Coronary Artery Dissection Following Pharmacologic Stress Echocardiography and the Follow-Up of Clinical Management: A Case Report

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Introduction

Spontaneous coronary artery dissection (SCAD) remains a diagnostic and therapeutic challenge in the evaluation of chest pain. Misinterpretation of symptom characteristics and failure to consider the patient's epidemiological profile may lead to inappropriate diagnostic workups and treatment strategies. The case described herein represents only the second report with similar characteristics published in major medical databases.[1] This case underscores the importance of considering uncommon causes of chest pain and acute coronary syndrome (ACS), while also providing a brief review of relevant literature. The report was prepared in accordance with the CAse REport (CARE) guidelines.[2]

Case report

The patient is a 42-year-old white female from São Paulo, Brazil. She is divorced, has completed high school, works as an administrative secretary, and identifies as Catholic. Her weight was 72 kg and height 1.75 m at presentation.

Primary symptoms

The patient reported the onset of chest pain, initially suggestive of noncardiac origin, in January 2024. The pain was described as mild, located in the thoracic region, and not associated with physical exertion. The symptoms persisted until March 2024, prompting her to seek evaluation by a private cardiologist. During the initial consultation, the clinical impression was that the patient likely had stable angina. The following medications were prescribed: acetylsalicylic acid at 100 mg/day, simvastatin at 20 mg/day, metoprolol at 50 mg/day, and enalapril at 20 mg/day. Previously, the patient was only taking desogestrel at 75 mg/day as an oral contraceptive. A dobutamine stress echocardiography was requested for further functional assessment.

Keywords

Blood Vessel Dissection; Coronary Disease; Stress Echocardiography; Case Reports

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Medical, family, and psychosocial history

The patient had a known history of sporadic migraine and iron-deficiency anemia. She denied smoking, alcohol consumption, and maintained a sedentary lifestyle. Her surgical history included cesarean section, saphenectomy for venous insufficiency, and partial thyroidectomy for a benign thyroid nodule. She reported four pregnancies, including three cesarean deliveries and one spontaneous abortion of unknown etiology. There was no significant family history of cardiovascular or rheumatologic diseases.

Relevant past interventions

On March 20, 2024, the patient underwent a dobutamine stress echocardiography at a private hospital. During the examination, she experienced a sudden onset of severe precordial pain, distinct from her previous symptoms, described as a crushing sensation rated 10/10 in intensity, radiating to the head, and accompanied by nausea and vomiting. The procedure was immediately interrupted.

She was referred to a public emergency department, where she was admitted with a working diagnosis of non-ST-segment elevation ACS (NSTE-ACS). Electrocardiogram findings were nonischemic; however, serial troponin measurements showed a positive curve. Diagnostic coronary angiography revealed a tortuous and elongated left anterior descending artery, which also supplied the inferior wall, with an image consistent with a type 1 coronary dissection (Figure 1) in the mid to distal segment. Transthoracic echocardiography showed a preserved left ventricular ejection fraction (47%), with hypokinesia of the basal segment of the inferior wall and the apical region. She was discharged on her initial medications, with the addition of clopidogrel at 75 mg/day and sublingual isosorbide dinitrate at 5 mg as needed for chest pain. She was referred to our tertiary public cardiology center for outpatient follow-up.

Physical examination and clinical findings at the tertiary cardiology center

The patient was first evaluated at our tertiary cardiology center on May 10, 2024. She reported persistent angina classified as Canadian Cardiovascular Society (CCS) class II, with characteristics distinct from the initial atypical pain in January 2024, but similar to the pain experienced during the ACS event. Physical examination was unremarkable, and both electrocardiogram and chest X-ray performed at our center were within normal limits.

Following clinical assessment, myocardial perfusion scintigraphy at rest and under pharmacologic stress using

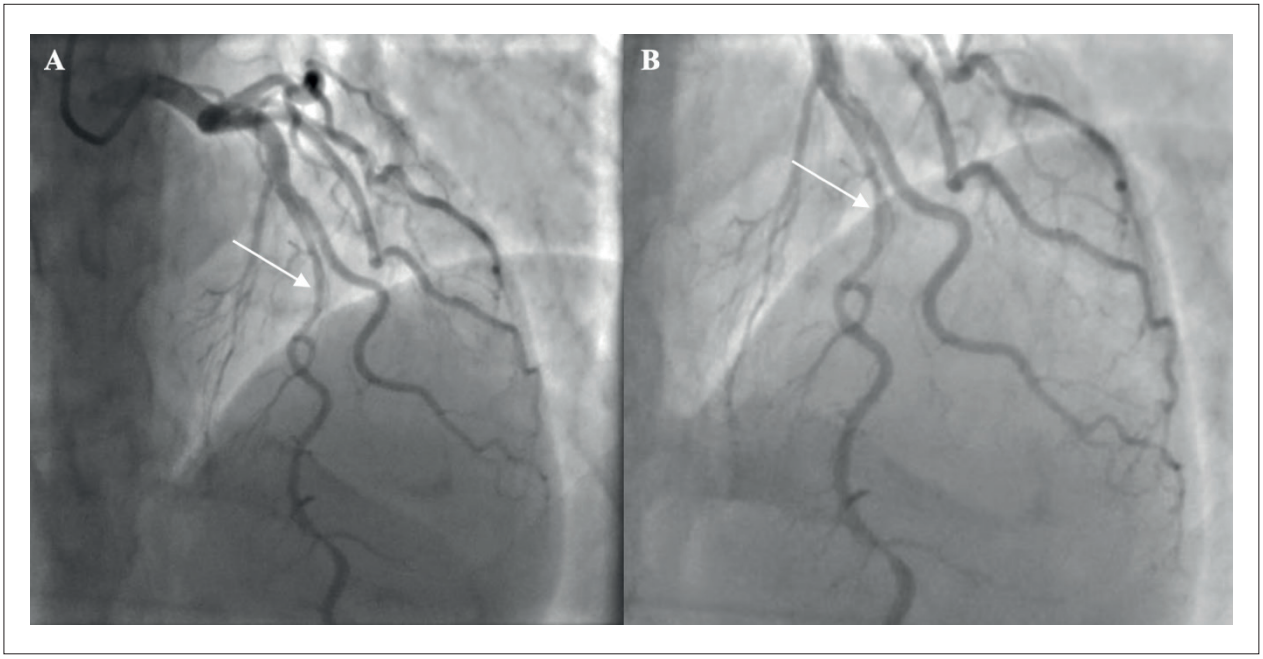

Figure 1 – *Dissection of the left anterior descending artery visualized in the right anterior oblique cranial view during cardiac catheterization (A). Magnified view of the dissection (B).*

sestamibi and dipyridamole was requested, along with coronary computed tomography (CT) angiography to monitor coronary anatomy and assess the dissection plane. It was decided to maintain clinical management with no indication for percutaneous intervention at that time. Anti-anginal therapy was optimized by increasing the metoprolol dose to 100 mg/day.

Follow-up and outcomes

The patient returned for follow-up on July 15, 2024. Clinically, she continued to experience episodes of nonspecific chest discomfort, associated with atypical chest pain described as a burning sensation lasting for hours, radiating to the left upper limb, and worsening when lying on the left side.

Laboratory tests revealed normal blood count, renal and liver function, no electrolyte abnormalities, urinalysis without crystals, and negative serologies for HIV, syphilis, and hepatitis B and C. Her LDL cholesterol level was 62 mg/dL, and NT-proBNP was 53 pg/mL. Myocardial perfusion scintigraphy showed no evidence of myocardial ischemia. Coronary CT angiography demonstrated a zero calcium score (Agatston method), no luminal coronary stenosis, and no residual signs of dissection (Figure 2).

Etiology and future follow-up

Following the July 2024 appointment, the patient continued regular follow-up at our center. She did not experience any further episodes of chest pain with characteristics suggestive of angina. Further investigation into the etiology of the coronary dissection was conducted, including assessments for Marfan syndrome, vasculitis, and collagen vascular diseases. All clinical and laboratory evaluations were negative.

Discussion

SCAD accounts for 0.1% to 4% of ACS and is more frequently observed in women, particularly young females.[3] The pathophysiology of nonatherosclerotic SCAD is not yet fully understood; proposed mechanisms include intimal tears, bleeding of the vasa vasorum, arterial inflammation, and vessel tortuosity. Approximately 20% of cases are classified as idiopathic. Other reported etiologies include pregnancy, use of oral contraceptives, genetic predisposition, and associations with certain conditions such as migraines, connective tissue disorders, and others.[4,5]

SCAD should be considered in any young patient presenting with ACS or cardiac arrest, particularly in females without traditional coronary artery disease risk factors.[6] Emotional stress, physical exertion, and hormonal influences have been identified as potential triggers for SCAD. The diagnostic workup should include electrocardiography, cardiac biomarkers, and coronary angiography. Echocardiography is valuable for assessing ventricular function, prognosis, and for differentiating from other conditions, such as Takotsubo syndrome. SCAD is classified into three angiographic types: type 1, characterized by contrast dye staining of the arterial wall with multiple radiolucent lumens; type 2, presenting as diffuse stenosis of varying severity, from mild narrowing to complete occlusion; and type 3, which mimics atherosclerosis, presenting as focal or tubular stenosis. Intracoronary imaging, coronary CT angiography, or cardiac magnetic resonance imaging may aid in diagnosis, particularly in ambiguous cases.[7,8]

Revascularization in patients with SCAD remains challenging and is associated with higher rates of procedural failure and complications. Therefore, conservative management is

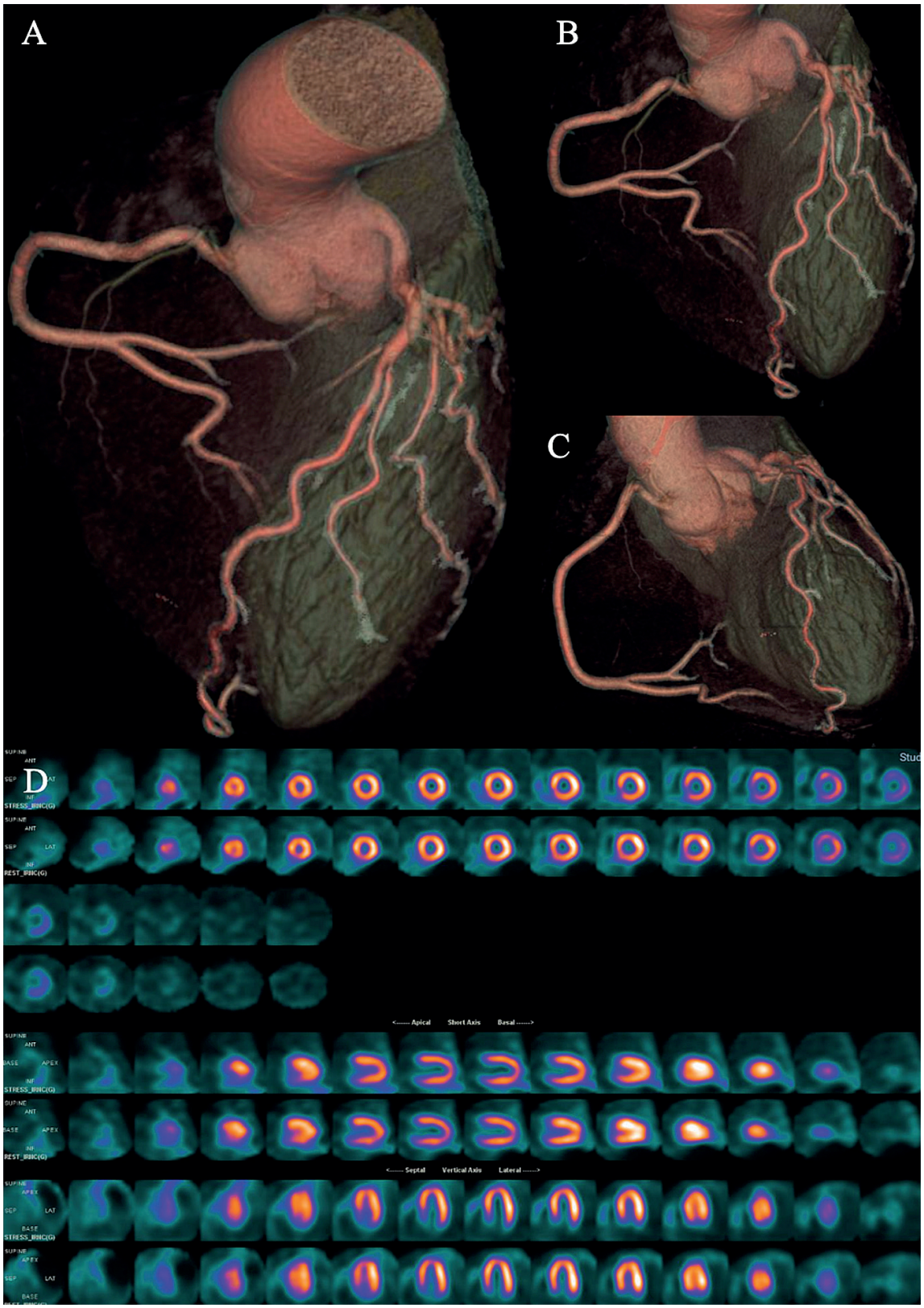

Figure 2 – *Coronary computed tomography angiography and myocardial perfusion scintigraphy during follow-up.*

generally recommended as the first-line treatment, unless the patient presents with ongoing ischemia, hemodynamic instability, or left main coronary artery involvement.[9] Angiographic "healing" of SCAD lesions has been frequently observed following conservative management. Regarding long-term management, there are currently no studies directly comparing different therapeutic strategies. As such, treatment is often extrapolated from ACS guidelines, typically including aspirin, beta-blockers, statins, and antianginal agents. It is considered reasonable to discontinue dual antiplatelet therapy once dissection healing is confirmed, which is generally expected to occur within 4 to 6 weeks following the event.[7,8]

To date, only one case report describing coronary artery dissection following stress echocardiography has been identified in major medical databases. In that case, a middle-aged male patient presented with significant ST-segment elevation followed by an accelerated idioventricular rhythm. Similar to our case, the patient was managed conservatively, with complete restoration of vessel patency.[1] A limitation of our report is the inability to definitively determine whether the dissection occurred during the dobutamine stress echocardiography or whether the exam exacerbated a pre-existing SCAD, as we do not have prior imaging of the patient's coronary anatomy. Nevertheless, the notable difference in the patient's symptoms before, during, and after the examination supports the possibility that the test acted as a potential trigger for the dissection.

Author Contributions

Conception and design of the research and writing of the manuscript: Artioli T, Santos BQM, Monteiro DC, Batista LB, Lorenzoni MZ, Jen HG, Vital KM, Vilalva KH; acquisition of data: Artioli T, Santos BQM, Monteiro DC, Batista LB, Lorenzoni MZ; analysis and interpretation of the data: Artioli T, Santos BQM, Monteiro DC, Batista LB; critical revision of the manuscript for intellectual content: Artioli T, Lorenzoni MZ, Jen HG, Vital KM, Vilalva KH.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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Study Association

This study is not associated with any thesis or dissertation work.

Ethics Approval and Consent to Participate

This study was approved by the Ethics Committee of the Instituto Dante Pazzanese de Cardiologia under the protocol number 89472225.7.0000.5462. All the procedures in this study were in accordance with the 1975 Helsinki Declaration, updated in 2013. Informed consent was obtained from all participants included in the study.

Use of Artificial Intelligence

The authors did not use any artificial intelligence tools in the development of this work.

Availability of Research Data

The underlying content of the research text is contained within the manuscript.

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