

# Cardiac Arrest and Tortuosity of Artery as Major Predictors Response of Rare Case of Spontaneous Coronary Artery Dissection

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## Abstract

**Background:** Spontaneous coronary artery dissection (SCAD) may be a predictor of fibromuscular dysplasia (FMD) that affects Caucasian women, lean, between 15 and 50 years of age and with no significant historical predominance in the female sex, from 75 to 100% of cases. Within this, it is known that the main predictors of this enzymatic derangement can be postpartum periods, multiparity, tissue disorder, hormone therapies (use of anti-conception), use of certain illicit drugs, physical stress and emotional stress. In smaller proportions, it is associated with disorders such as the Marfan, Ehlers-Danlos and Horner Syndromes.

**Objective:** It was to report a rare case of spontaneous coronary dissection, with previous involvement of cardiac arrest.

**Case:** Female patient, 40 years old, presented pain in precordial region in tightening with irradiation to left upper limb. The patient denied a history of hypertension, Diabetes mellitus, dyslipidemia and smoking, and was unable to report a family history. However, she reported an episode of sudden cardiac arrest three years before and presented with grade I obesity. She also presented tortuosity of the coronary arteries and did not report migraine headaches, and after exams (cineangiographies) she was diagnosed with SCAD.

**Conclusion:** Due to the episode of acute myocardial infarction and the presence of coronary tortuosity, as predictors of SCAD, the patient was diagnosed with this condition and, after the treatments mentioned in the above text, the patient remained asymptomatic.

**Keywords:** Spontaneous coronary artery dissection; Fibromuscular dysplasia; Cardiac arrest; Tortuosity

## Introduction

Spontaneous coronary artery dissection (SCAD) can manifest with acute ischemic chest pain, affecting patients with underlying coronary atherosclerosis, women in the third trimester of gestation or in the early puerperal period, and idiopathic patients [1-8]. Such pathology may be a predictor of fibromuscular dysplasia (FMD) that affects Caucasian, lean, 15- to 50-year-old women with no history [9]. There is a marked predominance in females, from 75.0 to 100.0% of the cases, with a mean age between 30 and 55 years [9]. Some studies also identify this lesion in older postmenopausal women.

In this context, SCAD is a non-traumatic event generated by the segmentation of the coronary artery wall, creating a false lumen [1-4]. It is an infrequent cause of acute coronary syndrome (unstable angina) and sudden death. As its pathological mechanism is poorly understood, it is only known that SCAD is associated with the vascular system, inflammatory processes and vasculopathies, and fibromuscular dysplasia (FMD) is the main one, with tortuosity being the main morphological sign [1].

Thus, the pathogenesis of FMD can occur due to the fragmentation of elastic fibers that are degraded by matrix metalloproteinases 2 and 9 (MMP2 and MMP9) [1]. However, the role of MPs and their inhibitors in the pathogenesis of FMD remains completely unexplored, being the main information gap that will be explored in the next investigations of the present and the next cases.

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As the pathophysiological control of the action of MMPs is still unclear, SCAD treatment remains in the decision to use invasive or non-invasive methods, through the analysis of clinical and angiographic factors that include the site of dissection, number of vessels affected, and condition Hemodynamic in hospital evolution [9]. As an example, coronary angioplasty with stent implantation is able to restore flow, alleviate symptoms and treat dissection, but has a 65% success rate. Coronary artery bypass surgery is reserved for multivessel SCAD and trunk dissections. In addition, there are no studies evaluating optimal long-term drug treatment [11].

The objective of the present study was to present a rare case report of spontaneous coronary dissection, with previous involvement of cardiac arrest.

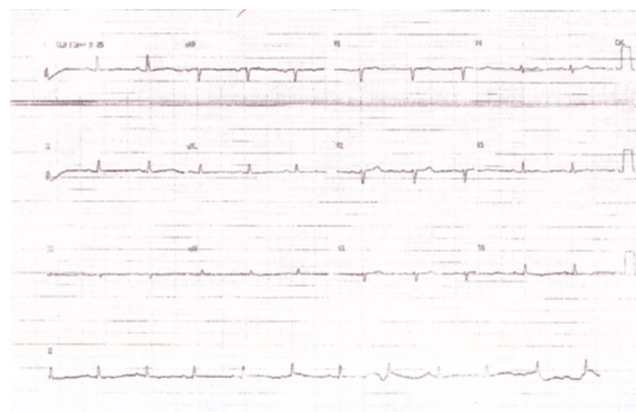
## Case Report

A 40-year-old female patient was admitted to the emergency department of the Beneficência Portuguesa Hospital from Sao Jose do Rio Preto, presenting pain in the precordial region in tightening with irradiation to the left upper limb. She reported that the pain had started one hour before the entrance, during her work shift as a nursing technique. The pain, in its beginning, was maintained for approximately 10 minutes, with improvement for 20 minutes, returning with greater intensity and taking it to the search the emergency service.

The patient denied a history of hypertension, Diabetes mellitus, dyslipidemia and smoking, and was unable to report a family history. However, she reported an episode of sudden cardiac arrest three years earlier and presented with grade I obesity. She presented tortuosity of the coronary arteries and did not report migraine headaches. An electrocardiogram (Figure 1) and laboratory tests were performed, which were within the normal range. The enzymes CPK-MB and Troponin underwent significant changes in 14 h of analysis (from 1.9 to 5.0 and from 8.25 to 68.50, respectively), which indicated myocardial injury. She was medicated with 200 mg of ASA, 300 mg of Clopidogrel, 80 mg of Atorvastatin and 5 mg of sublingual Isosorbide Dinitrate, showing improvement of the condition. Due to the possible myocardial ischemia, the medical team decided to hospitalize it with prescription ASA, Ticagrelor, Enoxaparin, Trimetazidine, Ramipril, Atorvastatin and Metoprolol Succinate.

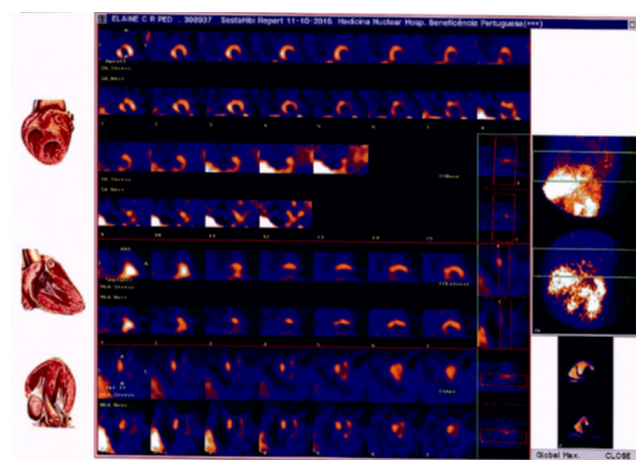
Two days after hospitalization, she presented again chest pain with irradiation for left upper limb, but this time with lower intensity. She was again submitted to laboratory tests that were normal. In less than 24 h, the exams showed significant increases of the CPK-MB and Troponin enzymes (from 1.4 to -41.3 and from 31.94 to 199.70, respectively), which revealed a myocardial necrosis. Thoraco-abdominal angio-tomography showed no abnormalities, with the following findings: thoracic and abdominal aortic permeability, supra-aortic trunks with normal limits, good visceral branches and pelvic vessels without evidence of parietal changes and Good permeability of the pelvic arteries. Myocardial perfusion scintigraphy was also performed (Figure 2), which revealed myocardial ischemia in a small area (approximately 9.0% of LV mass), in the inferior apical and inferolateral segments, with associated persistence component and suggestive zone of fibrosis at the cardiac apex.

"Cardiac catheterization" showed subocclusion only in the middle 1/3 of LAD (left anterior descending artery) and left ventricular systolic dysfunction, resulting in the diagnosis of spontaneous coronary artery dissection (SCAD). The medical team began treatment with triple antiplatelet aggregation and optimization of clinical therapy. After seven days, a significant decrease in CPK-MB (from 5.6 to 0.9) and Troponin (from 857.5 to 91.12) was observed in a laboratory reassessment.



**Figure 1:** Result of ECG-electrocardiogram within normality.

The patient remained asymptomatic until a new "catheterization" was performed 10 days after the onset of the disease. As it presented ischemic dysfunction (in scintigraphy), angioplasty was chosen. Three stents were then implanted in the anterior descending artery and a stent was placed in the first diagonal branch due to a retrograde dissection (Figures 3 and 4). The procedure proceeded well, with a good evolution of the patient, which remained asymptomatic.



**Figure 2:** Myocardial perfusion scintigraphy was performed, revealing myocardial ischemia in a small area (approximately 9.0% of LV mass), in the inferior apical and inferolateral segments, with associated persistence component and zone suggestive of fibrosis at the cardiac apex.



**Figure 3:** Images of cinecoronary angiography before stents implantation and after 4 stents. Cinecoronary angiography of the left coronary artery in PA-cranial showing dissection image in the middle segment of the anterior descending artery.

## Discussion

The present clinical case presented a spontaneous coronary artery dissection that is a rare cause of acute myocardial infarction, potentially fatal (50.0% with sudden death) and estimated incidence of cineangiographies from 0.04% to 0.20% [12], with previous rare case of acute myocardial infarction for 3 years as a predictor response of SCAD. The anterior descending coronary artery is the most affected vessel in SCAD, with a prevalence of 40.0 to 60.0%, followed by the circumflex artery (30.0%) and its branches, mainly in the medial and distal segments [9]. In addition, the disease may be associated with a genetic component, since the disease reaches primarily Caucasians with the HLA-DR6 histocompatibility antigen [10].



**Figure 4:** Images of cinecoronary angiography before stents implantation and after 4 stents. Final result with image luminal reduction of the anterior descending artery proximal to the stents.

Furthermore, at the molecular level, SCAD may also be related to the expression of MMPs and the consequent degradation of elastin [11]. As a result, the fragmentation of elastic fibers in the coronary arteries is due to the imbalance between metalloproteinase 2 (MMP2)

and metalloproteinase 9 (MMP9) on its inhibitor TIMP1 and TIMP2 [12]. Perhaps this would be the main cause of pathology and enzymatic homeostasis the form of treatment. Thus, we will make a future study about the effect of MMPs as a predictor of MFD, as a way to deepen the investigations.

At the genetic level, a common variant rs9349379 located on chromosome 6 in the phosphatase and actin-regulator gene 1 (PHACTR1) has been shown to increase the risk of foot-and-mouth disease (FMD) by about 40% [8]. Furthermore, rs9349379 correlates with the expression of PHACTR1 in fibroblasts of patients with foot-and-mouth disease and controls. The same allele that increases the risk of foot-and-mouth disease is at risk of cervical artery dissection and migraine, often reported in patients with FMD. However, the clear role of PHACTR1 in maintaining vascular vessel integrity is not fully elucidated [8,13-21].

In general, the diagnosis is performed by "catheterism". In its embodiment, an intramural hematoma of difficult visualization can be detected. Despite this, the diagnosis may be imprecise. Therefore, other technologies, such as coronary computed tomography (CCT) and intravascular ultrasound (IVUS), are being used to diagnose this condition. The symptomatology depends on which of the coronaries suffered the dissection process and how dissected it is, and the patient may present asymptomatic or with a clinical similar to AMI.

In this context, the prognosis of patients who survive has survival rates of around 80.0% in 30 months, but half of the patients can evolve with a second dissection after two months [12]. However, women present lower survival rates than men, especially when the event occurs outside the peripartum period [12].

As a literary support, studies have examined the hypothesis of the relationship between SCAD and the presence of FMD detected in other arterial sites. As an example, authors performed angio-CT and angio-MRI of whole body in 12 patients with SCAD, with three cases of renal FMD [9,13]. Thus, the low incidence of FMD is related to the low sensitivity of the imaging methods, not detecting stenosis of less than 50.0% in the middle and distal arterial segments [9].

In addition, other authors found a prevalence of 50.0% FMD in the external iliac artery of 16 patients with SCAD undergoing iliofemoral angiography [15]. Moreover, another study analyzed 50 patients with SCAD with 86.0% of them with FMD [16]. Thus, the evaluation of family members of patients with SCAD in order to investigate the presence of FMD is not well defined, only 7.0% of the patients had a family history.

Another study with a population of 144 patients reported treatment and follow-up for fibromuscular dysplasia. Follow-up after carotid surgery did not show cardiac arrest, stenosis or death. Persistent normalization of blood pressure was achieved in 11 (73.3%) cases. Thus, the surgical treatment of patients with some forms of FMD can provide satisfactory results if the recommended strategy is followed [2].

Also, between 2008 and 2010, a series of 5 patients hospitalized for acute coronary syndrome was evidenced by means of coronary angiography, spontaneous coronary dissection. In these cases, intracoronary ultrasonography was useful both for the diagnosis of spontaneous coronary dissection and for guiding percutaneous treatment [5,21].

To corroborate, a retrospective, unicentric study identified 25 patients with spontaneous coronary artery dissection, of whom 56.0%



were females, aged  $48.8 \pm 10$  years. In 92.0% of the cases the clinical picture was of acute coronary syndrome. The conservative strategy was performed in 56.0%, percutaneous coronary intervention in 40.0% and myocardial revascularization in 4.0%. The choice of different therapeutic strategies confirms the still controversial nature of the ideal approach of spontaneous coronary artery dissection, with an emphasis on individualized treatment [6,17,18].

## Conclusion

Due to the episode of previous acute myocardial infarction and the presence of coronary tortuosity, as predictors of SCAD, the patient was diagnosed with this pathology and, after the treatments mentioned in the text above, the patient remained asymptomatic. In addition, we will follow a study on the action of MMPs as predictors of spontaneous coronary artery dissection.

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