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# Type 2 variant A spontaneous dissection of the left anterior descending artery presenting as type A Wellens' syndrome: when percutaneous coronary intervention is needed

## Dissecção espontânea tipo 2 variante A da artéria descendente anterior apresentando-se como síndrome de Wellens tipo A: quando a intervenção coronária percutânea é necessária

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**ABSTRACT –** Spontaneous coronary artery dissection is defined as non-iatrogenic epicardial coronary dissection, not associated with atherosclerosis or trauma. Myocardial injury occurs due to coronary artery obstruction caused by intramural hematoma or intimal disruption, rather than atherosclerotic plaque rupture or intraluminal thrombus. We report a case of type 2 variant A spontaneous coronary artery dissection in the mid left anterior descending artery, presenting with type A Wellens' syndrome, which required percutaneous coronary intervention for relief of refractory angina and pulmonary congestion.

**Keywords:** Coronary vessels; Dissection; Wellens' syndrome; Coronary angiography; Percutaneous coronary intervention; Atherosclerosis; Atherosclerotic plaque; Hematoma; Thrombosis

**RESUMO –** A dissecção espontânea da artéria coronária é definida como uma dissecção coronária epicárdica não iatrogênica, não associada à aterosclerose ou a trauma. A lesão miocárdica ocorre devido à obstrução da artéria coronária causada por hematoma intramural ou ruptura da íntima, em vez de ruptura de placa aterosclerótica ou trombo intraluminal. Relatamos um caso de dissecção espontânea tipo 2 variante A no segmento médio da artéria descendente anterior apresentando-se como síndrome de Wellens tipo A, que necessitou de intervenção coronária percutânea para alívio de angina refratária e congestão pulmonar.

**Descritores:** Vasos coronarianos; Dissecção; Syndrome de Wellens; Angiografia coronária; Intervenção coronária percutânea; Aterosclerose; Placa aterosclerótica; Hematoma; Trombose

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## INTRODUCTION

Spontaneous coronary artery dissection (SCAD) is increasingly described as an important cause of acute coronary syndromes (ACS), particularly in young women. It is defined as non-traumatic, nor iatrogenic or atherosclerotic tear in the coronary wall. This condition has been underdiagnosed for decades, but with increased clinical suspicion, greater use of coronary angiography (CAG) and intravascular imaging (optical coherence tomography and intravascular ultrasound), diagnosis of SCAD has improved substantially.[1-3]

Compared with atherosclerotic disease, SCAD has unique risk factors and associated conditions, and different diagnostic, therapeutic, and prognostic implications. The true prevalence of SCAD remains uncertain, primarily because it is an underdiagnosed

condition. Missed diagnoses result from low suspicion of ACS in young women even with classic presenting symptoms, limitations of angiographic techniques, and lack of clinician familiarity with the condition.[1-3]

Type A Wellens' syndrome is characterized by biphasic (plus-minus) T waves in leads V2 and V3, in a pain-free state, plus isoelectric or minimally elevated (<1mm) ST-segment, and absence of precordial Q waves, with history of typical angina.[4,5]

We report a case of type 2 variant A SCAD in the left anterior descending (LAD) presenting as type A Wellens' syndrome, requiring percutaneous coronary intervention (PCI) for relief of refractory angina and pulmonary congestion. This study was evaluated by the Research Ethics Committee of the Hospital São Paulo of the Universidade Federal de São Paulo (protocol 4.071.731), CAAE 30384020.5.0000.5505.

## CASE REPORT

A 55-year-old woman, former smoker and with long-term hypertension was admitted to the emergency room complaining of rest typical angina, recurrent during the last two days. She denied any recent chest trauma as well any physical or emotional trigger. Admission high-sensitive troponin was about 18-fold the upper normal limit, and electrocardiogram (ECG), in a pain-free period, showed biphasic (plus-minus) T waves in the V2 and V3 precordial leads, compatible with type A Wellens' syndrome (Figure 1). Due to recurrent chest pain associated to *de novo* pulmonary congestion causing dyspnea and peripheral oxygen desaturation (88%), requiring nitroglycerin and supplementary oxygen, the patient was promptly referred to our cath lab. Urgency CAG was immediately performed via right distal transradial access, in the anatomical snuffbox (our default access site for any routine CAG and/or PCI[6-8]) and showed normal coronary arteries, except for a mid-LAD diffuse and severe luminal narrowing, bordered by normal proximal and distal segments (Figure 2 and Videos 1 to 4), compatible with type 2 variant A SCAD, according to the Saw angiographic SCAD classification.[3] Left ventriculography showed hypokinesia of mid-apical segments of anterior and septal walls. Despite the good results with conservative management, due to refractory angina and secondary pulmonary congestion, it was decided to perform PCI. After left coronary artery catheterization with a XB 3.5 6F guiding catheter (Cordis, Miami Lakes, Florida, United States), followed by extremely careful wiring crossing (Video 5) with a 0,014" 190cm BMW Universal II guidewire (Abbott, Abbott Vascular, Redwood City, CA, United States), a long 3.0/40mm drug-eluting stent (DES) was directly delivered through the narrowed segment, and

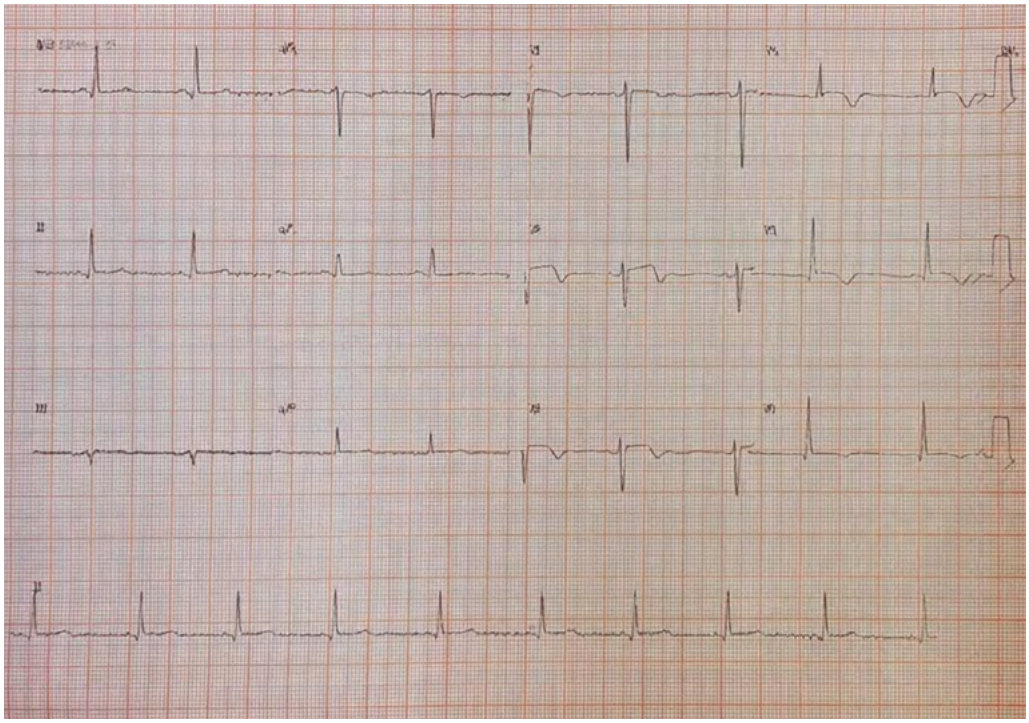


**Figure 1**. Baseline eletrocardiogram showing biphasic plus-minus T waves in the precordial leads, compatible with type A Wellens' syndrome.

properly deployed at nominal pressure. After that, two *de novo* findings were noted: a radiolucent flap behind stent struts, and a proximal stent edge haziness (after several administrations of intracoronary nitroglycerin to rule out spasm), both secondary to intramural hematoma (IMH) compression and disruption, with retrograde propagation (Video 6). Thus, an additional 3.5/15mm DES was carefully deployed (nominal pressure), with minimal overlap, and just an extra minimal post-dilation with the same balloon was performed to optimize the overlapping apposition, not contributing to more IMH progression. Great result with Thrombolysis in Myocardial Infarction (TIMI) 3 flow was achieved, with no branches lost or proximal or distal stents edge dissections (Figure 3 and Videos 7 to 10). Of note, intracoronary imaging modalities were not possible due to reimbursement constraints.

Same-day post-PCI transthoracic echocardiogram surprisingly showed complete resolution of all left ventricle wall motion abnormalities. Two days after, the patient was discharged home, with optimal medical treatment (aspirin, clopidogrel, atorvastatin, metoprolol and enalapril). Upon 14-day return visit, she was completely asymptomatic, and unremarkable initial clinical progression.

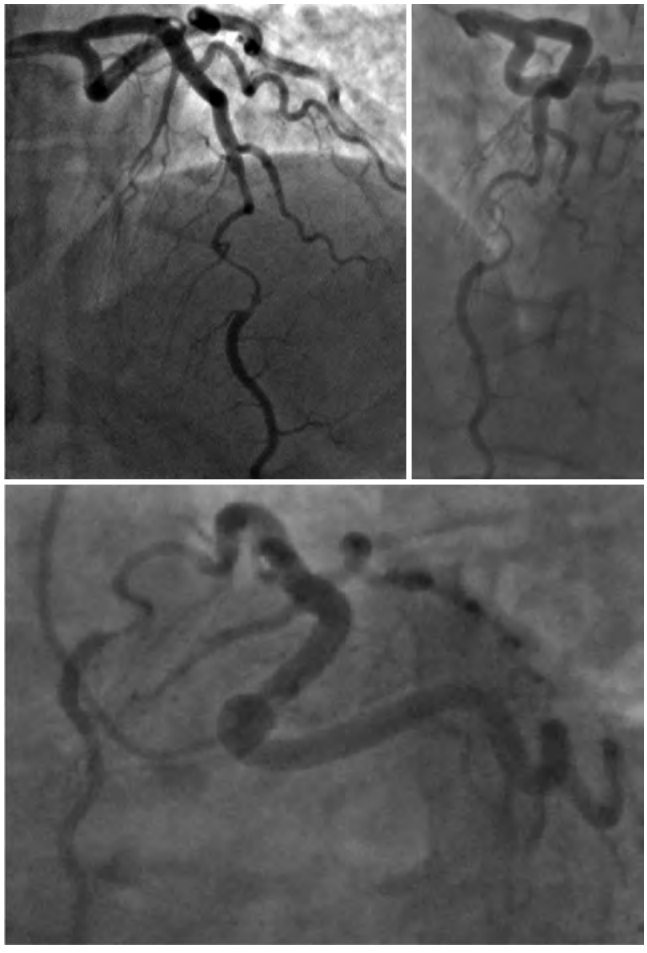
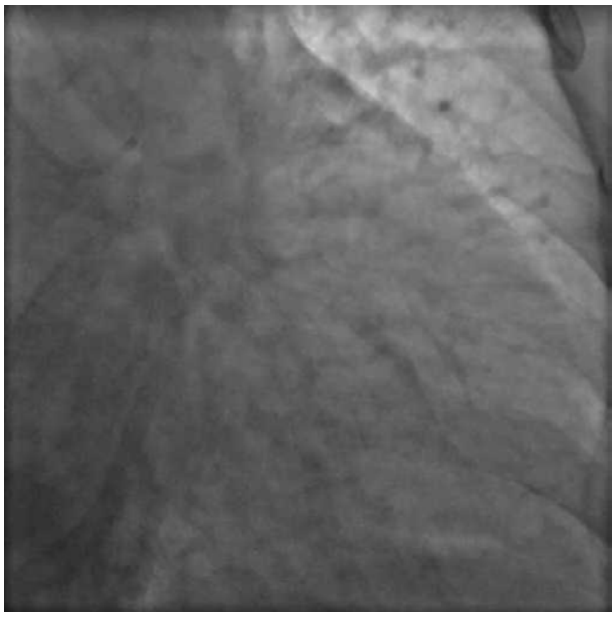
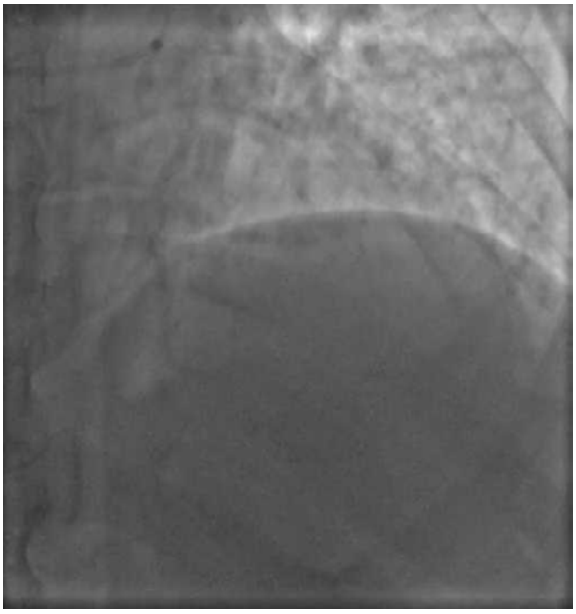


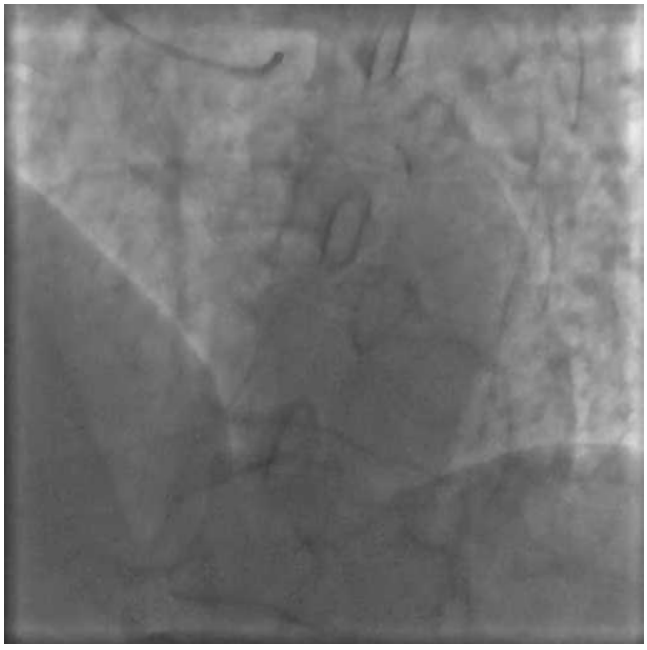
**Figure 2**. Baseline coronary angiography in different angiographic projections: mid left anterior descending diffuse and severe luminal narrowing, bordered by normal proximal and distal segments.



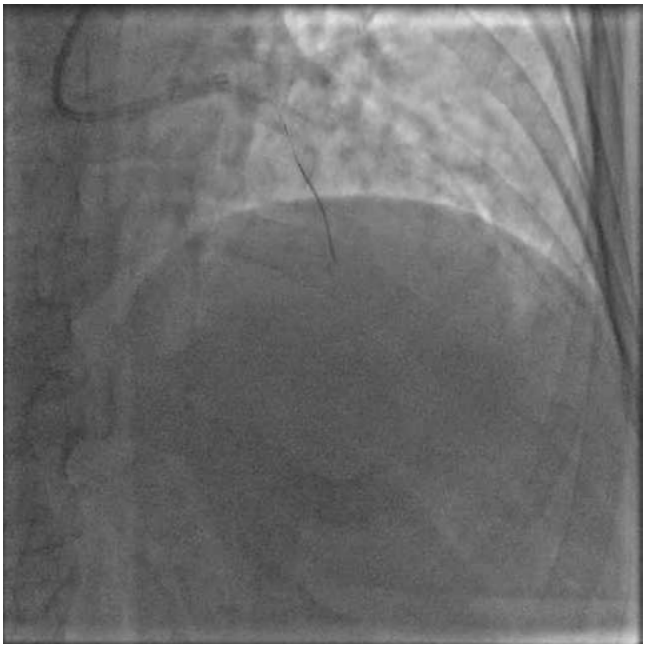
**Video 1**. Baseline coronary angiography. Mid left anterior descending diffuse and severe luminal narrowing, bordered by normal proximal and distal segments. Caudal view.



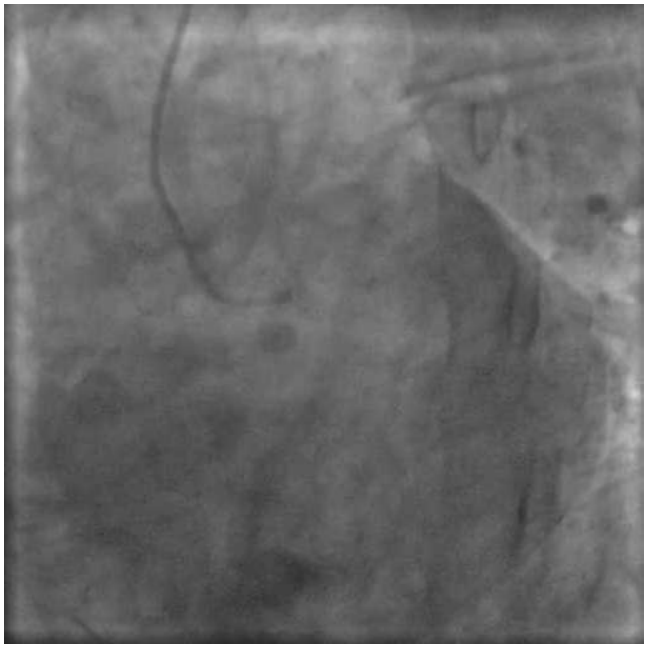
**Video 2**. Baseline coronary angiography. Mid left anterior descending diffuse and severe luminal narrowing, bordered by normal proximal and distal segments. Cranial view.



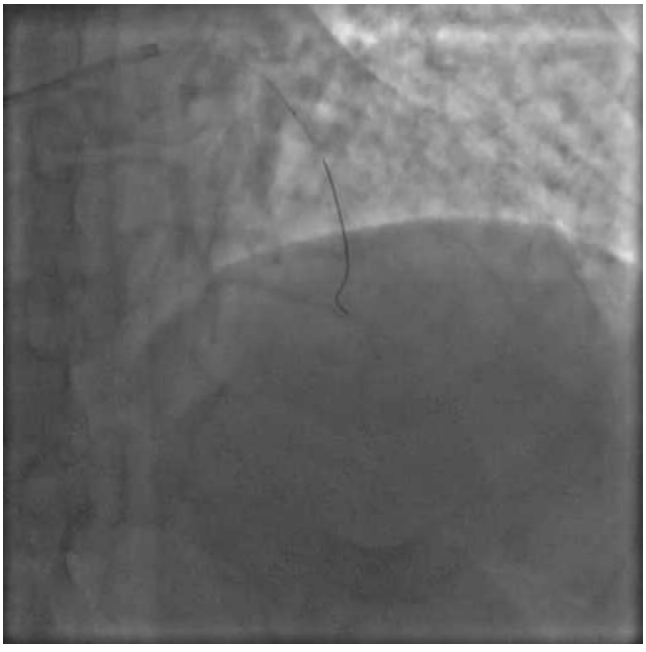
**Video 3**. Baseline coronary angiography. Mid left anterior descending diffuse and severe luminal narrowing, bordered by normal proximal and distal segments. Left anterior oblique cranial view.



**Video 5**. Careful wiring through the long, tortuous and angulated left anterior descending spontaneous coronary artery dissection segment.



**Video 4**. Baseline coronary angiography. Mid left anterior descending diffuse and severe luminal narrowing, bordered by normal proximal and distal segments. Spider view.



**Video 6**. Radiolucent flap behind stent struts and proximal stent edge haziness, secondary to intramural hematoma compression and disruption, with retrograde propagation.

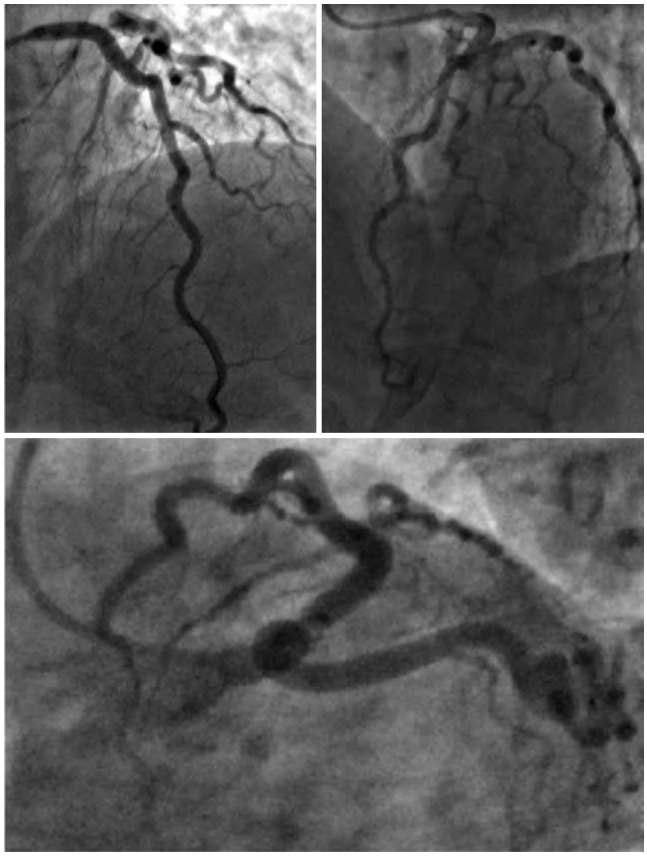
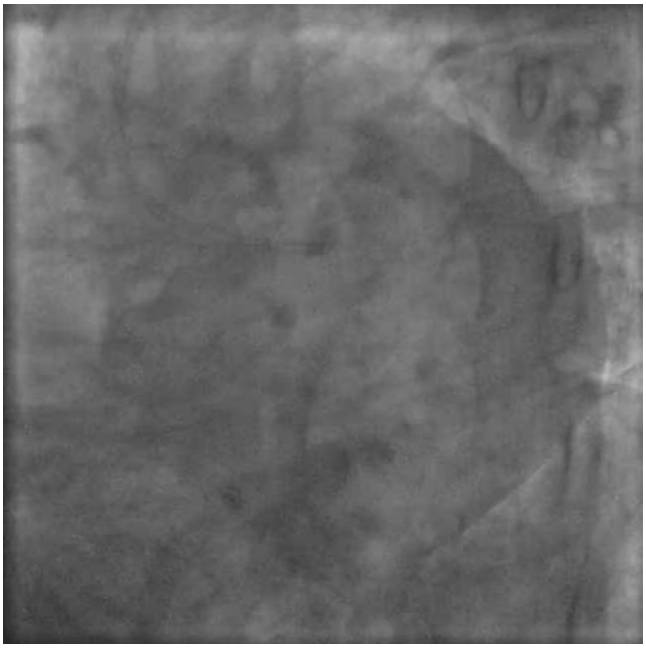
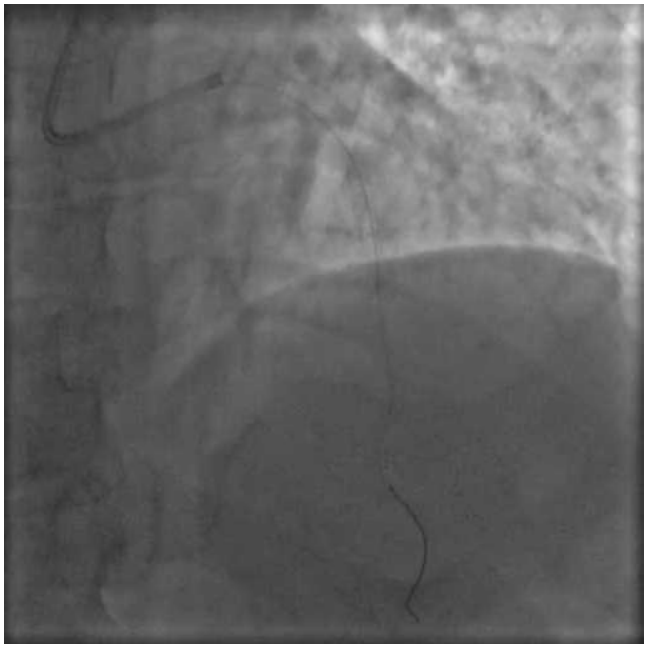


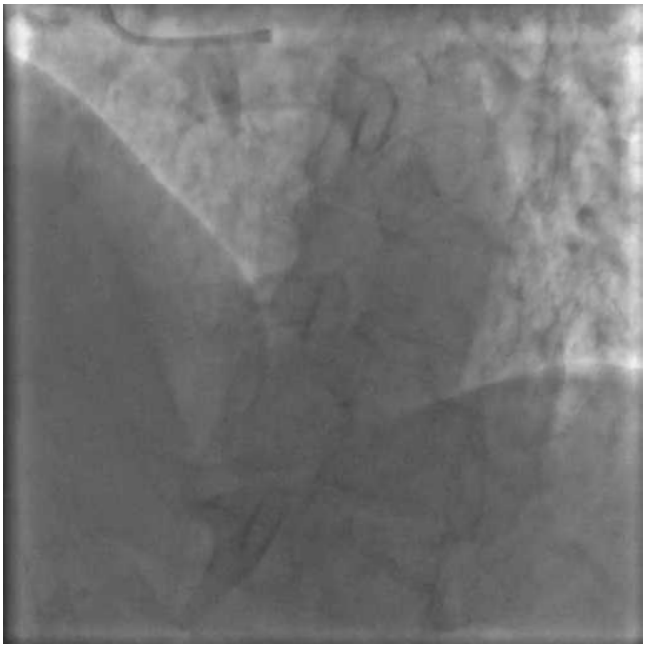
**Figure 3**. Result in different angiographic projections.



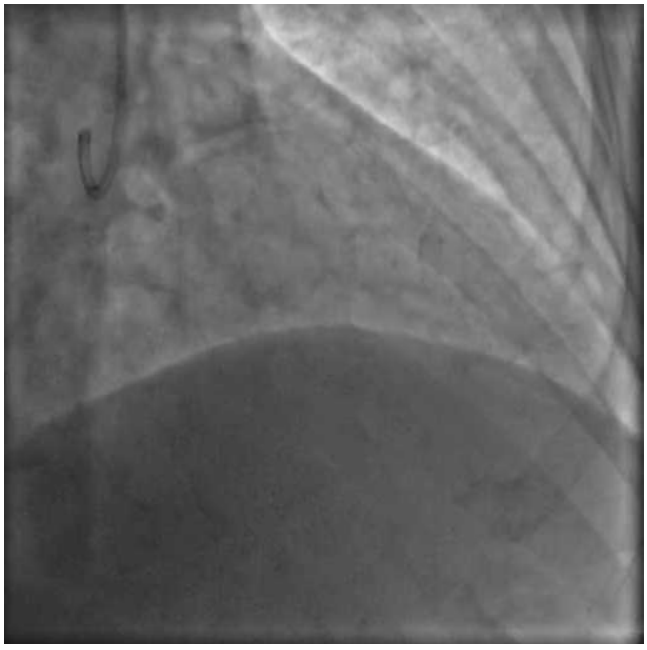
**Video 8**. Result. Spider view.



**Video 7**. Result. Cranial view.



**Video 9**. Result. Left anterior oblique cranial view.



**Video 10**. Final result. Right anterior oblique cranial view.

## DISCUSSION

Accurate diagnosis of SCAD in the early stages of ACS presentation is fundamental because management and investigation are different from those with atherosclerotic forms of coronary disease. Our SCAD patients have been continuously included in the Spontaneous Coronary Artery Dissection anaLysIs of the Brazilian Updated Registry (SCALIBUR Registry, ClinicalTrials.gov: NCT03398850)

Spontaneous coronary artery dissection is now estimated to be the underlying cause of up to 4% of ACS. In women aged under 60 years, SCAD accounts for 22% to 35% of ACS presentations. It has become increasingly apparent that multiple factors may predispose to an arteriopathy that weakens the arterial wall, and increases vulnerability to dissection. This can be exacerbated by precipitating stressors (emotional or physical), triggering dissection.[1,2]

The traditional angiographic description of SCAD emphasized the presence of multiple radiolucent lumens and extraluminal dye staining. However, contemporary angiographic SCAD series have shown this pathognomonic appearance is present in only a few cases.[1-3] Addressing the drawbacks of prior descriptions, the Saw angiographic SCAD classification was proposed and now has been commonly adopted. Type 1 refers to the classic appearance of multiple radiolucent lumens and/or arterial wall contrast staining. Type 2 refers to the presence of diffuse stenosis of varying severity and length (usually >20mm): variant 2A (as in the present case) is bordered by normal segments proximal and distal to the IMH, and variant 2B extends to the distal tip of the artery. Type 3 refers to focal or tubular stenosis, usually <20mm in length, that mimics atherosclerosis; intracoronary imaging is required to confirm the presence of IMH and to thus diagnose SCAD.[3]

Despite the inherent limitations of CAG that reduce the diagnostic capability for SCAD, it remains the first-line diagnostic imaging method, because it is widely available and recommended for early invasive management of ACS. Intravascular ultrasound and particularly optical coherence tomography provide detailed visualization of the arterial wall, refining the diagnosis of SCAD.[9] However, these tools have additional risks and costs, and are not readily available in all cath labs. As a consequence, conventional CAG remains instrumental in diagnosing SCAD, and cardiologists should become proficient at recognizing its various angiographic patterns. Fortunately, the need to use intracoronary imaging has diminished in recent years because of improved SCAD-pattern recognition on CAG.[1,2]

Usually, conservative management is preferred, except for high-risk and/or unstable patients, as in the present case, because PCI at the time of SCAD is associated with high failure rates, and most SCAD lesions heal spontaneously.[1,2] Despite lack of comparative studies assessing their superiority over conservative management, specific strategies have been described for SCAD PCI: meticulous angiographic and instrumentation techniques; long DES covering beyond proximal and distal edges of the IMH, to accommodate its propagation when compressed by the stent; and direct stenting without balloon predilation to avoid additional risks of extension of the IMH.[1] In the present case, all these recommendations were complied with.

Likewise, in the classic proximal LAD atherosclerotic stenosis, myocardial bridging[10] and SCAD, like in the present case, can mimic Wellens' syndrome.

In conclusion, the diagnosis of SCAD on CAG is elusive. Since most are not familiar with angiographic variants of SCAD, interventional cardiologists and clinicians should have a high index of suspicion for SCAD especially in young women presenting with ACS without traditional cardiovascular risk factors.

## 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: MDPO, TS, IRB, AT, CL and AC; data collection: MDPO, TS, IRB, AT, CL and AC; data interpretation: MDPO, TS, IRB, AT, CL and AC; text writing: MDPO and AC; approval of the final version to be published: MDPO, TS, IRB, AT, CL and AC.

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