Coronary vasospasm-induced syncope with dynamic changes of regional wall motion abnormalities confirmed real-time: a case report

Hak Seung Lee, Han-Mo Yang*, Bon-Kwon Koo, and Hyo-Soo Kim

Department of Internal Medicine and Cardiovascular Center, Seoul National University Hospital, 101 Daehang-ro, Chongno-gu, Seoul110-744, Republic of Korea

Received 24 February 2020; first decision 2 April 2020; accepted 30 June 2020; online publish-ahead-of-print 8 November 2020

Background
Coronary vasospasm is primarily characterized by transient and reversible vasoconstriction causing myocardial ischaemia and can manifest with various clinical features, including syncope.

Case summary
A 50-year-old man presented with recurrent episodes of syncope for 3 days. The last syncope history occurred during an early morning walk, accompanied by dizziness and loss of consciousness. There was no clear history of chest pain at the time. He smoked one pack of cigarettes daily and frequently consume alcohol. Approximately 3 h after admission, echocardiography initially revealed normal systolic function; however, during the examination, the patient suddenly complained of dizziness and regional wall motion abnormalities (RWMA) of the left anterior descending artery (LAD) territory were observed. Both RWMA and dizziness spontaneously improved within a few minutes. Emergency coronary angiography (CAG) was performed to confirm vasospasm. Coronary angiography revealed mild atherosclerosis of proximal LAD. After 3 min, he complained of dizziness and vague chest discomfort, and electrocardiogram revealed ST-segment elevation. We immediately performed angiography of the left coronary artery, and CAG revealed total occlusion of the proximal LAD without any provocation. After administration of intracoronary nitroglycerine, coronary flow was restored completely and ST-segment deviation normalized along with relief in chest discomfort. The patient’s symptoms have not recurred for 3 months while being on calcium channel blocker and long-acting nitrates.

Discussion
Coronary vasospasm can present as transient and dynamic myocardial ischaemia along with angina. Coronary vasospasm should always be considered in the differential diagnosis for syncope.

Keywords
Angina • Coronary vasospasm • Vasospastic angina • Regional wall motion abnormalities • Case report

Learning points
- Coronary vasospasm is a heterogeneous phenomenon that can present with transient total coronary artery occlusion along with angina, and these dynamic changes can be observed on echocardiography, electrocardiography, and invasive coronary angiography.
- Coronary vasospasm should always be considered in differential diagnoses of syncope as one of the causes of cardiac syncope.
- In such cases, symptoms can be effectively controlled through the lifestyle management and medications.
Introduction

Coronary vasospasm is primarily characterized by transient and reversible vasoconstriction of the major epicardial coronary arteries resulting in myocardial ischaemia, which is not associated with exercise or effort. Coronary vasospasm can manifest as several clinical features, including not only variant angina but also effort-induced angina, acute coronary syndrome, syncope, and sudden cardiac death with life-threatening arrhythmias or conduction disorders. We present the case of a patient who developed coronary vasospasm-induced syncope with spontaneous total occlusion of a coronary artery that was confirmed real-time using echocardiography and coronary angiography.

Timeline

| Emergency department visit and admission | 3-day history of four episodes of syncope |
| Echocardiography performed 3 h after admission | Sudden-onset dynamic changes in transient regional wall motion abnormalities with dizziness during the exam |
| Emergency coronary angiography performed 4 h after admission | Spontaneous total occlusion of the left anterior descending artery without provocation |
| 3rd day | Hospital discharge |
| 3rd month follow-up | Asymptomatic without further episodes |

Case presentation

A 50-year-old man visited the emergency room with complaints of recurrent episodes of syncope for 3 days. He had four episodes of transient loss of consciousness that lasted for approximately 30 s each accompanied by general weakness. The last syncope history occurred during an early morning walk, accompanied by dizziness and loss of consciousness. Chest pain was not apparent during the initial onset of symptoms. He had no history of hypertension, diabetes, or dyslipidaemia. He smoked one pack of cigarettes daily for 30 years with frequent consumption of alcohol. Cardiac auscultation of the patient revealed normal heart sounds and no murmurs. Brain computed tomography, electrocardiogram (ECG), laboratory findings, including cardiac enzymes, and chest radiography were normal.

Approximately 3 h after admission, transthoracic echocardiography was performed to evaluate the systolic function and investigate structural anomalies. During echocardiographic examination, there were initially no regional wall motion abnormalities (RWMA) though, the patient’s symptoms of pre-syncope recurred and interestingly anteroseptal and apical wall akinesia was observed. However, without any intervention, both wall motion abnormalities and dizziness spontaneously improved within a few minutes (Video 1).

Emergency coronary angiography (CAG) was performed immediately to confirm vasospasm as the cause of dynamic changes in the transient and reversible RWMA and dizziness. Coronary angiography revealed mild atherosclerosis of the proximal left anterior descending artery (LAD) and hypoplastic right coronary artery (Figure 1A). During the examination, he complained of dizziness and vague chest discomfort; simultaneously, ECG revealed ST-segment elevation (V1–V5) and ST-segment depression (leads III and aVF) (Figure 2). We immediately performed angiography of the left coronary artery, and CAG revealed total occlusion of the proximal LAD without any provocation, which occurred within 3 min of the initial angiography (Figure 1B). After administration of intracoronary nitroglycerine, the

Figure 1 (A) Mild stenotic lesion in the proximal left anterior descending artery was observed on the initial coronary angiography. (B) Spontaneous total occlusion of proximal left anterior descending artery was induced without provocation within 3 min of the initial angiography (yellow arrow). (C) After intracoronary administration of nitroglycerine, the coronary flow was restored completely.
coronary flow improved completely, the calibre of the vessel enlarged, and ST-segment deviation normalized along with relief in chest discomfort (Figure 1C, Video 2).

Diltiazem, isosorbide dinitrate, clopidogrel, and atorvastatin were administered for severe spontaneous vasospasm with mild atherosclerosis. Additionally, the patient was educated about the importance of adhering to the regular medications and was strongly advised to abstain from alcohol and smoking. Two days later, the patient was discharged and the symptoms have not recurred for 3 months.

**Discussion**

Vasospastic angina (VSA) is the transient occlusion of the epicardial coronary arteries, which occurs mainly at night or early morning and is not usually associated with increased myocardial oxygen requirement. To the best of our knowledge, this is the first reported case of direct observation of these dynamic and spontaneous changes using various modalities in real-time. The pathophysiology of VSA remains unclear. However, it is understood to be caused by impaired vasodilation secondary to smooth muscle hyperactivity and endothelial, autonomic, and microvascular dysfunctions due to the traditional risk factors of coronary artery disease. Since the episodes of coronary vasospasm are usually temporary, establishing a definite diagnosis of VSA is challenging. However, if VSA is suspected clinically, a provocation test is needed for a precise diagnosis. Fortunately, in this case, we were able to identify an episode of reversible and transient coronary vasospasm without provocation during the examination, which also revealed spontaneous RWMA, ECG changes, and total occlusion of the LAD. The combination of fixed, non-significant atherosclerotic
lesions and vasospastic activity resulted in total occlusion of the proximal LAD. Mild atherosclerotic lesions of the LAD observed even after nitroglycerine injection could be the nidus for coronary vasospasm. In this patient, however, smoking and alcohol could have also acted as triggers.

Depending on the extent and location of the coronary vasospasm, VSA can present with various clinical manifestations, including variant angina, effort-induced angina, acute coronary syndrome, and sudden cardiac death. Among them, the mechanism of syncope in patients with VSA is explained by atrioventricular block, ventricular tachycardia, pump failure due to left ventricular dysfunction, and vagal stimulation. When patients present with syncope with dizziness, as in this patient, it is challenging to suspect VSA based on the initial workup. This patient developed symptoms while undergoing echocardiography, which led to the confirmation of the diagnosis. Particularly, in East Asia, the incidence of coronary vasospasm is relatively high in patients with syncope, and some groups have suggested a diagnostic flow-chart for patients with syncope in Japan and Asia. The latest guidelines for syncope published by the European Society of Cardiology in 2018 recommend ECG monitoring and electrophysiology studies in the initial evaluation of syncope when accompanied with high-risk features; however, they do not address the need to consider VSA in the differential diagnosis. Irrespective of the racial differences, coronary vasospasm should be considered in patients with syncope.

All patients with VSA require lifestyle modifications and drug therapy. The primary treatment for VSA includes both calcium-channel blockers and nitrates, which can effectively prevent myocardial ischaemia. Lifestyle modifications include management of the risk factors of coronary artery disease, such as smoking cessation and alcohol restriction. Additionally, this patient was prescribed clopidogrel and statins due to the total fixed occlusion in LAD. Finally, because vasospasm-induced lethal arrhythmia was not documented, an implantable cardioverter-defibrillator was not considered.

Vasospastic angina with syncope is an uncommon but significant entity that should be carefully investigated. The present case suggests that VSA should be considered in the differential diagnosis of syncope and physicians should be familiar with the various manifestations and clinical presentation of VSA.

Lead author biography

Hak Seung Lee, MD, graduated in Medicine at Seoul National University, had residency training in Internal Medicine and Cardiology in Seoul National University Hospital (SNUH). He is a specialist in Cardiology and Interventional Cardiology. Currently, he works as a clinical fellow at SNUH.

Supplementary material

Supplementary material is available at European Heart Journal - Case Reports online.

Funding

This work was supported by the Korea Health Technology R&D Project ‘Korea Research-Driven Hospital’ (HI14C1277) and ‘National Research Foundation of Korea (NRF)’ (2020R1A2C1011311).

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

References

1. Prinzen et al. M, Kennamer R, Merli R, Wada T, Bor N. Angina pectoris. 1. A variant form of angina pectoris: preliminary report. Am J Med 1959;27:375-388.
2. Song JK. Coronary artery vasospasm. Korean Circ J 2018;48:767-777.
Coronary vasospasm-induced syncope with dynamic changes of RWMA confirmed real-time

3. Maseri A, Pesola A, Marzilli M, Severi S, Parodi O, L’Abbate A et al. Coronary vasospasm in angina pectoris. Lancet 1977;309:713–717.
4. JCS Joint Working Group. Guidelines for diagnosis and treatment of patients with vasospastic angina (Coronary Spastic Angina) (JCS 2013). Circ J 2014;78:2779–2801.
5. Takagi Y, Yasuda S, Tsunoda R, Ogata Y, Seki A, Sumiyoshi T et al.: Japanese Coronary Spasm Association. Clinical characteristics and long-term prognosis of vasospastic angina patients who survived out-of-hospital cardiac arrest: multicenter registry study of the Japanese Coronary Spasm Association. Circ Arrhythm Electrophysiol 2011;4:295–302.
6. Tandon V, Mosebach CM, Kumar M, Joshi S. Refractory vasospastic angina: when typical medications don’t work. Cureus 2019;11:e4134.
7. Mehta PK, Thobani A, Vaccarino V. Coronary artery spasm, coronary reactivity, and their psychological context. Psychosom Med 2019;81:233–236.
8. Beltrame JF, Crea F, Kaski JC, Ogawa H, Ong P, Sechtem U et al.: Coronary Vasomotion Disorders International Study Group (COVADIS). International standardization of diagnostic criteria for vasospastic angina. Eur Heart J 2017;38:2565–2568.
9. Whiting RB, Klein MD, Vander Veer J, Lown B. Variant angina pectoris. N Engl J Med 1970;282:709–712.
10. Igarashi Y, Yamazoe M, Suzuki K, Tamura Y, Matsubara T, Tanabe Y et al. Possible role of coronary artery spasm in unexplained syncpe. Am J Cardiol 1990;65:713–717.
11. Nishizaki M. Life-threatening arrhythmias leading to syncope in patients with vasospastic angina. J Arrhythm 2017;33:553–561.
12. Brignole M, Moya A, de Lange FJ, Dhaenens MC, Elliott PM, Fanciulli A et al.; ESC Scientific Document Group. 2018 ESC Guidelines for the diagnosis and management of syncope. Eur Heart J 2018;39:1883–1948.
13. Lanza GA, Careni G, Crea F. Mechanisms of coronary artery spasm. Circulation 2011;124:1774–1782.
14. Task Force Members; Montalescot G, Sechtem U, Achenbach S, Andreotti F, Budaj AC, Bugiardini A et al. 2013 ESC guidelines on the management of stable coronary artery disease: the Task Force on the management of stable coronary artery disease of the European Society of Cardiology. Eur Heart J 2013;34:2949–3003.
15. Yasue H, Takizawa A, Nagao M, Nishida S, Horie M, Kubota J et al. Long-term prognosis for patients with variant angina and influential factors. Circulation 1988;78:1–9.