

Vasospastic Angina Presenting With Syncope and Chest Pain: A Case Report and Brief Literature Review

By Shahbaz A. Malik, MD; Thomas T. Porter, MD;
Gregory Pavlides, MD, PhD; and Yiannis Chatzizisis, MD, PhD

Abstract

A 65-year-old male presented to the hospital with chest pain associated with recurrent syncope. He had a history of coronary artery disease and a long-standing history of smoking. While he was hospitalized, he had an episode of chest pain during which he was found to have transient ST segment elevation in the inferior leads. He was also noted to have a brief cardiac tachyarrhythmia. Coronary arteriography revealed vasospasm of the left anterior descending artery and right coronary artery, which were relieved to a significant extent after administration of intracoronary nitroglycerin. Subsequent angiograms and fractional flow reserve studies, demonstrated underlying non-obstructive coronary artery disease at the sites of spasm. No percutaneous coronary intervention was pursued. The patient was started on a calcium channel blocker on dismissal from the hospital. Upon follow up several months later, he remained free of symptoms that brought him to the hospital.

Introduction

Prinzmetal et al.¹ put forward a clinical syndrome of angina occurring at rest accompanied by ST segment elevation. Symptoms and electrocardiogram (EKG) changes responded quickly to sublingual nitrates. The condition was referred to as variant angina. The term now in use to describe this clinical entity is vasospastic angina. This is thought to occur from spasm of the smooth muscle layer of the coronary arterial wall. Here we present such a case and discuss the relevant literature on this elusive condition.

Case Presentation

A 65-year-old male presented to the hospital with complaints of chest pain that woke him up from sleep. The pain was described as a squeezing sensation across his chest, radiating to his left arm and into his neck. He had brief loss of consciousness while experiencing this pain. He was very active at baseline, with his job requiring heavy lifting and experienced no anginal symptoms with this. He had recurrent episodes of chest pain and reported three episodes of syncope over the past year. He was also hospitalized one month prior to his current presentation, after losing consciousness at work during an episode of chest pain. This resulted in a fall, in which he struck his

head. Workup at that time included a 12-lead EKG, which showed a normal sinus rhythm. A transthoracic echocardiogram was done as well, which revealed preserved left ventricular ejection fraction, no regional wall motion abnormalities and presence of a patent foramen ovale. Computed tomography of the chest with contrast was unremarkable for any acute pathology including pulmonary embolism. Computed tomography of the head did show a small left tentorial subdural hematoma. During that hospital stay, one month before the current one described in our case, the patient was evaluated by neurosurgery and no interventions were felt to be warranted. The patient was eventually diagnosed with syncope. He was scheduled to follow up with his cardiologist, at a referral facility where he received most of his outpatient care, and was subsequently discharged home.

The patient had a history of coronary artery disease (CAD) for which he underwent percutaneous coronary intervention at an outside facility, with a drug-eluting stent implanted in the left anterior descending artery, one year ago. He had been on aspirin and clopidogrel thereafter. His outpatient cardiologist discontinued clopidogrel six months ago, and the patient himself stopped aspirin a

few weeks prior to his current presentation. He was not on any other medications at home. The patient had a long-standing history of smoking but denied any recreational drug use.

Physical examination of the patient in the emergency department revealed a blood pressure of 113/74 mmHg and pulse rate of 77 beats per minute. Patient was afebrile and saturating at 98 percent on room air. No adventitious breath sounds were heard on lung auscultation. Auscultation of the precordium revealed a regular rhythm,

normal rate, normal S1 and S2 without any murmurs. Pulses were palpable and symmetrical in bilateral upper and lower extremities, no peripheral edema was noted and cranial nerves were without abnormalities. A 12-lead EKG in the emergency department revealed sinus rhythm, with normal intervals and no ST segment changes. The patient was admitted to the hospital for further management. All laboratory values were within normal limits except for a mild elevation in troponin, which later on repeat testing returned to within normal limits as well.

Figure 1. Conventional 12 lead EKG during an episode of chest pain, showing ST segment elevations in the inferior leads.

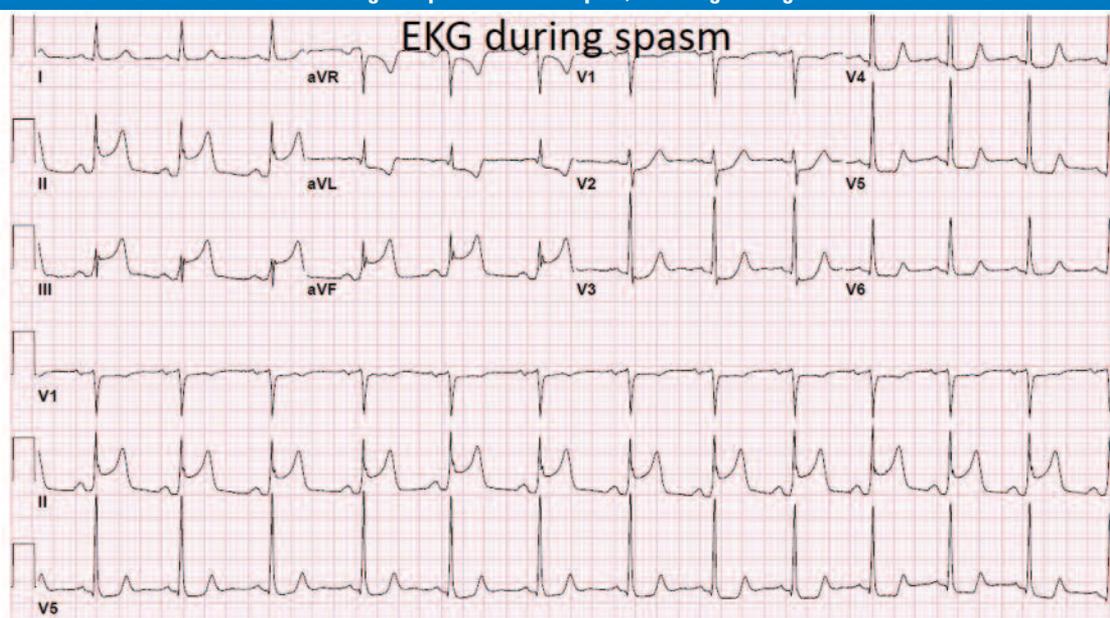


Figure 2. Follow up 12 lead EKG showing improvement of ST segment elevations after resolution of chest pain.

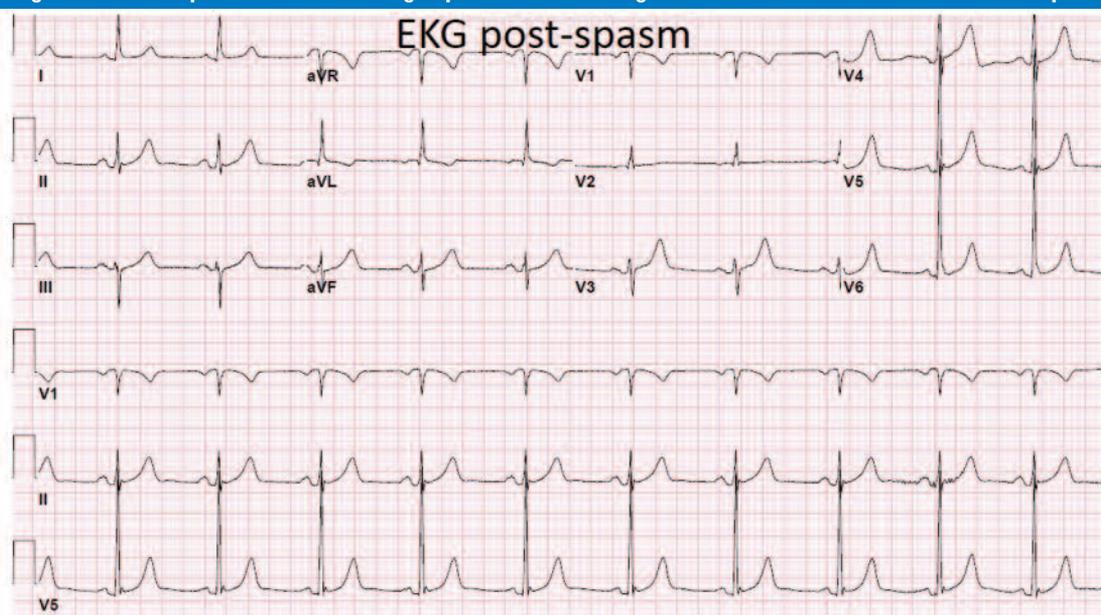


Figure 3. Left panel shows severe spasm seen in the right coronary artery at the mid vessel, prior to receiving intracoronary nitroglycerin. Right panel shows release of spasm in the right coronary artery after administration of 200 micrograms of intracoronary nitroglycerin.

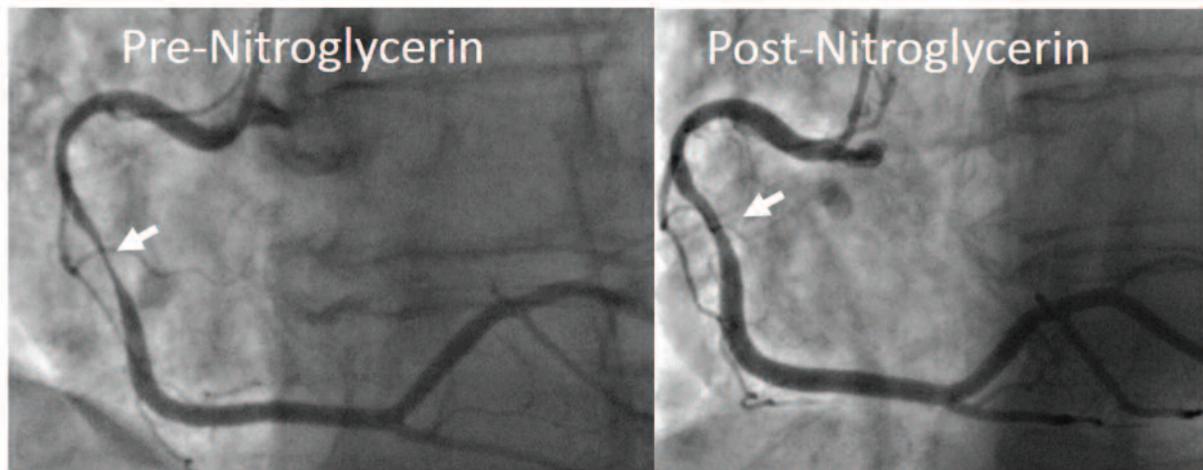
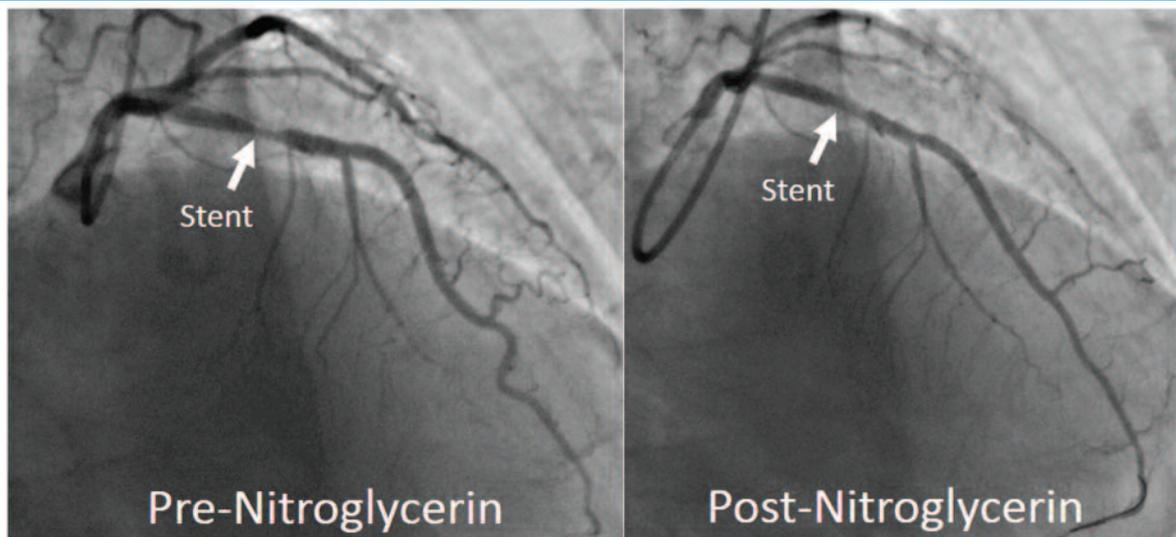


Figure 4. Left panel shows in-stent restenosis in the left anterior descending artery. Right panel shows improvement in stenosis in the left anterior descending artery, at the site of the previously placed stent, after administration of 200 micrograms of intracoronary nitroglycerin.



During this hospital stay, the patient developed recurrent chest pain, similar in character to the initial description. The pain would wax and wane. Stat EKG during an episode of chest pain showed concave upward ST segment elevation in inferior leads with reciprocal ST segment depression in precordial leads (Figure 1). Right-sided EKG revealed no ST segment elevations in right-sided leads. Interestingly when a repeat conventional 12-lead EKG was done a few minutes later, while the patient was free of chest pain, near complete resolution of ST segment elevations were observed (Figure 2). Further evaluation of telemetry strips from this time showed a regular, wide

complex, monomorphic tachycardia, consistent with ventricular tachycardia. This sustained for approximately 60 seconds, with spontaneous termination. No further arrhythmias were noted during his hospital stay. Transthoracic echocardiogram was performed and revealed preserved left ventricular ejection fraction with mild hypokinesis of basal to mid inferior walls. The right ventricle appeared normal in function and no pericardial abnormalities were seen.

The patient was subsequently taken to the cardiac catheterization laboratory for coronary angiography. This

revealed a right dominant coronary system. The right coronary artery (RCA) showed 80 percent stenosis in the mid segment (Figure 3), which was improved to 60 percent after administration of 200 micrograms (μg) of intracoronary (IC) nitroglycerin (Figure 3). There was no angiographic evidence of CAD in the left main coronary artery and left circumflex artery. There was 80 percent in-stent restenosis in the proximal left anterior descending artery (LAD) (Figure 4), which was also improved to 50 percent after administration of 200 μg of IC nitroglycerin (Figure 4). The LAD stenosis was further interrogated by intravascular ultrasound (IVUS) (Figure 5) and fractional flow reserve (FFR) (Figure 6). It was found to be non-

Figure 5. Intravascular ultrasound images of the left anterior descending artery, proximal to, inside, and distal to the intracoronary stent.

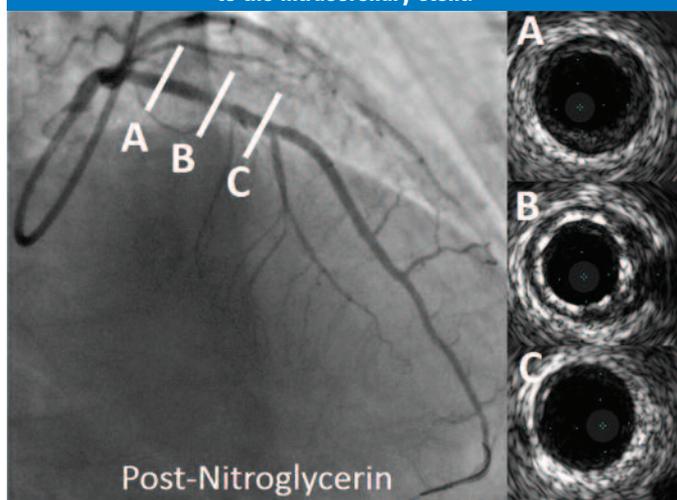
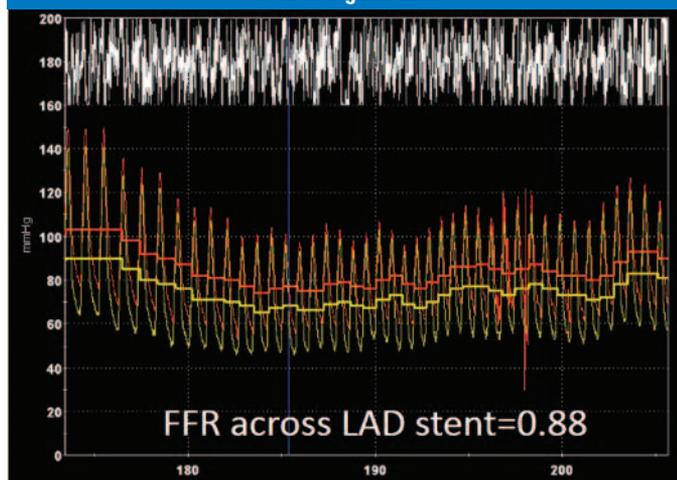


Figure 6. Interrogation of the left anterior descending artery with the help of fractional flow reserve which was found to be non-significant.



significant (minimal luminal area of 4.8 mm^2 by IVUS and FFR value at 0.88). Overall these findings were suggestive of coronary artery spasm in RCA and LAD in addition to presence of moderately stenotic atherosclerotic plaque, with spasm improving after administration of nitrates.

The patient was discharged on an oral calcium channel blocker (diltiazem extended release, 90 mg once daily) with the intention of up-titrating the dose as he tolerated the drug, during subsequent follow up visits. He was instructed to avoid beta-blockers given presence of coronary vasospasm and was strongly advised lifestyle modifications, including smoking cessation. Upon follow up approximately six months after dismissal from the hospital, the patient had experienced no further episodes of chest pain or loss of consciousness. He reported taking all of his medications, was tolerating them well, and did in fact attribute his lack of symptoms to them.

Discussion

In this report we describe a case of a male presenting with recurrent chest pain and loss of consciousness. An episode of chest pain occurred while he was hospitalized, during which transient ST segment elevations on EKG, and arrhythmia on telemetry monitoring were captured. He was ultimately found to have coronary artery vasospasm in the LAD and RCA, coupled with coronary artery atherosclerosis. There was improvement in spasm of both aforementioned arteries upon administration of IC nitroglycerin. Improvement of the LAD spasm, at the location of the prior stent, suggested that the stent was likely apposed against the spastic coronary artery wall.

Spasm of the smooth muscle layer of the coronary arterial wall causes vasospastic angina. Vascular smooth muscle hyperactivity is thought to play a pivotal role in the development of spasm.² Endothelial dysfunction and lack of endothelial nitric oxide activity also plays an important role. In coronary arteries with atherosclerosis, endothelial dysfunction results in vasoconstrictor responses that can be focal.^{3,4} Cigarette smoking has been reported to be a crucial risk factor for coronary vasospasm.⁵ Use of multiple drugs including ephedrine based products, cocaine and amphetamines are also reported to be potential triggers.⁶ Exercise does not seem to have a provocative effect in terms of vasospasm.^{7,8}

Our patient also reported syncope associated with his chest pain. Ventricular arrhythmias occurring in patients with vasospastic angina was first described by Prinmetal

et al.¹ Vasospastic angina has been reported to manifest as pre-syncope or syncope, related to arrhythmias.⁹ Heart block has also been reported in the literature.¹⁰ Given findings of VT on telemetry monitoring during an episode of chest pain, without any recorded episodes of bradycardia or heart block (secondary to RCA spasm) throughout hospitalization, our patient's syncope seemed most likely related to the cardiac tachyarrhythmia. A cryptogenic stroke involving the patient's patent foramen ovale (PFO) was thought to be unlikely given lack of any neurological deficits.

The diagnosis of vasospastic angina is supported by anginal type chest pain occurring at rest with transient ST segment elevation on EKG during an episode, with lack of high-grade coronary artery obstruction on coronary angiography. A 12-lead EKG is recommended in all patients.³ Ambulatory EKG monitoring can aid in detection of transient ST segment changes in asymptomatic patients.¹¹ Coronary arteriography with acetylcholine provocation can be used to induce coronary vasospasm and thus make the diagnosis.^{3,12} In healthy coronary arteries, acetylcholine induces release of endothelial nitric oxide, which offsets its parasympathetic action on smooth muscle cells, thereby resulting in vasodilation. In contrast, in the setting of dysfunctional endothelium the vasodilatory effect of acetylcholine is diminished and the drug induces vasoconstriction.³

Acute attacks can be managed with sublingual nitroglycerin.³ Refractory spasm may require intracoronary injection of nitrates.³ In terms of prevention of coronary spasm attacks, calcium channel blockers and long-acting nitrates have been reported to be effective. Potency of nitrates is reduced by nitrate tolerance.³ Lifestyle modification, especially smoking cessation, is recommended.¹³ The exact management of aborted cardiac arrest is unclear for patients with vasospastic angina, as is the role of implantable cardioverter defibrillator placement.¹² Ahn et al.¹⁴ reported a nonsignificant trend of a lower rate of cardiac death in patients with than those without implantable cardioverter defibrillators. Long-term prognosis of patients with vasospastic angina is effected by underlying CAD — the greater the extent of obstructive CAD, the less favorable the prognosis.¹⁵

In conclusion, our patient presented with syncope and chest pain and was found to have vasospasm in two coronary arteries, which was relieved by nitroglycerin. Interestingly one area of spasm was at the site of a

previously placed intracoronary stent, which also responded to the aforementioned medication. Thus, coronary artery spasm is a valid consideration in all patients presenting with chest pain and EKG changes suggestive of ischemia.

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About the Authors:

Shahbaz A. Malik, MD, PGY-IV, Cardiology Fellow, Department of Internal Medicine, Division of Cardiology, University of Nebraska Medical Center, Omaha, Nebraska.

Thomas T. Porter, MD, Professor, Internal Medicine, Theodore F. Hubbard Distinguished Chair of Cardiology, Department of Internal Medicine, Division of Cardiology, University of Nebraska Medical Center, Omaha, Nebraska.

Gregory Pavlides, MD, PhD, Professor, Internal Medicine, Vincent Miscia Chair of Interventional Cardiology, Department of Internal Medicine, Division of Cardiology, University of Nebraska Medical Center, Omaha, Nebraska.

Yiannis Chatzizisis, MD, PhD, Associate Professor, Internal Medicine, Department of Internal Medicine, Division of Cardiology, University of Nebraska Medical Center, Omaha, Nebraska.

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