Texas Heart Institute Journal

Case Reports

Spontaneous Multivessel Coronary Spasm During Diagnostic Coronary Angiography

Ramses Ramirez Damera, MD[1,2]; Muhammad Ijlal Khan, MD[1,2]; Volodymyr Oliynyk, DO[1,2]; Aamir Javaid, MD[3]

[1] College of Medicine, University of Central Florida; Orlando, Florida
[2] Department of Internal Medicine, HCA Healthcare Greater Orlando; Orlando, Florida
[3] Department of Cardiovascular Medicine, Osceola Regional Medical Center, Kissimmee, Florida

Acute vasospastic angina, formerly known as Prinzmetal angina, is characterized by transient electrocardiographic changes that are not related to exertion. Its atypical presentation makes it difficult to establish the diagnosis, so it is probably underrecognized and therefore mismanaged. We treated a 49-year-old woman who presented with a 2-day history of chest pain associated with palpitations. Abnormal radionuclide stress test results prompted diagnostic coronary angiography, during which the patient reported chest pain and became hemodynamically unstable. Active coronary vasospasm at multiple sites was treated with intracoronary nitroglycerin and nicardipine, leading to immediate recovery.

Our case highlights the importance of accurate, timely diagnosis of vasospastic angina, and of early recognition and management of spontaneous coronary spasm during angiography. ***(Tex Heart Inst J 2022;49(2):e207357)***

Citation:
Ramirez Damera R, Khan MI, Oliynyk V, Javaid A. Spontaneous multivessel coronary spasm during diagnostic coronary angiography. Tex Heart Inst J 2022;49(2):e207357. doi: 10.14503/THIJ-20-7357

Key words:
Angina pectoris, variant/etiology; coronary disease/therapy; coronary vasospasm/complications/diagnosis/drug therapy/etiology; coronary vessels/pathology; risk factors; treatment outcome; vasoconstriction/physiology

Corresponding author:
Ramses Ramirez Damera, MD, Department of Internal Medicine, Willis-Knighton Health System, 2400 Hospital Dr., Suite 130, Bossier City, LA 71111

E-mail:
ramsesramirez30@gmail.com

Vasospastic angina (VSA), formerly called Prinzmetal angina, is characterized by transient electrocardiographic (ECG) changes unrelated to exertion.[1] Its exact pathophysiology is unknown; however, a higher incidence has been observed within certain patient populations, such as active smokers.[2] In some cases, VSA can lead to syncope, dysrhythmias, acute coronary syndromes, and sudden cardiac arrest. Diagnostic difficulty suggests that VSA is underrecognized and therefore mismanaged. Timely diagnosis is essential for guiding pharmacologic management and preventing unnecessary interventions such as percutaneous coronary stenting.[3] The impact of VSA cannot be underestimated, especially in view of a report in which left main coronary artery (LMCA) obstruction of less than 25% was described in 20% of patients who underwent coronary artery bypass grafting for presumed LMCA disease.[4] That finding suggests that coronary artery vasospasm went unrecognized when those patients underwent diagnostic angiography. We present the case of a patient in whom VSA spontaneously occurred in multiple vessels during diagnostic coronary angiography.

Case Report

A 49-year-old woman presented with a 2-day history of chest pain associated with palpitations. The pain was intermittent, brief, not associated with exertion or other symptoms, did not radiate, and resolved spontaneously. The patient reported no similar past episodes. Her medical history included active smoking, Graves disease, and gastroesophageal reflux disease. Physical examination revealed a body mass index of 26.1, vital signs within normal limits, and no acute findings.

Laboratory test results included the following: thyroid-stimulating hormone, 1.24 μIU/mL; brain natriuretic peptide, 463 pg/mL; and cardiac troponin I <0.02 ng/mL that was zero when retested. An ECG showed nonspecific T-wave abnormalities in the anterior leads (Fig. 1). An echocardiogram showed nothing abnormal. A radionuclide stress test, performed because of the patient's symptoms and risk factors, revealed evidence of reversible ischemia in the left ventricular anterior wall and part of the inferior wall (Fig. 2).

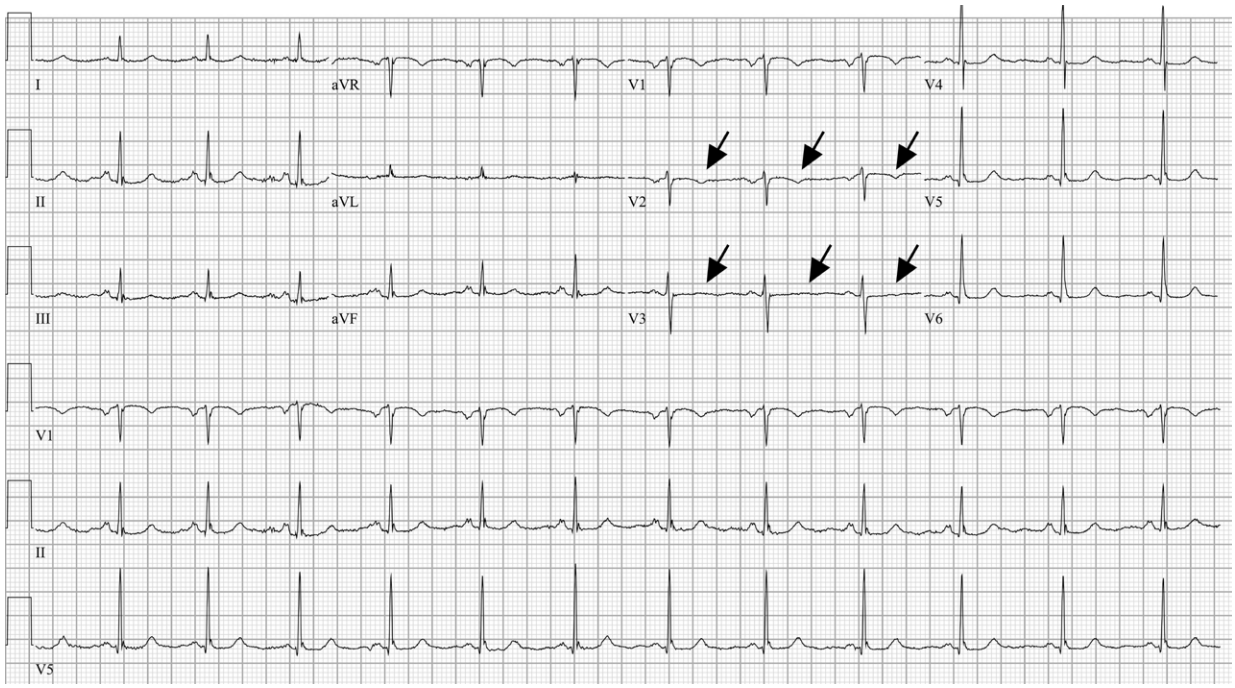

Fig. 1 *Electrocardiogram shows nonspecific T-wave abnormalities in the anterior leads (arrows).*

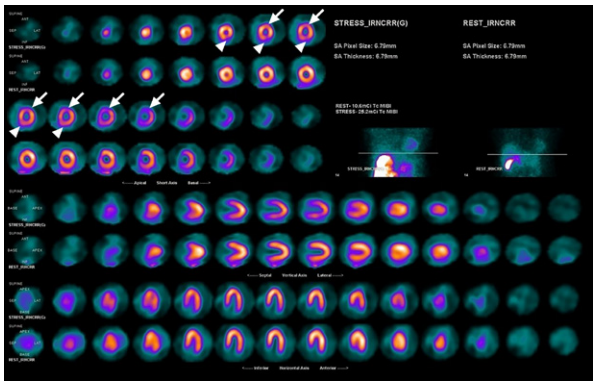

Fig. 2 *Radionuclide stress test results show evidence of reversible ischemia throughout the left ventricular anterior wall (arrows) and part of the inferior wall (arrowheads).*

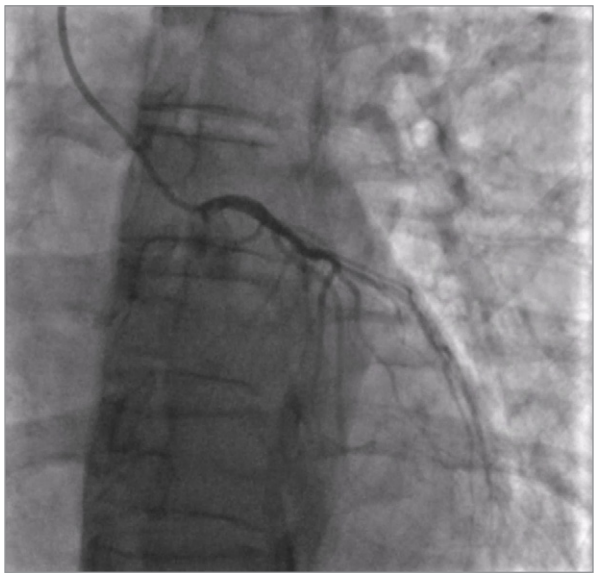

Fig. 3 *Initial angiogram shows normal flow through the left anterior descending and left circumflex coronary arteries.*

Supplemental motion image is available for Figure 3.

Coronary angiograms initially showed no substantially obstructed flow in the LMCA and its branches (Fig. 3). However, as we evaluated the left anterior descending coronary artery (LAD), the patient reported chest pain, at which time ST-segment elevations abruptly developed in the anterolateral leads, and marked sinus bradycardia led to hemodynamic compromise. An angiogram showed total occlusion of the mid LAD (Fig. 4A) and subtotal occlusion of the left circumflex coronary artery (Fig. 4B). A 100-μg bolus of intracoronary nitroglycerin and 200 μg of intracoronary nicardipine brought immediate symptomatic improvement. The patient's hemodynamic status resolved, and angiographic patency was confirmed by Thrombolysis In Myocardial Infarction grade 3 flow to the affected areas (Fig. 4C). Proximal coronary spasm in the right coronary artery resolved without intervention (Fig. 5).

After the procedure, our goal was to prevent further VSA episodes. Before the patient's discharge from the hospital, she was counseled on smoking cessation and avoiding previously known triggers. We prescribed isosorbide mononitrate (30 mg), amlodipine (5 mg), and atorvastatin (40 mg). The patient reported no symptoms at her next 3 clinical visits.

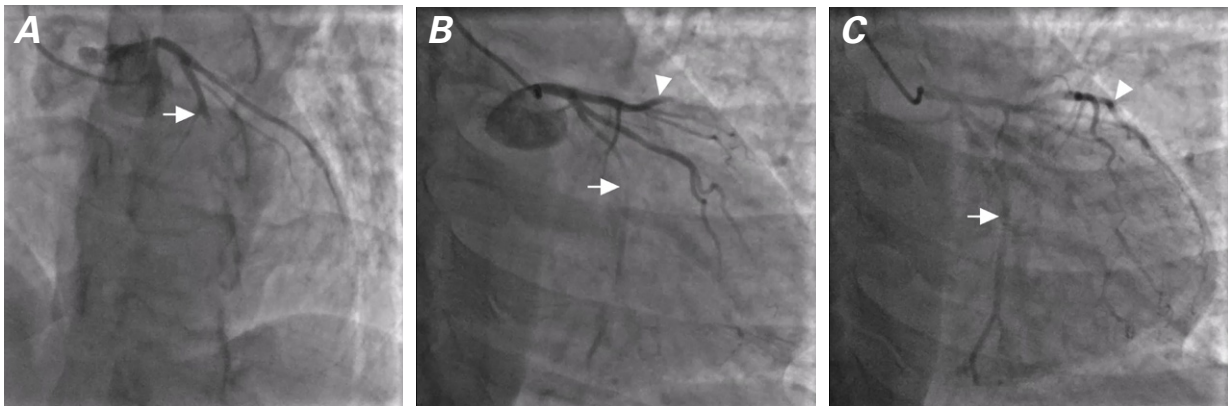

Fig. 4 *Coronary angiograms show* ***A****) occlusion of the mid left anterior descending coronary artery (LAD) by severe spasm (arrow),* ***B****) subtotal occlusion of the left circumflex coronary artery (LCx) (arrow) and improving LAD flow (arrowhead), and* ***C****) Thrombolysis In Myocardial Infarction grade 3 flow to the LCx (arrow) and LAD (arrowhead) after vasodilator administration.*

Supplemental motion images are available for Figures 4A, 4B, and 4C.

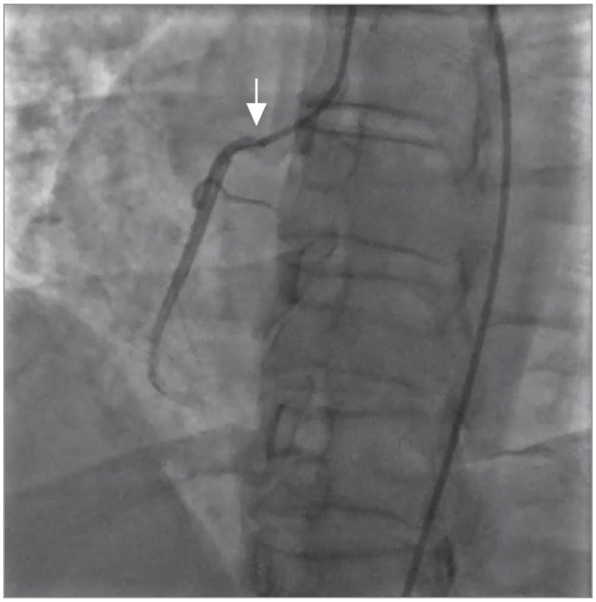

Fig. 5 *Coronary angiogram shows proximal right coronary artery spasm (arrow); and, in accompanying motion image, immediate spontaneous resolution (blue arrow).*

Supplemental motion image is available for Figure 5.

Discussion

Vasospastic angina is defined as coronary artery vasospasm reactive to nitrates, in association with transient ischemic ST-segment changes on ECG and >90% transient coronary artery constriction on angiograms. The angina typically occurs after midnight in clusters of 2 to 3 episodes; each cluster lasts 30 to 60 min. The angina occurs spontaneously at rest or during routine daily activities, but it is not triggered by exertion. It occurs in 3.5 to 9.6 of 10,000 people, with the highest prevalence reported in Japan.[5,6]

Smoking is one of the strongest risk factors for VSA.[7] Glucose intolerance and dyslipidemia, also more prevalent in the at-risk population, may result from endothelial damage consequent to the oxidative stress associated with these metabolic derangements.[8,9] The Japanese Circulation Society considers it a class I indication to educate patients on lifestyle modification and smoking cessation, as was done in the hospital for our patient.[10] We also think that her well-controlled Graves disease increased her risk for coronary vasospasm. The relationship between Graves thyrotoxicosis and VSA has been well described, and some authors suggest that this association may occur even in patients who have euthyroid Graves disease.[11,12]

In 2017, the Coronary Vasomotion Disorders International Study Group published standardized diagnostic criteria for VSA, involving 3 considerations: classic clinical manifestations of VSA, documented myocardial ischemia during spontaneous episodes, and coronary artery spasm on angiograms.[5] Coronary vasospasm typically occurs in one vessel at a time, which enables the clinician to locate the area with use of noninvasive tests. Conversely, our patient had total and subtotal occlusions of multiple vessels simultaneously, which led to hemodynamic instability and almost to cardiac arrest. This multivessel presentation is extremely rare.

Currently, the European Society of Cardiology recommends prescribing long-acting nitrate agents plus calcium channel blockers in patients who remain symptomatic even after therapy with first-line agents.[13] We prescribed both to our patient, because the benefit of preventing further catastrophic spasms distinctly outweighed the small risk associated with taking low-dose nitrates. Our adding statin therapy conformed with class IIb recommendations from the 2013 Japanese Circulation Society Guidelines for VSA, and with other reports of the benefit of statins.[10,14,15]

Conclusion

Our case illustrates how the accurate and timely diagnosis of VSA guides rapidly effective therapy and helps to avoid invasive procedures such as percutaneous coronary artery stenting or surgical revascularization.

Published: 4 April 2022

Funding/support: This research was supported in whole or in part by HCA Healthcare, an HCA Healthcare affiliated entity, or both. The views expressed are those of the authors and do not necessarily represent the official views of HCA Healthcare or its affiliated entities.

References

1. Prinzmetal M, Kennamer R, Merliss R, Wada T, Bor N. Angina pectoris. I. A variant form of angina pectoris; preliminary report. Am J Med 1959;27:375-88.
2. Takaoka K, Yoshimura M, Ogawa H, Kugiyama K, Nakayama M, Shimasaki Y, et al. Comparison of the risk factors for coronary artery spasm with those for organic stenosis in a Japanese population: role of cigarette smoking. Int J Cardiol 2000;72(2):121-6.
3. Vishnevsky A, Fischman DL, Savage MP. Intracoronary nitroglycerin: recognizing coronary spasm first and foremost to avoid unnecessary coronary stents. Expert Rev Cardiovasc Ther 2017;15(10):727-8.
4. Ilia R, Shimony A, Cafri C, Weinstein JM. Angiographic characteristics of catheter-induced spasm of the left main coronary artery. Am J Cardiol 2016;117(4):571-3.
5. Beltrame JF, Crea F, Kaski JC, Ogawa H, Ong P, Sechtem U, et al. International standardization of diagnostic criteria for vasospastic angina. Eur Heart J 2017;38(33):2565-8.
6. Kawano H, Motoyama T, Yasue H, Hirai N, Waly HM, Kugiyama K, Ogawa H. Endothelial function fluctuates with diurnal variation in the frequency of ischemic episodes in patients with variant angina. J Am Coll Cardiol 2002;40(2):266-70.
7. Nobuyoshi M, Abe M, Nosaka H, Kimura T, Yokoi H, Hamasaki N, et al. Statistical analysis of clinical risk factors for coronary artery spasm: identification of the most important determinant. Am Heart J 1992;124(1):32-8.
8. Shinozaki K, Suzuki M, Ikebuchi M, Takaki H, Hara Y, Tsushima M, Harano Y. Insulin resistance associated with compensatory hyperinsulinemia as an independent risk factor for vasospastic angina. Circulation 1995;92(7):1749-57.
9. Suzuki M, Nishizaki M, Arita M, Kakuta T, Numano F. Impaired glucose tolerance with late hypersecretion of insulin during oral glucose tolerance test in patients with vasospastic angina. J Am Coll Cardiol 1996;27(6):1458-63.
10. JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (coronary spastic angina) (JCS 2013). Circ J 2014;78(11):2779-801.
11. Goldstein JA. Coronary artery spasm and thyrotoxicosis: the best index is that of suspicion. Coron Artery Dis 2014;25(2):96-7.
12. DeTommasi A, Rogge S, Houghton JL. Refractory vasomotor angina in subclinical hyperthyroidism demonstrating focal and segmental coronary vasoconstriction. J Invasive Cardiol 2003;15(5):289-92.
13. Roffi M, Patrono C, Collet JP, Mueller C, Valgimigli M, Andreotti F, et al. 2015 ESC Guidelines for the management of acute coronary syndromes in patients presenting without persistent ST-segment elevation: Task Force for the Management of Acute Coronary Syndromes in Patients Presenting without Persistent ST-Segment Elevation of the European Society of Cardiology (ESC). Eur Heart J 2016;37(3):267-315.
14. Yasue H, Mizuno Y, Harada E, Itoh T, Nakagawa H, Nakayama M, et al. Effects of a 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitor, fluvastatin, on coronary spasm after withdrawal of calcium-channel blockers. J Am Coll Cardiol 2008;51(18):1742-8.
15. Ishii M, Kaikita K, Sato K, Yamanaga K, Miyazaki T, Akasaka T, et al. Impact of statin therapy on clinical outcome in patients with coronary spasm. J Am Heart Assoc 2016;5(5):e003426.