SPONTANEOUS DISSECTION OF THE RIGHT CORONARY ARTERY IN A PATIENT WITH ACUTE CORONARY SYNDROME

DISSECÇÃO ESPONTÂNEA DA ARTÉRIA CORONÁRIA DIREITA EM PACIENTE COM SÍNDROME CORONARIANA AGUDA

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ABSTRACT

This study describes a case of acute coronary syndrome with ST-segment elevation and spontaneous dissection of the right coronary artery, diagnosed by coronary angiography in a male patient who underwent angioplasty and stent implantation three years ago.

Keywords: Coronary Artery Disease; Percutaneous Coronary Intervention; Myocardial Infarction; Spontaneous Coronary Artery Dissection; Stents.

RESUMO

Apresentamos um caso de síndrome coronariana aguda com supra desnivelamento do segmento ST (SCACSST) e dissecção espontânea da artéria coronária direita, evidenciada através da cineangiocoronariografia, em paciente do sexo masculino, previamente submetido à angioplastia primária com implante de stent há 03 anos.

Palavras-chave: Doença da Artéria Coronária; Intervenção Coronária Percutânea; Infarto do Miocárdio; Dissecção espontânea da artéria coronária; stents.

INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is a rare type of acute myocardial ischemia, usually reported as isolated cases in the literature[1,2]. This underdiagnosed ischemia presents a varied clinical presentation that may lead to acute coronary syndromes (ACS) and sudden death3,4.

The etiology of SCAD is unknown; however, it is usually observed in arteries free of atheromatous obstruction2 and after the invasive approach of ACS5. The presence of atheromatosis in the arteries does not exclude SCAD since endothelial dysfunction may be the pathophysiological basis for the onset of dissection2.

This study aimed to report the following case due to its peculiarity, specifically the occurrence of SCAD in a patient previously affected by ACS with ST-segment elevation who underwent stent implantation three years ago.

CASE REPORT

A 50-year-old male patient, hypertensive, diabetic, ex-smoker (two packs/day for 20 years) with a previous history of ACS and implantation of a non-pharmacological stent in the proximal and mid one-third of the right coronary artery (three years ago). He sought medical attention due to tightness and precordial pain. After 24 hours of admission, the patient presented a clinical worsening with strong intensity pain with irradiation to the back associated with vomiting, sweating, and palpitations in the last three hours. He had reported progressive angina during physical efforts and regular use of bisoprolol, simvastatin, and acetylsalicylic acid a few months ago.

The electrocardiogram on admission revealed sinus rhythm with isolated ventricular extrasystoles, a QS pattern in the lower leads, and no signs of acute ischemia. He was admitted to the emergency room and medicated with acetylsalicylic acid, morphine,

and isosorbide dinitrate. The first dosage of biochemical markers of myocardial necrosis was normal (troponin 0.007 ng/mL, normal < 0.014 ng/mL).

After ten hours, the patient reported another episode of chest pain associated with dynamic alteration of the ECG and ST-segment elevation in lower leads. Continuous infusion of intravenous nitroglycerin was started, and the patient was taken to the Hemodynamics laboratory for urgent coronary angiography. The examination conducted through the right femoral artery revealed diffuse proliferative restenosis in the proximal and mid-third of the right coronary artery and a spontaneous spiral dissection in the transition from the mid-third to the distal, immediately after the distal edge of the stent (Figure 1). The other coronary arteries and their respective branches did not present significant obstructive lesions. First, we chose percutaneous coronary intervention with pre-dilation of all segments affected by restenosis, followed by the implantation of two zotarolimus-eluting stents (3.5 x 38 mm and 3.0 x 24 mm) with a small overlap between them. Control laminography revealed a good final result, with no residual lesion, no dissection image, and maintenance of the distal thrombolysis in myocardial infarction III flow (Figure 2).

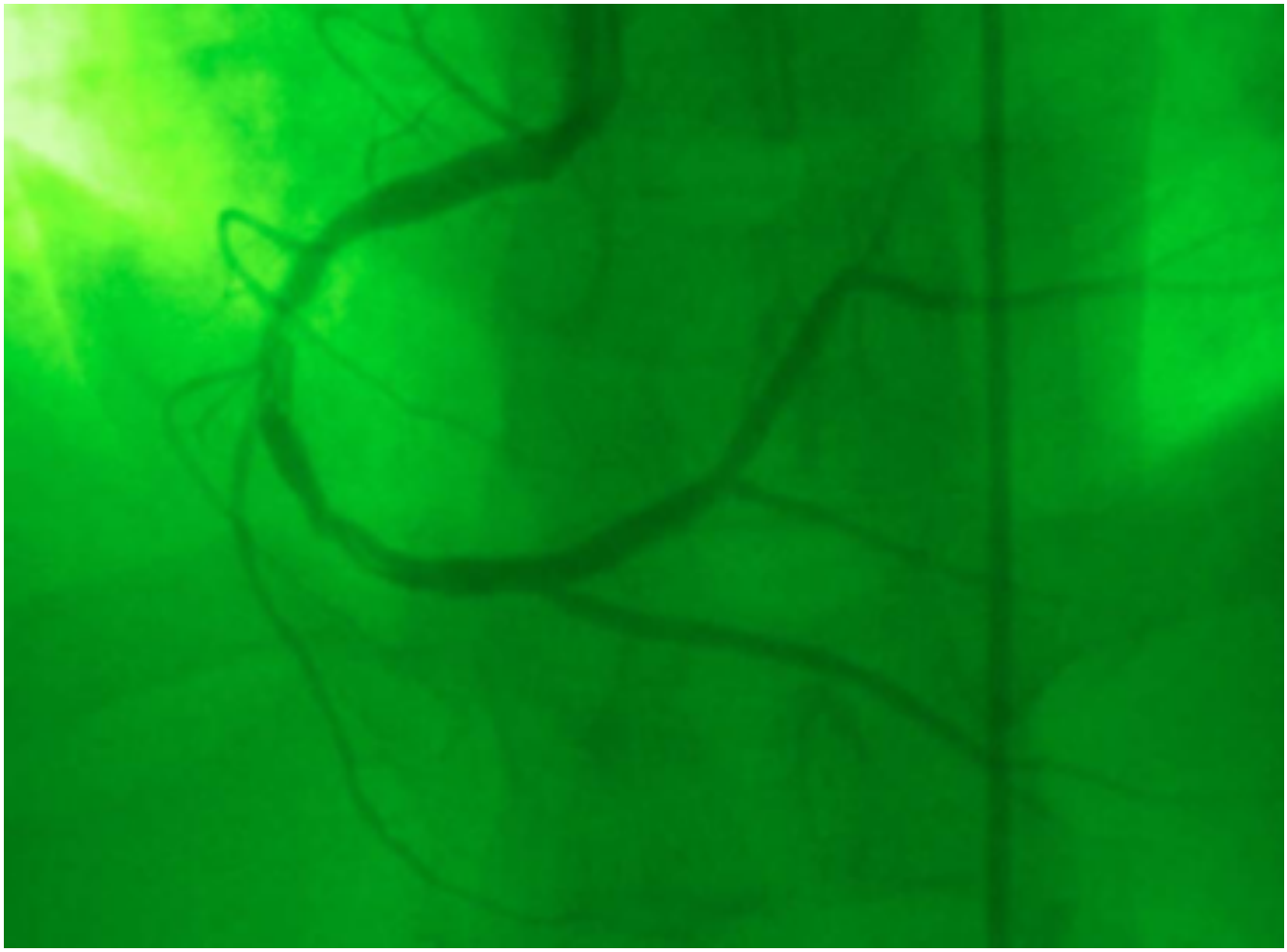

Figure 1. Right coronary artery in posteroanterior cranial showing the line of dissection starting at the level of the stent previously implanted in the one-third proximal artery.

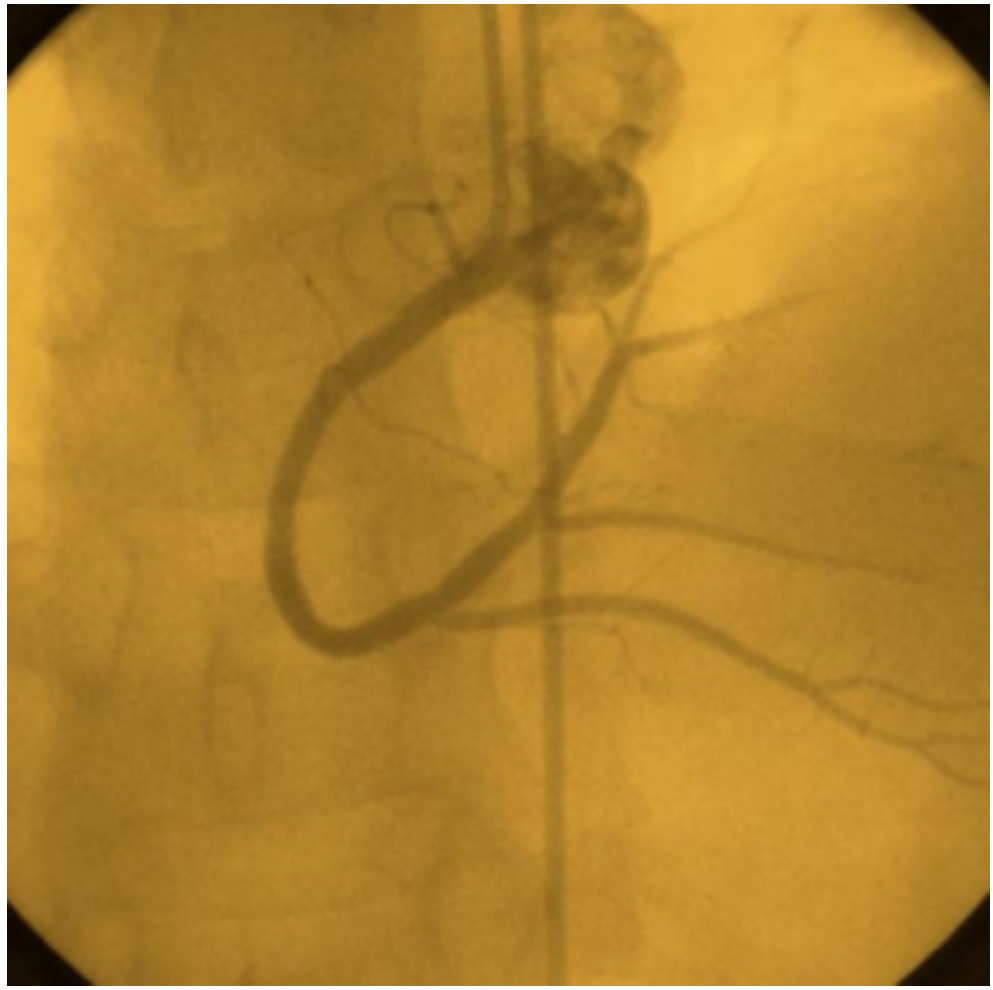

Figure 2. Right coronary artery in posteroanterior cranial after the implant of Zotalimus-eluting stents in one-third of proximal, mid, and distal arteries.

After angioplasty, the patient progressed satisfactorily without complications and was discharged on the fifth postoperative day with prescriptions for enalapril, bisoprolol, acetylsalicylic acid, clopidogrel, and atorvastatin for use at home. Two months later, the patient returned for outpatient follow-up, and he was asymptomatic and clinically stable. Maintenance of dual antiplatelet therapy was indicated for at least one year (from the date of the infarction), and periodic clinical reevaluations were recommended.

COMMENTS

The first report of SCAD was made by Pretty in 1931 after performing a necropsy on a 42-year-old woman who evolved to sudden death after presenting chest pain6. Since then, sporadic case reports have been published.

SCAD predominantly affects young female patients under 50 years old, using contraceptive drugs or around the time of delivery, and without a history of cardiovascular risk factors7. However, this disease may be associated with underlying coronary atherosclerosis, connective tissue diseases, vasculitis, cocaine abuse, and thoracic trauma[1]. SCAD rarely affects males, and about half of the cases among them are preceded by intense physical exertion, similar to the case described in this study. Although the cause is unknown, the literature indicates that its incidence is low, ranging from 0.1% among patients with stable anginal symptoms to 4.0% of all cases of ACS[8].

Two pathophysiological mechanisms may be related to SCAD. The first is attributed to increased shear stress in the endothelial wall, and the second is related to hemorrhage in the middle layer, which may arise from tissue weakening of the arterial wall associated with inflammation, abnormal collagen synthesis, or rupture of the vasa vasorum[9]. In both cases, the coronary artery lumen reduces, resulting in myocardial ischemia[1].

The ACS spectra can be described as a clinical presentation of SCAD, including sudden death. Autopsy is usually the diagnosis of SCAD since angio-

graphic documentation is limited. However, in sporadic cases, such as the present study, SCAD may be diagnosed by coronary angiography. The few reports of SCAD diagnosed by this type of intervention show that the anterior descending coronary artery is the most affected vessel (75%), followed by the right coronary artery (20%), the circumflex artery and its branches (4%), and the left main coronary artery in rare cases (< 1%)[8].

Interventions for SCAD range from clinical treatment to myocardial revascularization surgery or stent implantation. Some criteria, such as the degree of clinical severity, hemodynamic status, and topography of the dissection, should be considered when establishing a treatment[10].

Although percutaneous coronary intervention for SCAD is associated with a high rate of technical failures, in this particular case, the medical team chose this procedure due to an ACS with ST-segment elevation, restenosis of the stent, and alteration of the distal flow through the coronary artery. The percutaneous coronary intervention was promptly performed with the implantation of Zotarolimus-eluting stents, achieving immediate success without intercurrences.

We report a rare and successful case of SCAD diagnosed by coronary angiography in a male patient. Other imaging techniques, such as intracoronary ultrasonography and optical coherence tomography, when available, coupled with early coronary angiography in ACS, may help the therapeutic and prognostic decisions on SCAD[11].

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