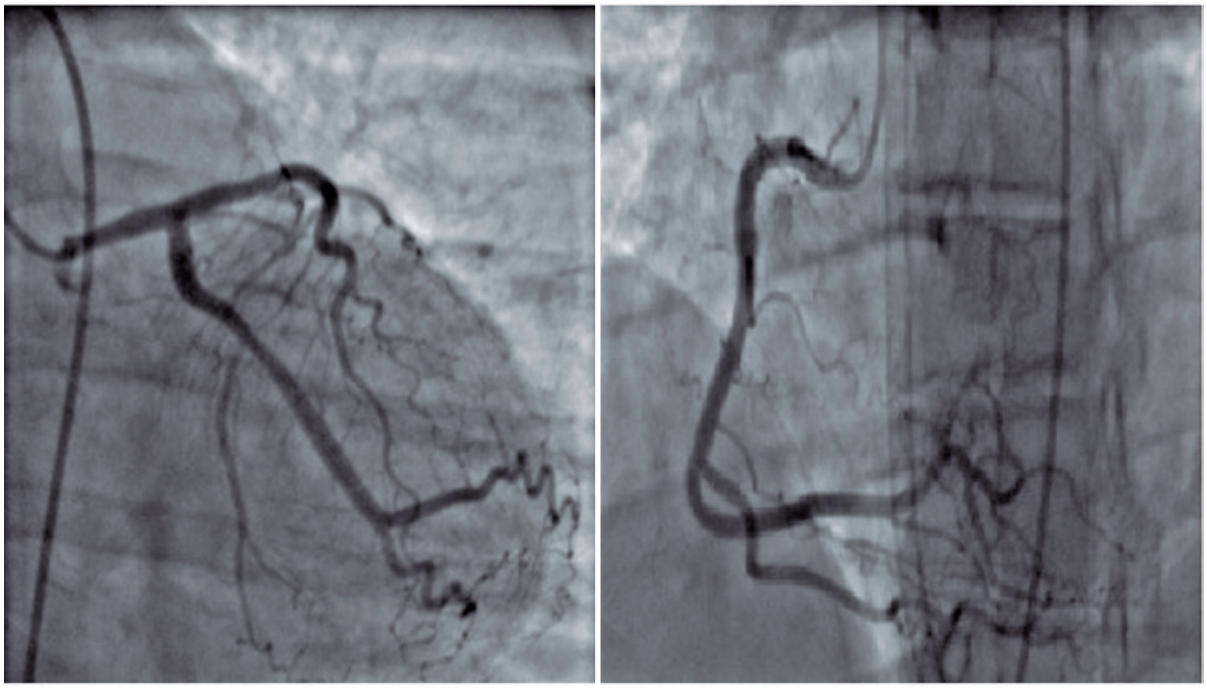


Figure 3. Coronary angiography showed left anterior descending artery with 30% ostial stenosis, and left circumflex artery with 30% middle third stenosis.

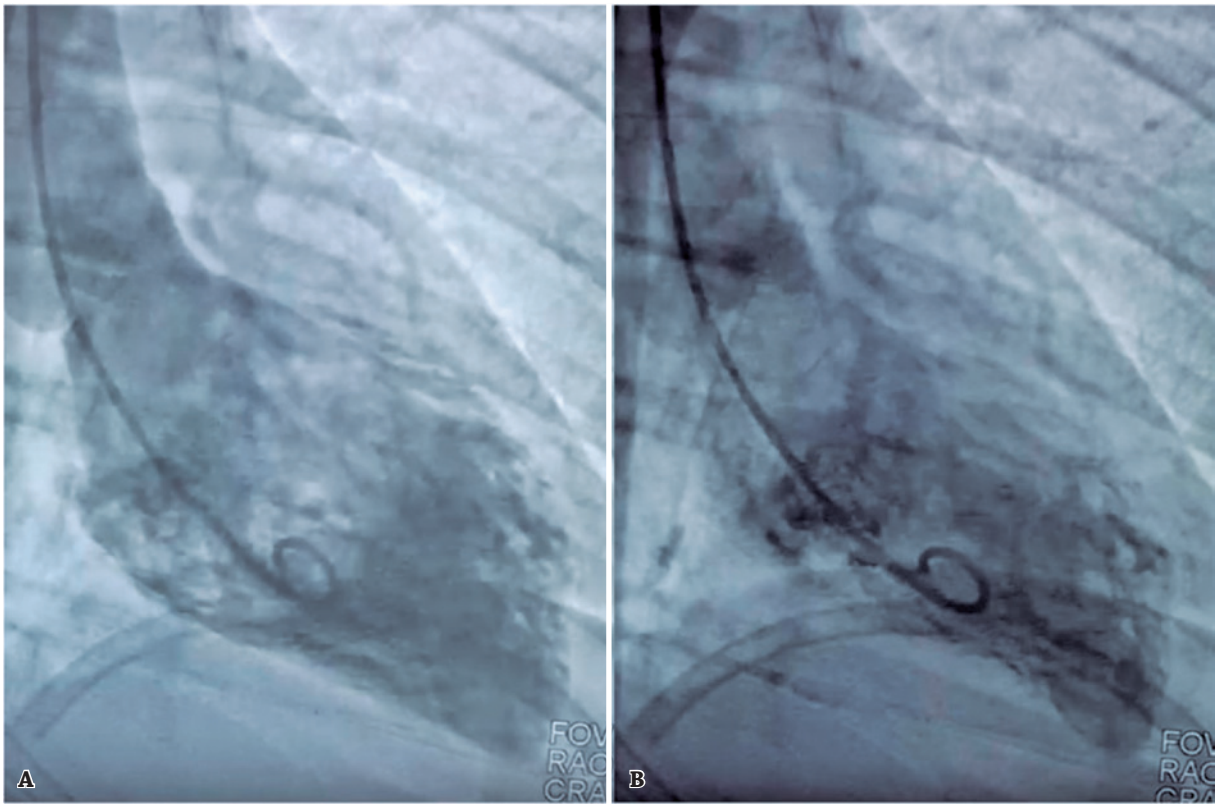
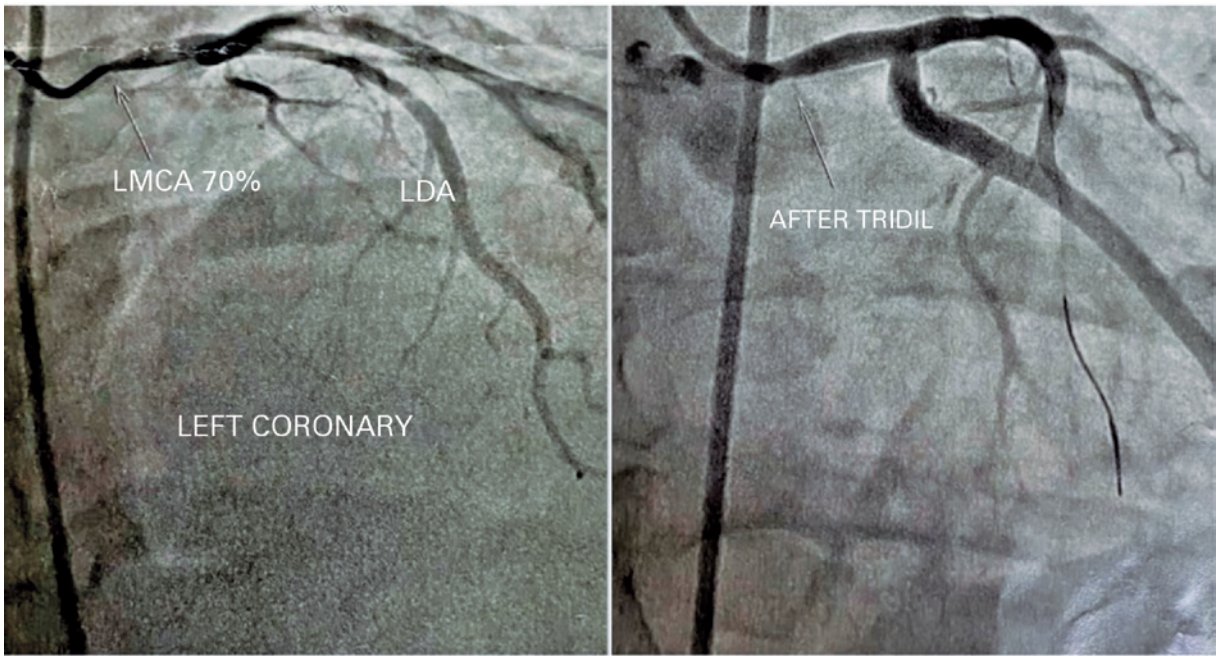


Figure 4. Left ventriculography revealed basal hyperkinesia with hypokinesia in other segments, in addition to moderate systolic dysfunction. (A) Cardiac diastole. (B) Cardiac systole.



LMCA: left main coronary artery; LDA: left anterior descending artery.
Figure 5. Coronary angiography performed 2 years ago with evidence of obstructive lesion in the left main coronary artery of 70%, which was reduced to 30% after nitroglycerin administration.

DISCUSSION

Vasospastic angina is an uncommon form of CA and ventricular arrhythmia, which is diagnosed due to recurrent episodes of chest pain, usually occurring at rest. It is also characterized by transient ST-segment elevation or depression on ECG obtained at the time of pain, and subsequently, the absence of high-grade coronary stenosis on arteriography.[1,2]

Coronary vasospasm may occur in the absence of any previous increase in myocardial oxygen demand (e.g., exercise) and in vessels with or without angiographically detectable lesions. The original descriptions reported anatomically focal spasm sites, but diffuse spasms have been described and can occur in angiographically normal coronary vessels, although they are more common at sites of atherosclerotic plaques.[5] The episodes occur most frequently from midnight to early morning, when the systemic vagal tone is the highest.[6]

The precipitating factors of vasospasm include smoking, magnesium deficiency, physical or emotional stress, the use of sympathomimetic agents, and hyperventilation. In addition to being a former smoker, the patient in this case had also been exposed to emotional stress the night before the event.[7]

Recently, it was demonstrated that patients without obstructive coronary artery disease have a good long-term prognosis. In a study of patients with vasospastic angina and single-vessel disease or stenosis of ≤70% in any major coronary artery, 1-and 5-year survival rates of 99% and 95%, respectively, were observed. In contrast, patients with multi-arterial disease had 1-and 5-year survival rates of 87 and 77%, respectively.[8]

Treatment includes smoking cessation and pharmacological therapy. Calcium channel blockers can be initiated, such as diltiazem at 240 to 360mg per day. The use of sublingual nitroglycerin has also been suggested to abort angina episodes, decreasing the incidence of myocardial infarction and life-threatening arrhythmia associated with these episodes. Non-selective beta-blockers should be avoided because they increase the risk of vasospasm.[9]

There is a lack of data regarding the best approach for aborted cardiac arrest. Studies suggest the placement of an implantable cardioverter-defibrillator, initiation of therapy with CCB and statins, and avoidance of percutaneous coronary intervention due to the increased possibility of diffuse spasm during and after the procedure.[10]

Differential diagnosis includes stress cardiomyopathy (also called apical ballooning syndrome, Takotsubo cardiomyopathy or "broken heart" syndrome), a syndrome characterized by transient regional systolic dysfunction, mainly of the left ventricle, in the absence of angiographic evidence of obstructive coronary artery disease or acute plaque rupture. It is more common in women than men and occurs predominantly in older adults exposed to physical or emotional stress.[11,12]

Vasospastic angina remains an underdiagnosed condition. This illness should be considered in cases of angina presentations and electrocardiographic changes that are

not attributable to obstructive coronary artery disease or unstable plaque rupture. Survivors of these complications are at an increased risk of recurrence despite normal cardiac function and optimal medical therapy. However, since this is a rule-out diagnosis, other possible causes should be investigated and treatment should be individualized.

SOURCE OF FINANCING

None.

CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: MCRDB, CACB and GCO; data collection: MCRDB, CACB and GCO; data interpretation: MCRDB, CACB and GCO; text writing: MCRDB, CACB and GCO; approval of the final version to be published: ANA, AFTG and FJNM.

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