



# Left main coronary artery spasm detected by intravascular ultrasound: a case description and literature analysis

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## Introduction

Epicardial coronary artery spasm, an endotype of ischemia with nonobstructive coronary artery (INOCA), is characterized by transient chest symptoms with electrocardiography (ECG) changes but without obstructive coronary artery disease (1). Occasionally, however, epicardial coronary spasm can manifest as persistent chest pain that persists even after a single dose of nitrates. There are a few reports indicating that such a spasm can occasionally occur in the left main coronary artery (LMCA), which without adequate recognition and management, can result in serious consequences such as sudden cardiac death (2,3). The LMCA spasm case we report here is unique in that the patient complained of persistent chest pain and underwent exercise treadmill testing (ETT), coronary computed tomography angiography (CCTA), and coronary angiography (CAG) over 2 consecutive days, all revealing severe stenosis of the left main artery until an intravascular ultrasound (IVUS) confirmed coronary spasm.

## Case presentation

A 50-year-old male with a medical history of hypertension and tobacco use was admitted to hospital complaining of chest pain radiating to the left shoulder, which occurred in the morning with or without exertion and which increased

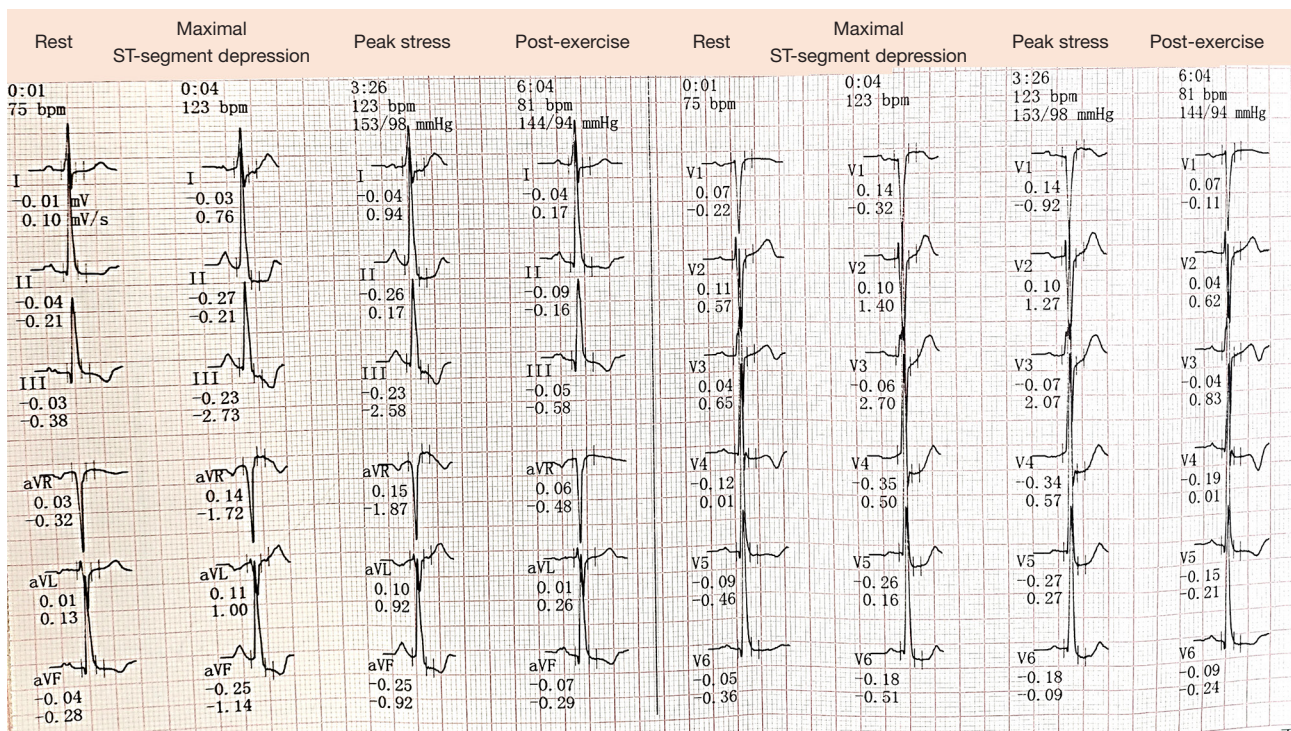
in frequency over the previous 20 days. This patient denied previous conditions, reported no history of palpitation, lower extremity swelling, cough, orthopnea, or paroxysmal nocturnal dyspnea. He had neither cardiac risk factors nor known coronary artery diseases. He received no surgery and had no family history. He denied consuming alcoholic or having any drug dependencies. His vital signs remained stable. He was differentially diagnosed with acute coronary syndrome, pericarditis, myocarditis, pneumonia, and acute pulmonary embolism.

Positive ETT findings were established in this patient. He exercised 3 min 26 s with the standard Bruce protocol to a peak heart of 123 beats/min (72% maximal predicted heart rate) and 5.1 metabolic equivalents and had no chest pain. The test was stopped for ST-segment elevation [ $>1$ -mm horizontal ST-segment elevation in lead augmented voltage right arms (aVR) and V1 and  $>1$ -mm horizontal, downsloping, or upsloping ST-segment depression in lead I, II, III, augmented voltage left foot (aVF), V3, V4, V5, and V6 at peak stress; *Figure 1*].

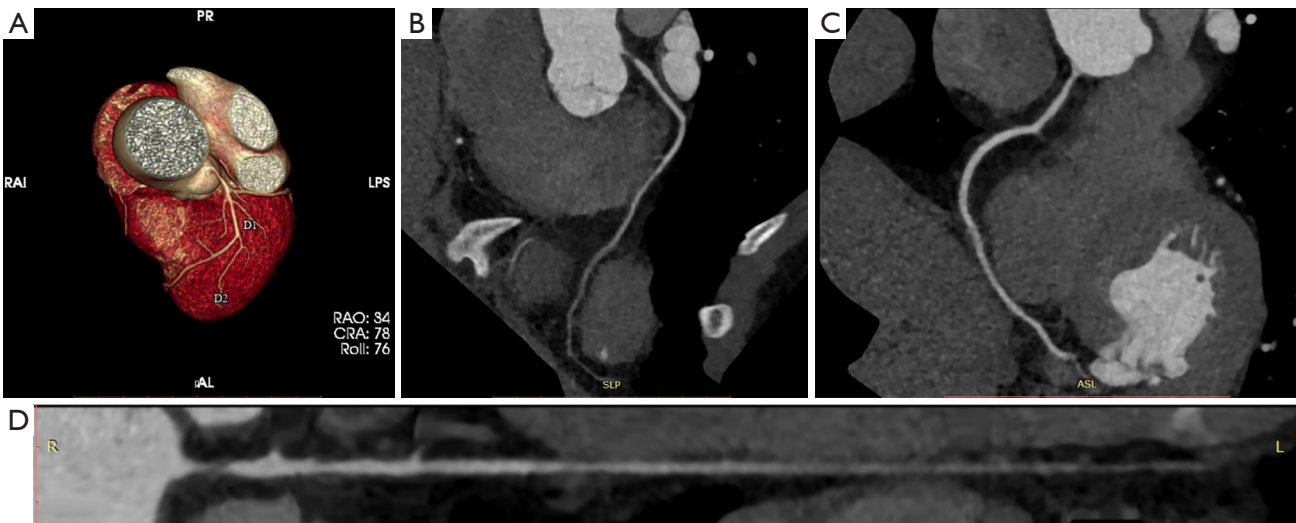
The patient underwent a CCTA for coronary stenosis evaluation in the outpatient department (*Figure 2*). He was admitted to the emergency department with severe constriction around the heart that began as he was stepping out of the CCTA room.

ECG performed in the emergency room showed 1-mm ST-segment elevation in lead aVR and 1-mm ST-segment

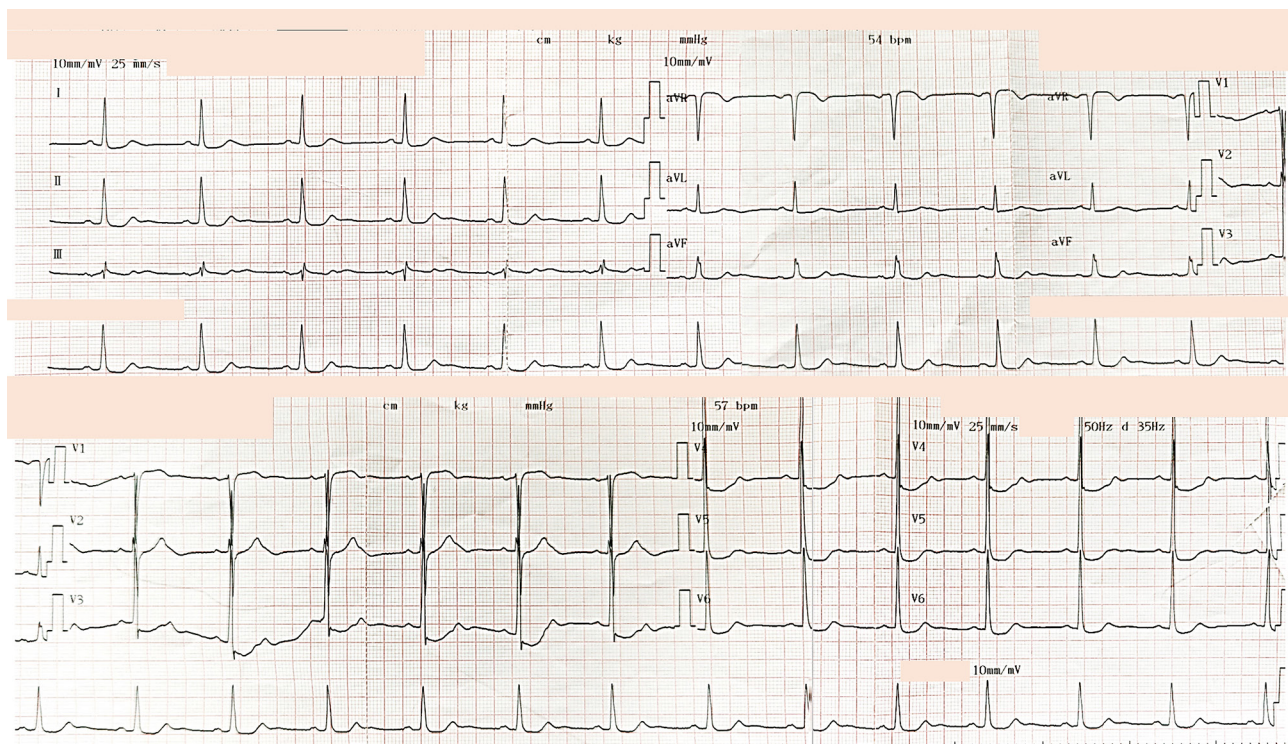
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**Figure 1** ETT showed ST-segment elevation in lead aVR and V1 and ST-segment depression in lead I, II, III, aVF, V3, V4, V5, and V6 at peak stress. ETT, exercise treadmill testing; aVR, augmented voltage right arms; aVL, augmented voltage left arms; aVF, augmented voltage left foot.



**Figure 2** CCTA showed >50% ostial stenosis of the LMCA. (A,B,D) CCTA demonstrated significant narrowing of the LMCA ostial. (C) CCTA demonstrated no significant narrowing of the right coronary artery. PR, posterior right; RAI, right anterior inferior; LPS, left posterior superior; AL, anterior left; D, diagonal branch; RAO, right anterior oblique; CRA, cranial; SLP, superior left posterior; ASL, anterior superior left; R, right; L, left; CCTA, coronary computed tomography angiography; LMCA, left main coronary artery.



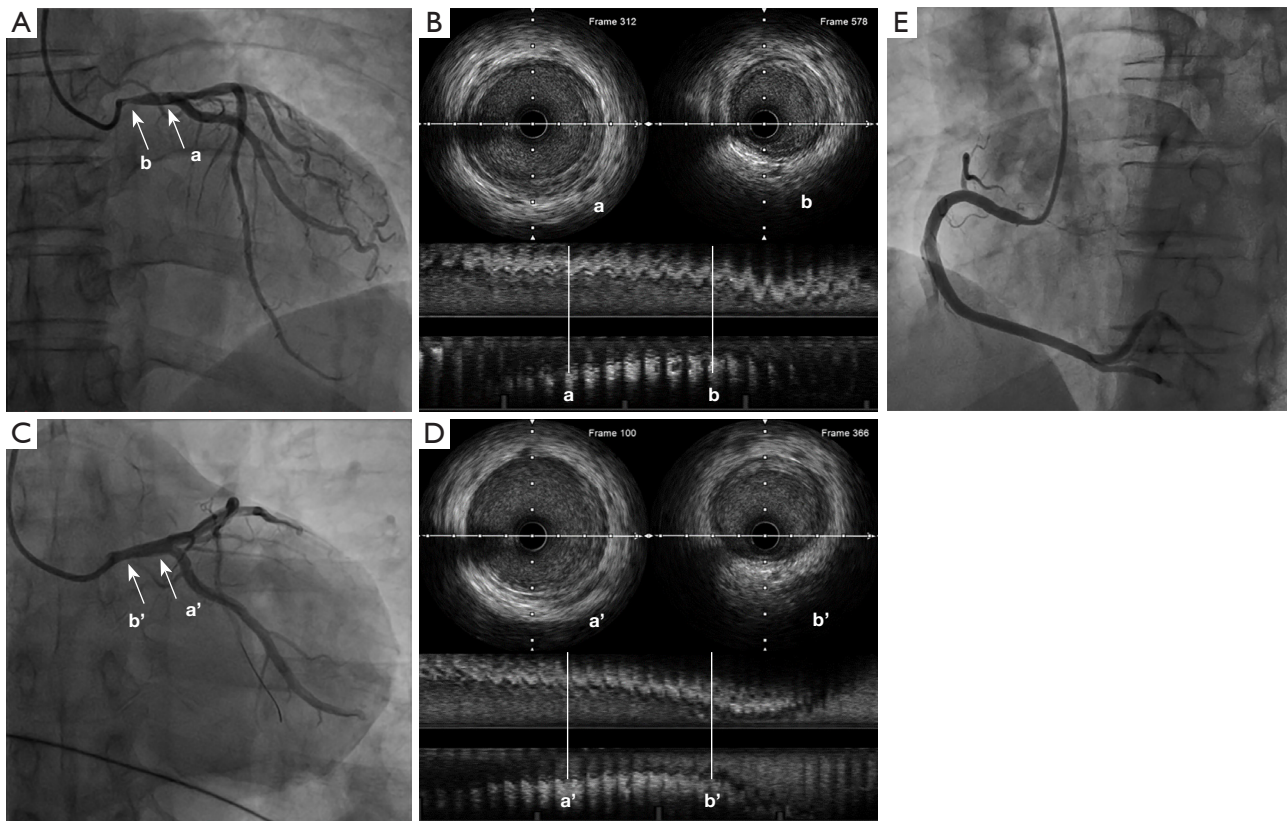
**Figure 3** Electrocardiography showing ST-segment elevation in lead aVR and lead V1, with diffuse ST-segment depressions. aVR, augmented voltage right arms; aVL, augmented voltage left arms; aVF, augmented voltage left foot.

elevation in lead V1, with diffuse ST-segment depressions (Figure 3). According to transthoracic echocardiography, the patient experienced a 48% left ventricular ejection fraction, and his left ventricular wall motion was severely restricted. Laboratory testing demonstrated high levels of troponin I [0.032 ng/mL; reference range (RR): <0.023 ng/mL], myoglobin (514 ng/mL; RR: <112 ng/mL), D-dimer (3,120 ng/L; RR: <500 ng/L), and N-terminal pro-brain natriuretic peptide (8,300 ng/L; RR: <450 ng/L). Lipid profiles suggested that his levels of cholesterol, high-density lipoprotein, low-density lipoprotein, and triglycerides were normal. There was nothing remarkable about his complete metabolic panel except the potassium level (3.41 mmol/L, RR: 3.5–5.3 mmol/L). Cell counts were normal as evidenced by the complete blood count.

In initial risk stratification, he was classified as having very high-risk non-ST-elevation acute coronary syndrome based on the American College of Cardiology/American Heart Association 2007 guidelines for the management of patients with unstable angina/non-ST-elevation myocardial infarction and thus underwent CAG. A 70% stenosis of the ostial portion of the LMCA was detected via CAG, but

the rest of the coronary arteries were normal, which was consistent with the results of CCTA. However, LMCA spasm was suspected in this patient despite the absence of mild conditions in other coronary arteries, and so it was decided that an IVUS was to be performed to corroborate the findings of CAG after intracoronary administration of 200 µg of nitroglycerine prior to percutaneous coronary intervention, which showed negative remodeling of the LMCA without significant atherosclerotic change. After additional administration of 500 µg of nitroglycerine, CAG and IVUS were performed again, which showed a dilated LMCA, suggesting coronary artery spasm was the underlying LMCA stenosis that was detected in the initial CAG, IVUS, and CCTA (Figure 4).

A repeat ECG after CAG also showed nonspecific ST-segment and T-wave changes in the anterior leads. Cardiovascular magnetic resonance (CMR) was performed 7 days after the event. Edema, measured as a high signal, was absent on T2-weighted images. Necrosis, quantified manually as the percentage of left ventricle midmyocardial circumference, was absent on late-gadolinium enhancement images (Figure 5).



**Figure 4** Coronary angiography and IVUS showed severe stenosis of the ostial portion of the LMCA. After additional administration of nitroglycerine, coronary angiography and IVUS showed a dilated LMCA. (A) Coronary angiography demonstrated significant narrowing of the LMCA ostial before infusion of nitroglycerine (the arrow indicates the LMCA shaft; the b arrow indicates the ostial LMCA). (B) IVUS imaging after intracoronary administration of 200 µg of nitroglycerine (a, normal lumen of the LMCA shaft; lumen area =14.78 mm<sup>2</sup>; plaque burden =32%. b, persistent negative remodeling of the ostial LMCA without significant atherosclerotic change; lumen area =6.89 mm<sup>2</sup>; plaque burden =36%). (C) Coronary angiography demonstrated adequate dilation of the ostial LMCA after additional administration of 500 µg of nitroglycerine (the a' arrow indicates the LMCA shaft; the b' arrow indicates the ostial LMCA). (D) IVUS imaging after additional administration of 500 µg of nitroglycerine (a', normal lumen of the LMCA shaft; lumen area =15.32 mm<sup>2</sup>; plaque burden =25%; b', adequate dilation of the ostial LMCA; lumen area =8.45 mm<sup>2</sup>; plaque burden =33%). (E) Coronary angiography detected no significant narrowing of the right coronary artery before infusion of nitroglycerine. IVUS, intravascular ultrasound; LMCA, left main coronary artery.

After being diagnosed with LMCA spasm, this patient was treated with diltiazem (90 mg) orally once daily, aspirin (100 mg) orally once daily, and ticagrelor (90 mg) orally twice daily. He was also reminded to avoid exposure to triggers of coronary artery spasm such as cigarette smoking. The patient was discharged after 7 days of treatment without complications or adverse reactions. He was relieved that episodes of chest pain did not recur after 12 months of follow-up. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013) (4).

Written informed consent was obtained from the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

## Discussion

In patients with suspected ischemic heart disease, obstructive coronary artery diseases do not seem to be the primary cause, and greater attention should be paid to identifying epicardial coronary artery spasm. Out of the roughly 400,000 suspected cases of ischemic heart disease,



**Figure 5** ECG and CMR after coronary angiography. (A) Electrocardiography showing nonspecific ST-segment and T-wave changes in the anterior leads. (B) Edema was absent on T2-weighted images. (C) Necrosis was absent on late-gadolinium enhancement images. aVR, augmented voltage right arms; aVL, augmented voltage left arms; aVF, augmented voltage left foot. PIL, posterior inferior left; ECG, electrocardiography; CMR, cardiovascular magnetic resonance.

no more than 37.6% are reported to have developed obstructive coronary artery diseases (5). The diagnosis of coronary spasm, one of the subtypes of INOCA, should follow the clinical diagnostic flow of INOCA (1).

The identification of patients with epicardial coronary artery spasm is difficult, and this condition can occasionally recur and result in severe ischemia in extreme cases (6,7). Coronary artery vasospasms can be regarded as a risk factor for isolated coronary ostial stenosis (8). When spasticity persists, CCTA and CAG may identify the epicardial coronary artery spasm as ischemia-causing stenosis (2,9). With a maximum scan diameter of 15 mm and a depth of

10 mm, IVUS can distinguish plaques from vascular walls, measure plaque burden, and identify LMCA spasm. Isolated lesions often indicate epicardial coronary artery spasm and can be used as a trigger point to initiate IVUS.

Resting myocardial work indices perform well in assessing the global and regional stress of myocardial perfusion in patients with INOCA (10).

Patients with coronary spasm tend to have a good prognosis in terms of mortality. Meanwhile, patients with epicardial spasm have higher rates of nonfatal myocardial infarction and repeat angiography, while patients with microvascular spasm are at increased risk for recurrent

angina pectoris and psychological disorders (7).

The mechanisms behind epicardial coronary artery spasm are possibly related to endothelial dysfunction, increases in vasoconstrictive substances, growing vulnerability to vascular smooth muscle cells, or abnormality of sympathetic tone (11,12). Vasospasm of the LMCA is an extremely rare but occasionally life-threatening cardiovascular event. Stenting may not be the best solution for LMCA spasm considering the underlying pathophysiologic mechanisms of spasticity, and spasticity may reoccur at the edge of the stent after intervention (13). When a drug-resistant spasm is encountered, drug-eluting stents are relatively safe to use according to the guidelines for diagnosis and treatment of vasospastic angina and coronary microvascular dysfunction (14). Vasodilators including nitrates, nondihydropyridine calcium-channel blockers, dihydropyridine calcium-channel blockers, or nicorandil are the most effective treatment options for coronary artery spasm and should be taken especially before sleep; meanwhile, all types of beta-blockers should be avoided (13,15,16). Quitting smoking, controlling risk factors (hypertension, dyslipidemia, diabetes mellitus), and avoiding precipitating factors (mental stress, exercise in the early morning, cold exposure, hyperventilation, Valsalva maneuver, alcohol consumption, cocaine use) are highly recommended to prevent coronary artery spasm (13). For LMCA spasm that may lead to major adverse cardiovascular events, dual-antiplatelet therapy, particularly a low dose of aspirin, which blocks vasoconstrictor the properties mediated by thromboxane A<sub>2</sub>, should be considered (17).

This case report involved a few limitations which should be considered. Besides ST-segment depression or elevation, a transient leftward shift of the QRS axis  $\geq 15^\circ$  and negative U waves are also highly likely to occur in the stenosis of the left main coronary (18,19). However, the ECG detail recordings of at least two heartbeats in the ETT were not fully preserved.

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### Footnote

*Conflicts of Interest:* All authors have completed the ICMJE uniform disclosure form (available at <https://qims.com>).

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The authors have no conflicts of interest to declare.

*Ethical Statement:* The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this article and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

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