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Coronary-artery spasm after coronary artery bypass graft surgery without extracorporeal circulation. Diagnostic and management

Espasmo coronariano no pós-operatório de cirurgia de revacularização do miocárdio sem circulação extracorpórea. Diagnóstico e manejo

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Resumo

O espasmo de artérias coronárias no perioperatório de cirurgia de revascularização do miocárdio é uma complicação grave, com elevada mortalidade. Paciente de 51 anos submetido à cirurgia de revascularização do miocárdio sem circulação extracorpórea. Apresentou no 1°dia de pós-operatório (PO) alteração enzimática e supradesnivelamento do segmento ST, evoluindo, em seguida, em fibrilação ventricular, com reanimação cardiopulmonar com sucesso. Cateterismo cardíaco demonstrou espasmo importante de todas as artérias coronárias e da anastomose entre artéria torácica interna esquerda com artéria interventricular anterior. Utilizados vasodilatadores intracoronarianos e intra-enxerto, com restabelecimento de seus calibres usuais, imediata melhora clínica e estabilidade hemodinâmica. Com evolução satisfatória, o paciente recebeu alta hospitalar no 13º PO.

Descritores: **Revascularização miocárdica. Ponte de artéria coronária. Vasoespasmo coronário. Cateterismo cardíaco.**

Abstract

Coronary artery spasm in perioperative of coronary artery bypass graft surgery is a serious complication, with high rate mortality. Patient 51 years-old submitted to coronary artery bypass graft surgery without Extracorporeal Circulation. The patient evolved in 1st post operative (PO) day with enzymatic alteration and ST-elevation, developing soon afterwards in ventricular fibrillation, defibrillation with success. Cardiac catheterization showed important spasm of all coronary arteries and anastomosis between the left internal thoracic artery and the left anterior interventricular artery. Intracoronary Vasodilators and intra-graft, with re-establishment of their usual and immediate calibers to improve clinic and Hemodynamic stability was used. Satisfactory evolution, discharged at 13rd PO day.

Descriptors: **Myocardial revascularization. Coronary artery bypass. Coronary vasospasm. Heart catheterization.**

INTRODUCTION

The spasm of coronary arteries (CAS) in the intraoperative and postoperative (PO) for coronary artery bypass grafting surgery is a rare complication with an incidence of 0.8% to 1.3%, which may bring catastrophic consequences, with high mortality [1-3]. It presents itself as an important cause of myocardial ischemia, of multifactorial aspect, with sudden appearance and it may predispose to serious cardiac arrhythmia, cardiogenic shock and death during surgery for coronary artery bypass grafting [4-6].

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The authors present the case of a patient undergoing coronary artery bypass grafting without cardiopulmonary bypass (CPB), which developed severe CAS diagnosed by cardiac catheterization after episode of ventricular fibrillation (VF) and cardiogenic shock.

This patient agreed to this publication, signing a consent term. This study was submitted to the Research Ethics Committee at the Hospital Real e Benemérita Sociedade Portuguesa de Beneficência and it was approved under protocol 404-08.

CASE REPORT

Patient 51 years, male, untreated hypertensive patient and former smoker 3 years earlier (smoked 20 cigarettes per day for 35 years) hospitalized for a month due to unstable angina and discharged with anti-ischemic therapy and programming for coronary angiography.

Cardiac catheterization revealed 80% obstruction of proximal left anterior interventricular artery (AIA) and other arteries free of lesions (Figure 1). Soon after the procedure, he developed a new episode of chest pain, and then he was referred to the Intensive Care Unit, where he received therapy for unstable angina and scheduled surgery for myocardial revascularization. Electrocardiogram showed extensive anterior ischemia (Figure 2) and values for troponin and CK-MB <0.01 and 12, respectively.

The patient then underwent coronary artery bypass grafting with distal anastomosis of left internal thoracic artery (LITA) for AIA without CPB. The intraoperative and

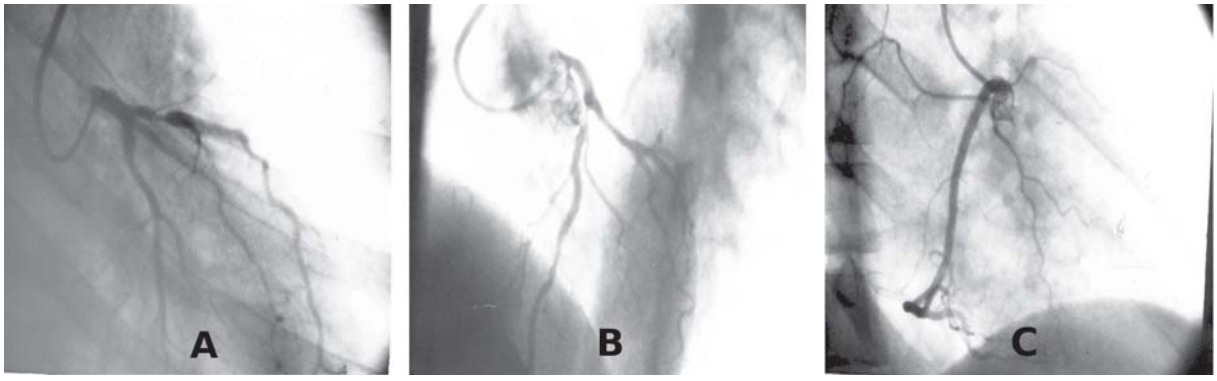

Fig. 1 - Preoperative coronary angiography showing a severe lesion in1/3 of the proximal of the AIA and CX without obstructive lesions in RAO (A) and LAO cranial (B). RC free of lesions (C)

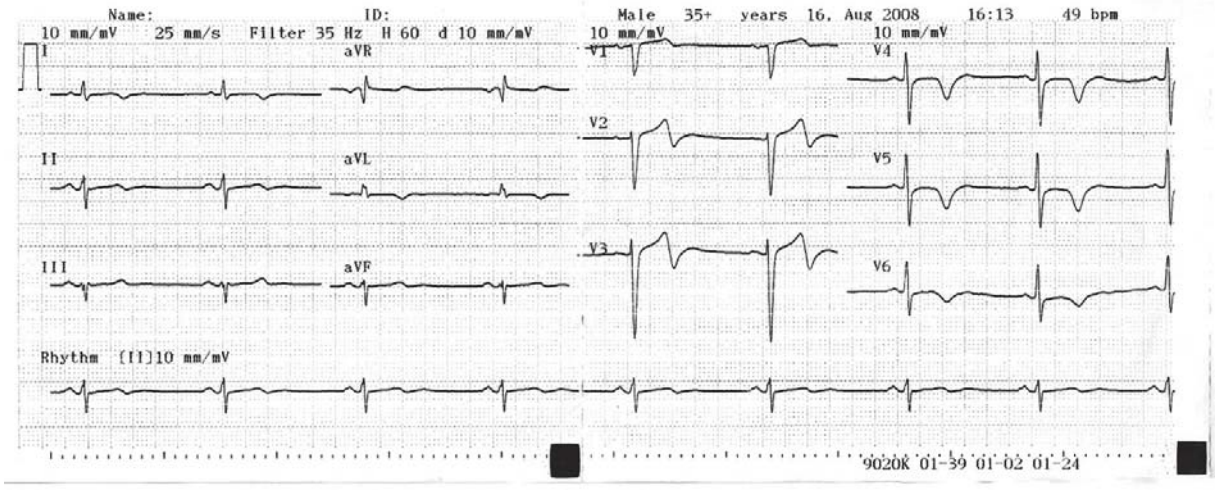

Fig. 2 - Pre-operative electrocardiogram: an extensive anterior ischemia

immediate postoperative (IPO) were uneventful. Values of troponin and CK-MB 0.067 (normal: 0.010 ng/ml) and 22 (normal: 25 U/L), respectively.

On the 1st postoperative day, the patient presented EKG with abnormal nonspecific repolarization in the anterior wall, without clinical signs and hemodynamic stability. Evolutionarily, onset of pain, sharp in the left hemithorax and in the insertion site of the pleural drain; being prescribed painkillers, performed electrocardiogram, which showed ST-segment elevation of 1 mm in leads V2-V5 (Figure 3), and dosed troponin (0.099) and CK-MB (51).

The patient had a cardiopulmonary arrest (CPA) in VF. He presented exams gasometry, sodium, potassium and magnesium within normal limits. Performed cardiopulmonary resuscitation (CPR) with tracheal intubation and defibrillation with two 360J shocks with reversion to sinus rhythm, and amiodarone 300 mg, however, the patient presented hemodynamic instability, requiring use of norepinephrine and dobutamine. Referred to the hemodynamic service for emergency cardiac catheterization. During the examination, presented a new CPA in VF, being performed a new defibrillation (360J), again reverting to sinus rhythm. Evidenced important spasm of all coronary arteries and anastomosis between LITA with AIA (Figure 4). Applied intracoronary vasodilators and intra-graft (isosorbide mononitrate-5 20 mg and nitroglycerin 200 mg), with restoration of normal calibers of the coronary arteries and LITA (Figure 5) followed by immediate clinical and hemodynamic stability without the need to use intra-aortic balloon. An echocardiogram was

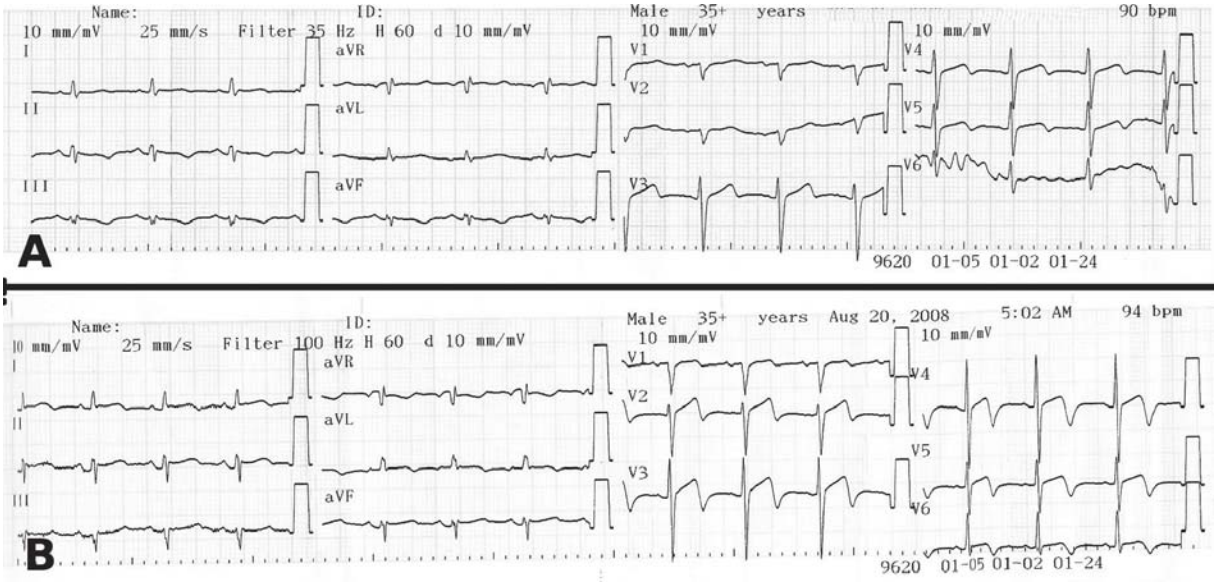

Fig. 3 - Electrocardiogram in the IPO (A). Change of nonspecific repolarization on the anterior wall. Electrocardiogram on the first postoperative day (B). ST-segment elevation of 1 mm in leads V2 - V5

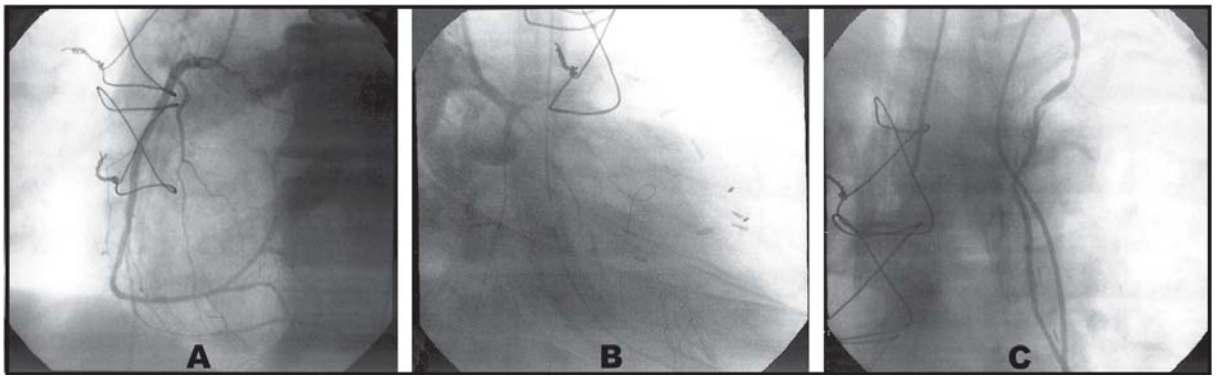

Fig. 4 - Angiographic demonstration of the right coronary artery and spasm of the entire length of the left coronary artery and its branches (A) and left internal mammary artery and anastomosis between LITA and AIA (B)

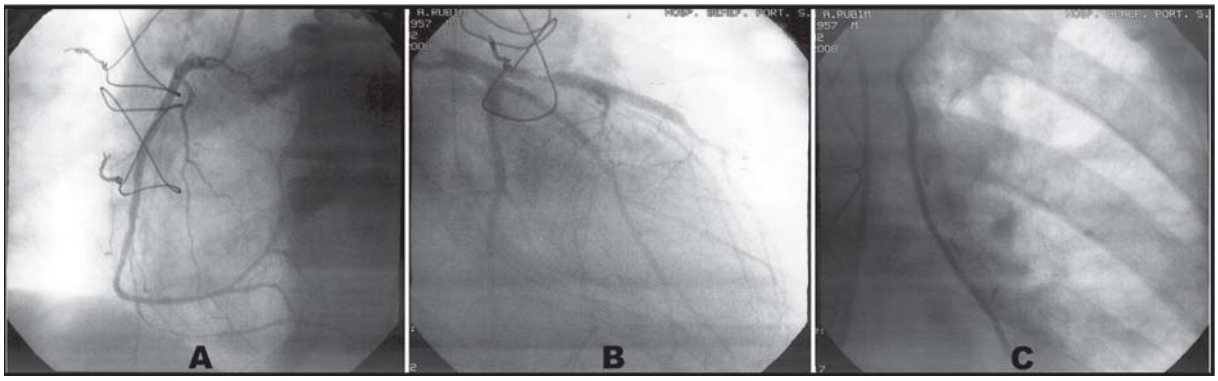

Fig. 5 – Angiography of the right coronary artery (A), left coronary artery (B) and anastomosis between LITA and AIA (C) with restoration of normal calibers of the coronary arteries and their branches after vasodilator

then performed and demonstrated diffuse involvement of the left ventricle (LV) of a significant degree, with an ejection fraction of 39%. The patient was discharged from the intensive care unit on the 7th postoperative day and he was discharged from hospital with a satisfactory outcome, with echocardiogram with an ejection fraction of 79% at the 13th postoperative day.

DISCUSSION

The mechanism of CAS was recognized long ago. In 1959, Prinzmetal described for the first time a reversible type of angina caused by alteration of coronary vagal tone [7]. In 1972, Bentivoglio reported myocardial infarction caused by CAS in 22 patients, however, with no angiographic confirmation [7]. In 1977, Oliva et al. angiographically confirmed the CAS [7].

Koshiba et al. [8] studied the clinical and surgical profile of 115 cases of CAS related to cardiac surgery. The mean patient age was 64 years, with 94% of the cases over 50 years, being more prevalent among males (84%). Most events were documented on the intraoperative (76%), and 11% in the IPO.

There are several factors that can predispose to CAS in the intra-and postoperative period of cardiac surgeries, as the presence of coronary atheromatosis, but there are reports in the literature of CAS in apparently normal coronary arteries [9]. Other factors related to the etiology of coronary spasm of peri-operative myocardial revascularization are inadequate anesthesia, manipulation of the coronary arteries during surgery, elevation of endogenous catecholamines, administration of exogenous catecholamines, hyperventilation, alkalosis, low body temperature, release of vasoconstrictive factors as thromboxane A2 from platelets, autonomic stimulation and hypomagnesaemia [7,9]. This patient was uneventful in the IPO and had, at the time of onset of signs and ischemic symptoms caused by coronary spasm, normal biochemistry, body temperature and physiological oxygen saturation, with no hydroelectrolytic disturbances.

During the spasm and following reperfusion complications may occur as a potentially fatal arrhythmia, either slow (as atrioventricular blocks) or tachyarrhythmias such as ventricular tachycardia or fibrillation [9]. These last two are the most likely causes of death in these patients, which is a late manifestation of myocardial ischemia [10]. CAS is also an important cause of myocardial ischemia, which may be associated with cardiogenic shock and death in the postoperative of myocardial revascularization [5].

The early angiographic diagnosis must be made in the course of CAS refractory to drug therapy in order to obtain the exact diagnosis and drug treatment [7]. The small number of cases reported in the literature makes difficult the recognition of the factors that may lead to this complication, and the institution of specific treatment [8]. Suspected CAS occurs when, after CABG, there is sudden clinical instability, such as chest pain, electrical instability, the ST segment changes or arrhythmias and myocardial necrosis markers. It is of great value to highlight the characteristic of sudden onset of the angina, ST segment elevation and the change of segmental motion as peculiarities of the phenomenon of spasm.

Given the suspicion of CAS, the patient should be referred immediately to cardiac catheterization, which is the gold standard for diagnosis [2,5]. In case of confirmation of CAS, treatment should be promptly initiated. This treatment consists of intracoronary injection of vasodilators such as nitroglycerin or calcium channel blockers [5]. However, some cases do not always respond to these drugs, requiring other vasoactive drugs to stabilize and even temporary mechanical support, such as intra-aortic balloon.

The spasm of coronary arteries is multifactorial, sudden, and largely improved with the administration of intravenous nitrates and calcium channel blockers. It shall be part of the differential diagnosis as a cause of MI and low output syndrome in patients submitted to heart surgery.

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