Case Report

# Multivessel Coronary and Cerebral Vasospasm in a Post-Acute Myocardial Infarction Patient

*Mateus Amorim Aboboreira,*[1] *Julio Paiva Maia,*[1] *Vitória Burin Borges,*[1] *Felipe Hideaki Ueda,*[1] *Marcos Franchetti*[1]

*Universidade Estadual de Maringá,*[1] *Maringá, PR – Brazil*

## Introduction

Coronary vasospasm was initially described by Prinzmetal et al. as angina that occurs at rest, without being provoked by an increase in oxygen demand, unlike classic Heberden's angina, which is induced by physical or emotional stress.[1] The pathophysiology of coronary vasospasm is multifactorial, and various precipitating factors exist.[2] The present case demonstrates an intriguing scenario that highlights the complexities of clinical management and etiological determination of vasospasm in a post-acute myocardial infarction patient.

### Case Report

We report the case of a male patient, Asian ethnicity, 58 years old, with no history of systemic arterial hypertension or diabetes mellitus, an active smoker since the age of 23, allergic to dipyrone, and using a nicotine patch. He was diagnosed with ST-elevation myocardial infarction (STEMI) in March 2023, presenting electrocardiographic changes with areas of ischemia in the anterior wall (Figure 1). On that occasion, he was referred for urgent hemodynamic study, which revealed obstructive lesions of 80% and 70% in the left anterior descending artery (LAD) and first diagonal branch (DG1), respectively. The remaining arteries did not show significant lesions.

A zotarolimus-eluting stent (Resolute Onyx™) measuring 4.0 × 38 mm was implanted in the proximal and middle third of the LAD, and balloon dilation was performed in the DG1, using a provisional bifurcation technique.

The patient developed hyperactive delirium 4 days after the procedure, due to withdrawal syndrome (nicotine dependence). The patient also presented with acute kidney injury, which was managed conservatively with hydration and discontinuation of vasodilators. Additionally, the patient developed bronchopneumonia, characterized by frequent aspirations and abundant secretions, which was treated with intravenous antibiotic therapy. Chest computed tomography scan revealed moderate bilateral pleural effusion, ground-glass opacities, and bilateral consolidations. Neurologically, the patient experienced a decreased level of consciousness with right hemiplegia. Brain magnetic resonance imaging showed signal alteration at the cortico-subcortical junction of the left cerebral hemisphere, involving the left frontal and parietal cortices, suggestive of an acute ischemic lesion (Figure 2). Cranial computed tomography scan showed no abnormalities.

Concurrently, the patient presented a new episode of STEMI, this time in the inferior wall. A new catheterization was performed, which revealed vasospasm throughout the right coronary artery, mainly in the middle and distal thirds, causing distal occlusion of the posterior ventricular branch. The left main coronary artery showed discrete spasm. In the LAD, the previous stent showed no signs of complications, but spasm was present in the middle third, causing an obstruction greater than 70%. Vasospasm was also observed in the diagonal branch, without signs of worsening atherosclerotic lesion in its proximal third. The circumflex artery presented irregularities and discrete vasospasm (Figure 3).

Given these findings, the etiological hypothesis for the new acute coronary syndrome was coronary vasospasm, with no identifiable cause at first, as well as cerebral ischemia due to arterial vasospasm.

Initial management, after diagnosis, was nitroglycerin to reverse the stenosis, which was successful. The patient remained in the intensive care unit, still experiencing episodes of hyperactive delirium. He showed full recovery of motor strength with no ischemic sequelae, progressed with gradual improvement in renal function and infectious condition, and was discharged in April 2023. Currently, he remains asymptomatic under routine clinical follow-up, still a smoker, but with a significant reduction in smoking load (2 cigarettes per day).

## Discussion

Coronary vasospasm can present as stable angina, sudden cardiac death, or acute coronary syndrome, affecting patients with or without atherosclerosis.[3] Its prevalence varies by race and region, and it is a leading cause of ischemic heart disease in patients with non-obstructive coronary lesions. It is more common in men aged 40 to 70 years, with the highest rates in Japanese (24.3%), followed by Taiwanese (19.3%) and Caucasian (7.5%) populations.[4,5] Clinical manifestations range from asymptomatic angina to severe cardiac events, often occurring at rest, especially at night or early morning.[3]

The patient matched the high-prevalence profile for coronary vasospasm, considering his Asian ethnicity, male sex, and age (58 years). Smoking, a major risk factor for coronary artery spasm in men[6] and the Japanese population,[7] was also present. After LAD stent implantation, his clinical picture suggested a new acute coronary syndrome, initially

## Keywords

Infarction; Intracranial Vasospasm; Myocardial Infarction.

**Mailing Address: Mateus Amorim Aboboreira •**
Universidade Estadual de Maringá, Departamento de Medicina. Avenue Mandacaru, 1590, University Hospital, Block S05, Room 10. Postal Code: 87020-900, Maringá, PR – Brazil
E-mail: mateusamorim995@gmail.com
Manuscript received March 18, 2025; revised manuscript June 12, 2025; accepted July 14, 2025.
Editor responsible for the review: Solange Nogueira

**DOI:** https://doi.org/10.36660/ijcs.20250046

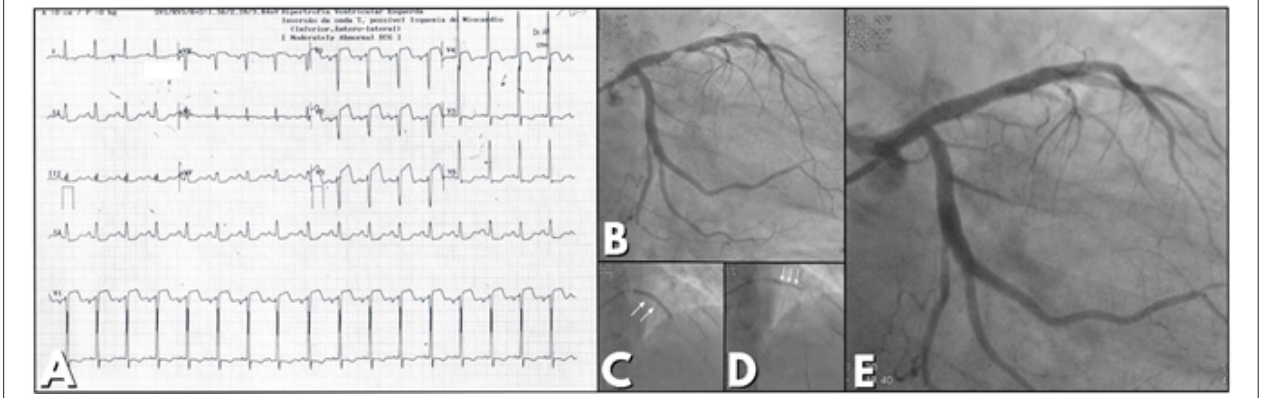



**Figure 1 –** *A) Pre-procedure electrocardiogram; B) Pre-procedure coronary angiography; C) Stent implantation in the left anterior descending artery; D) Balloon dilation in the first diagonal branch; E) Post-procedure coronary angiography.*

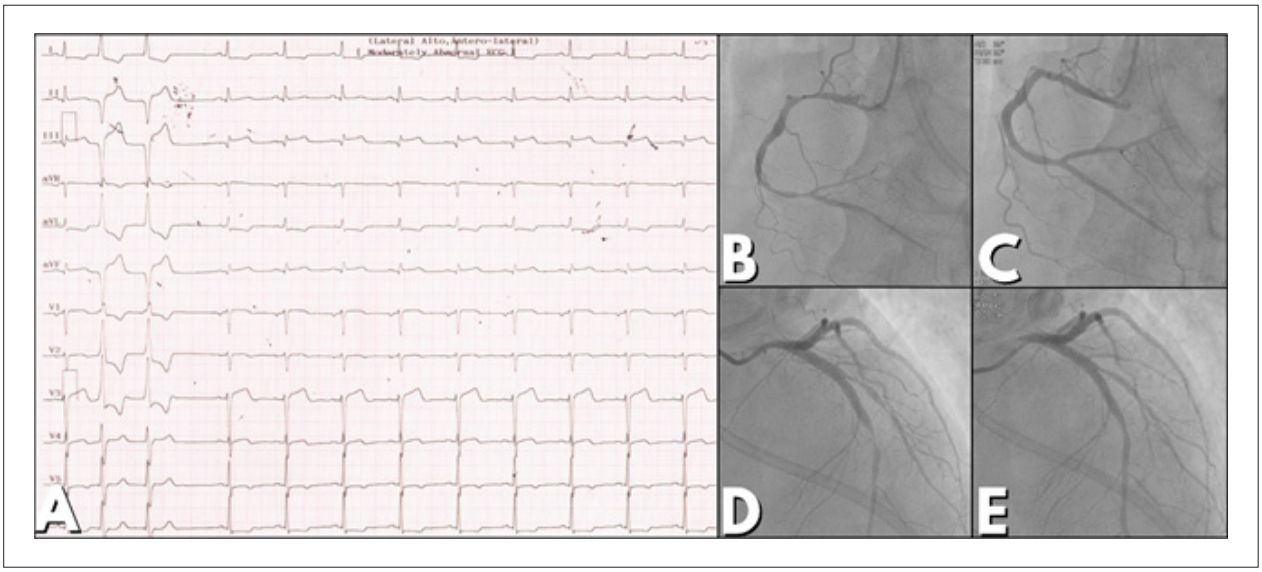



**Figure 2 –** *A) New electrocardiogram indicating ST elevation in the inferior wall; B) Vasospasm of the right coronary artery; C) Reversal of right coronary artery stenosis with nitroglycerin; D) Vasospasm of the left anterior descending artery; E) Reversal of vasospasm in the left anterior descending artery.*

presenting as classic angina. Despite the diagnostic challenge, the similarity between vasospasm and acute myocardial infarction did not hinder appropriate management, which includes symptom evaluation, ischemia confirmation, and catheterization to demonstrate arterial spasm,[8] which in this case occurred in both the coronary territory and the cerebral circulation. Catheterization was performed due to electrocardiogram changes and the higher prevalence of acute myocardial infarction in atherosclerotic patients compared to vasospasm.

The pathophysiology of vasospasm is not clearly understood, and it involves multiple factors related to the autonomic nervous system, endothelial dysfunction, inflammation, oxidative stress, smooth muscle hyper-reactivity, atherosclerosis, thrombosis, and genetic predisposition. The precipitating factors for vasospasm include chronic mental stress, mild exercise (in the early morning hours), cold exposure, hyperventilation, magnesium deficiency, Valsalva maneuver, and consumption of alcohol, cocaine, sympathomimetic and parasympathomimetic agents, beta blockers, anticholinesterases, and amphetamine derivatives.[2] This patient was in continuous use of a nicotine patch for smoking cessation. Although nicotine patches are not directly mentioned among the precipitating factors of coronary artery spasm, they may have an effect similar to some of these factors, especially concerning the activation of the sympathetic system. However, some meta-analyses that investigated the use of nicotine patches and other medications, such as bupropion, indicated that nicotine patches do not appear to significantly increase the risk of severe cardiovascular events or angina.[9] The

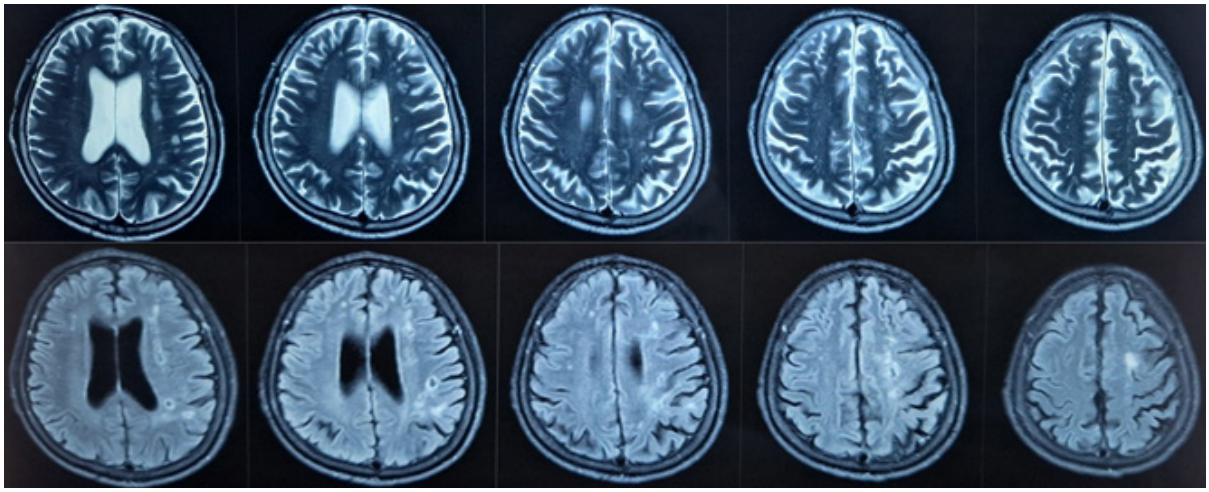


**Figure 3 –** *Cerebral magnetic resonance imaging with ischemia suggesting embolic lesions.*

evidence found was not enough to suggest that these medications increase the risk of severe cardiovascular complications.[10] It is worth noting that the low incidence of such events may have limited the ability to detect statistically significant differences in the most severe outcomes of these studies.

It is still worth mentioning Kounis syndrome as a diagnostic possibility, which is poorly known in the medical field and has few reports in the literature. It is characterized by the simultaneous occurrence of acute coronary syndrome and a hypersensitivity reaction.[11,12] The diagnosis was ruled out due to the absence of allergic symptoms, such as urticaria, angioedema, or bronchospasm.

The initial treatment of coronary vasospasm involves nitrates, such as nitroglycerin, for immediate spasm relief and blood flow restoration. Long-term management prioritizes calcium channel blockers as first-line therapy, with long-acting nitrates added in refractory cases. Additional measures include smoking cessation, stress control, magnesium correction, and avoiding triggering agents, such as beta blockers and sympathomimetic agents. Preventing modifiable risk factors, especially smoking, is crucial to reducing recurrence and improving prognosis.[2] The patient in this case remains a smoker, but he has significantly reduced daily cigarette consumption.

## Conclusion

This case underscores the complexity of coronary vasospasm management, highlighting its multifactorial etiology and key risk factors. The recurrent acute coronary events and transient neurological symptoms emphasize the need for a thorough diagnostic approach, including catheterization. While the role of nicotine patches remains uncertain, it is crucial to investigate potential triggers in high-risk patients. Early treatment with vasodilators and calcium channel blockers proved effective, ensuring a favorable outcome.

## Acknowledgment

We thank Dr. Miyoko Massago for her support, guidance, and valuable contribution to obtaining approval from the Research Ethics Committee, which were essential to the completion of this work.

## Author Contributions

Conception and design of the research: Aboboreira MA, Maia JP, Borges VB, Ueda FH, Franchetti M. Acquisition of data: Aboboreira MA. Analysis and interpretation of the data: Aboboreira MA, Maia JP, Borges VB, Ueda FH, Franchetti M. Writing of the manuscript: Aboboreira MA, Maia JP, Borges VB, Ueda FH, Franchetti M. Critical revision of the manuscript for intellectual content: Aboboreira MA, Borges VB.

### Potential Conflict of Interest

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

### Sources of Funding

There were no external funding sources for this study.

### 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 Marimed Serviços Médicos S/A under the protocol number 8.717.801. 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 and Other Materials

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

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