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CASE REPORT

Acute Ischemic Stroke as the Only Clinical Manifestation of Spontaneous Coronary Artery Dissection in an Athlete: A Case Report

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Introduction

Spontaneous coronary artery dissection (SCAD) is a rare but important cause of acute coronary syndrome.[1] SCAD involves the spontaneous formation of an intramural hematoma in coronary arteries, frequently presenting as severe acute-onset chest pain.[1] Although extremely rare, cases of acute ischemic stroke (AIS) secondary to SCAD have been reported in the literature,[2,3] none of which were secondary to intracoronary thrombus displacement.

In this report, we present the case of a 39-year-old man with no atherosclerotic risk factors who presented with AIS and myocardial infarction secondary to SCAD.

Case Report

A 39-year-old tennis coach with no significant medical history or atherosclerotic risk factors presented to the emergency department with sudden mental confusion, dysarthria, and left leg paresis.

He was admitted to a tertiary hospital 1 hour after the onset of symptoms, which regressed almost completely after 2 hours of observation. On admission, the patient had a regular pulse (72 bpm) and blood pressure of 129/70 mm Hg. The clinical impression was AIS. A non-contrast computed tomography of the head was performed 1 hour after admission and showed no abnormalities. Subsequent magnetic resonance imaging of the brain showed signs suggestive of acute cortical damage due to ischemia in the region of the right middle cerebral artery. Doppler imaging of the carotid and vertebral arteries showed normal measurements.

Electrocardiography showed sinus rhythm with inferior Q waves, minor right bundle branch block, and diffuse repolarization abnormalities. An echocardiogram showed inferior wall hypokinesia and good global systolic function. Troponin dosage was 12 ng/mL (normal dose is ≤ 0.04 ng/mL), and acute inferior wall myocardial infarction was diagnosed. The patient denied precordial pain. Laboratory screening for hypercoagulability syndromes was negative.

Coronary angiography revealed proximal occlusion of the right coronary artery (receiving grade 2 coronary collaterals) and no stenosis in other arteries (Figures 1a and 1b). During the second contrast injection, a more proximal occlusion of the right coronary artery was observed, including subocclusion of a right marginal branch that had previously been open, suggesting instability and proximal mobilization of a thrombus. Left ventriculography showed significant hypokinesia of the inferior wall with preserved global systolic function.

Since the patient was stable and without ventricular dysfunction, having right coronary artery occlusion > 24 hours and grade II collaterals, we decided not to treat the affected artery at that time. Coronary dissection was considered a pathophysiological mechanism for the acute coronary syndrome and mobilization of the coronary thrombus, which had been suggested as a hypothesis for cerebral embolism. Transesophageal echocardiography indicated no intracavitary thrombi or signs of intracardiac shunts. The patient was discharged on the fourth day after myocardial infarction, with clinical treatment including clopidogrel, aspirin, beta-blockers, and statins.

Keywords

Blood Vessel Dissection; Acute Coronary Syndrome; Ischemic Stroke.

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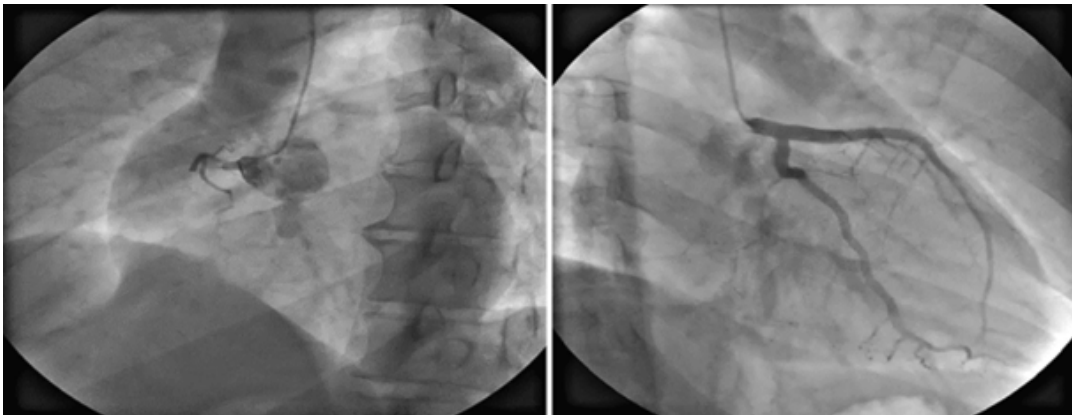

Figures 1a and 1b - Right and left coronary angiographies, performed 2 days after the initial hospitalization, showing proximal occlusion of the right coronary artery (image suggestive of a thrombus proximal to the occlusion) (1a) and the left coronary artery without atherosclerosis (1b).

The patient remained stable, with mild dysarthria and no paresis. In the second month of follow-up, he reported mild chest discomfort and dyspnea upon physical exertion. A second coronary angiography was performed, which identified a recanalized right coronary artery, with subocclusive stenosis in the proximal segment and an image compatible with a long segment of coronary dissection (Figure 2a). Angioplasty of the right coronary artery was performed, and 2 stents were implanted in the proximal and middle thirds of the right coronary artery with good results (Figure 2b).

The patient is currently asymptomatic, with mild dysarthria and no further events 2 years after the initial event.

Discussion

SCAD is a rare but potentially fatal condition thought to account for 1%-4% of all cases of acute coronary syndrome.[1] A Canadian cohort described it as occurring mainly in women, especially during the postmenopausal period.[4] The associated risk factors are fibromuscular dysplasia, peripartum, and connective tissue diseases, such as Marfan and Ehlers-Danlos syndromes.[1,4] Connective tissue diseases and fibromuscular dysplasia alter the vascular architecture, predisposing individuals to SCAD.[5] Genetic variants in FBN1, the gene associated with Marfan syndrome, and COL3A1, the gene associated with arterial dissections/ruptures and Ehlers-Danlos Syndrome, have been discovered in patients with SCAD.[5]

Physical exertion can provoke acute ischemic events, although primarily in adults with atherosclerotic disease.[6] Vigorous exercise increases coronary shear stress, which initiates the dissection.[7] Therefore, SCAD should be considered in young individuals who present with exercise-related acute ischemic events.

The incidence of an AIS event in patients with myocardial infarction is relatively low, varying from 1%-5%.[8] Several pathophysiological mechanisms can explain simultaneous ischemia in cardiac and cerebral regions. First, the same traditional risk factors act in the development and rupture of atherosclerotic plaques in both areas.[8] Furthermore, an association between AIS and acute coronary syndrome can occur through embolization during percutaneous revascularization and the mobilization of intracoronary thrombi. AIS can also occur as a result of transient arrhythmias (such as atrial fibrillation), hypokinesia, intraventricular thrombi in patients with left ventricular dysfunction, or low cerebral perfusion due to cardiogenic shock.[8]

In our patient, spontaneous mobilization of intracoronary thrombi was considered the most probable pathophysiological mechanism to explain ischemia in the cardiac and cerebral regions, given that coronary angiography suggested instability and mobilization of the intracoronary thrombus and that no other causes of AIS were observed. Although the possibility of iatrogenic dissection of the right coronary artery during angiography should be considered, Figure 1 was obtained through a non-selective injection of contrast medium into the right coronary sinus, which rules out this possibility.

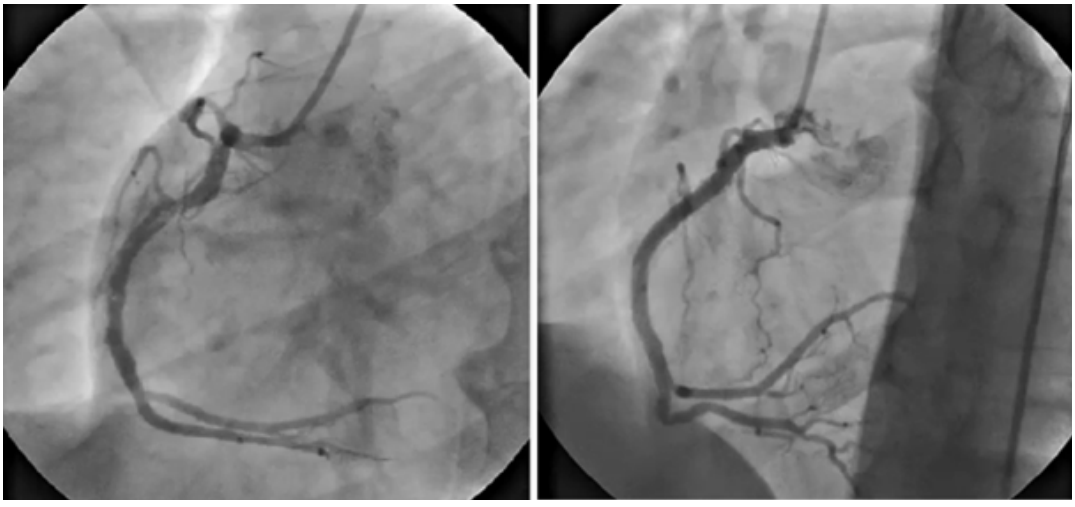

Figure 2a and 2b: (2a) Second coronary angiography, performed 30 days after the initial event, showing spontaneous recanalization of the right coronary artery and an image suggestive of extensive coronary dissection; (2b) Final angiographic result after 2 coronary stents were implanted.

Spontaneous migration of an intracoronary thrombus is not usually attributed to AIS in the peri-infarct period. However, clinical studies assessing the aspiration of thrombi during percutaneous coronary intervention in patients with myocardial infarction have identified an association between thrombus manipulation and the occurrence of AIS.[9]

Patients who concomitantly present acute coronary syndrome and AIS pose significant challenges in acute treatment.[8] In general, medical treatment is preferred over immediate revascularization for clinically stable patients with SCAD. Other factors to consider include the region at risk, the amount of myocardium at risk, and the level of distal flow in the affected vessel.[10] Thrombolysis is associated with clinical deterioration in patients with myocardial infarction due to SCAD.[10]

The percutaneous approach was postponed due to the risks associated with accessing the false lumen of the coronary vessel, as well as the possibility of thrombus migration into microcirculation after recanalization. In this case, initial clinical treatment proved very beneficial.

Conclusion

SCAD should be considered as an explanation for ischemic events in young patients without major risk factors for coronary artery disease, in athletes, and clinical situations such as peripartum and connective tissue diseases. In patients with an AIS during the peri-infarction period, identifying the cause of the AIS is important, especially for secondary prevention of new events. Mobilization of intracoronary thrombi is a rare cause, but it can be suggested in the absence of more common causes.

Author Contributions

Conception and design of the research: Rodrigues IM, Almeida RP, O'Connel JL; acquisition of data, analysis and interpretation of the data and critical revision of the manuscript for intellectual content: Rodrigues IM, Silva DML, Souza FM, Ontivero GNL, Almeida RP, O'Connel JL; writing of the manuscript: Rodrigues IM, Silva DML, Souza FM, Ontivero GNL.

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 Universidade Federal de Uberlândia under the protocol number 68845323.0.0000.5152. 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.

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