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CASE REPORT

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Widespread ST-segment elevation due to diffuse coronary artery spasm: A case report

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Abstract

Coronary artery spasm (CAS) is considered an important mechanism of acute coronary syndrome but not very common in the clinical setting. We report a case of a 42-year-old woman with chest pain lasting for 4 h due to diffuse CAS, which led to widespread ST-segment elevation in multiple leads of the electrocardiogram and elevated cardiac troponin T. Emergency coronary angiography at admission showed significantly different morphological results from the second angiography during hospitalization, indicating the patient's discomfort was due to CAS rather than stenosis. Our case illustrates that diffuse CAS can cause widespread ST-segment elevation and severe ACS.

KEYWORDS
cardiogenic syncope, coronary artery spasm, ST-segment elevation, vasospastic angina

1 | INTRODUCTION

Vasospastic angina (VA), once known as Prinzmetal angina or variant angina, is a clinical disease characterized by coronary artery spasm (CAS) causing resting angina pectoris and a rapid response to fast-acting nitrates (Kusama et al., 2011). However, diffuse and persistent CAS can cause myocardial infarction with electrocardiogram (ECG) and myocardial enzyme changes. The incidence of CAS shows racial differences. Japanese patients exhibited a threefold greater incidence of spasm and greater vasoconstriction of nonspastic segments after acetylcholine than Caucasians (Pristipino et al., 2000). The clinical manifestation of CAS ranges from asymptomatic to syncope and even death. Syncope was reported to occur in 12.5% (30 of 240 patients) of patients with variant angina (Kishida et al., 1996).

2 | CASE PRESENTATION

A 42-year-old woman with chest pain lasting for 4 h was admitted to our cardiac intensive care unit. She also reported that she had symptoms of syncope, which lasted for 2–3 min, and became awake on her own without twitching on the limbs or incontinence. She had a history of hypertension and cerebral infarction for more than 9 years and was taking amlodipine to control blood pressure. On admission, the physical examination revealed a heart rate of 87 beats/min, a blood pressure of 143/89 mmHg, and an enlarged heart boundary to the left. Findings from biomarker assays showed increased cardiac troponin T (81.4 ng/L, reference 0–14 ng/L). The ECG on admission revealed sinus rhythm with ST-segment elevation in the anteroseptal, inferior, and right precordial leads (V1-V3, II, III, aVF, and V3R-V6R) and ST-segment depression in the lateral leads (I, aVL) accompanied by T-wave inversion in leads V2-V4, I and aVL (Figure 1). An emergency coronary angiogram was performed, which demonstrated the coronary vascular bed was slender with multiple diffuse stenoses, but none of the major coronary arteries was completely occluded (Figure 2; Videos S1 and S2). Diffuse CAS was suspected, which is an infrequent cause of acute coronary syndrome (ACS). The patient was treated with intravenous vasodilators, and her symptoms were gradually alleviated. The next day, the patient experienced chest pain, which was unbearable, along with nausea and vomiting, and the ECG revealed sinus rhythm with ST-segment depression in leads V3-V6,

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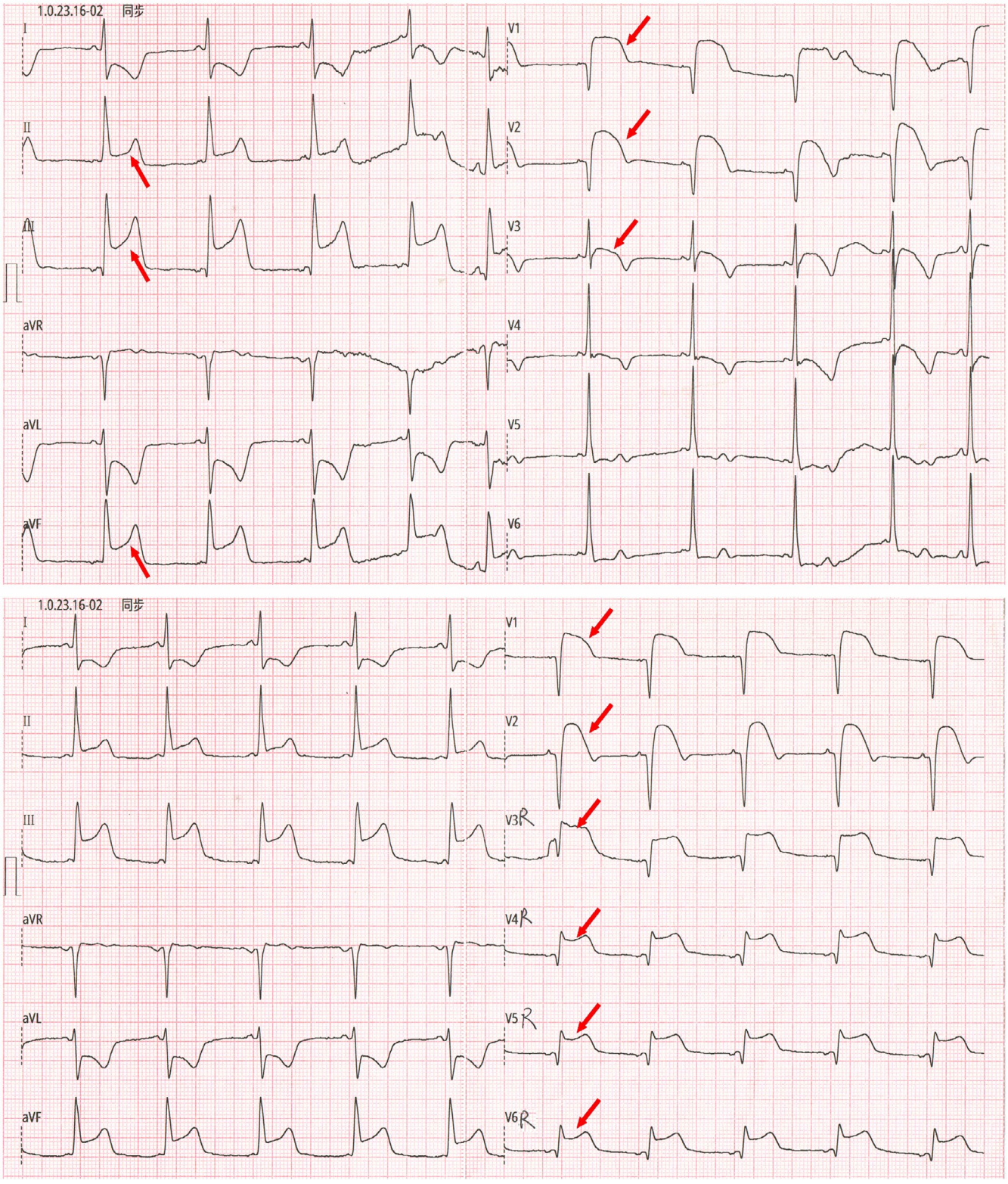

FIGURE 1 Eighteen-lead ECG findings on admission revealing widespread ST-segment elevation. (red arrows: ST-segment elevation at the anteroseptal, inferior, and right precordial leads)

I, and aVL accompanied by T-wave inversion in leads V1-V4, I, and aVL (Figure 3). After treatment with morphine, nitroglycerin, and diltiazem, the patient's pain eased. Another coronary angiogram was performed but revealed completely normal epicardial coronary arteries (Figure 4; Videos S3 and S4). ECG results on the following days showed that the ST-segment depression was improved and T-wave inversion returned to upright (Figure 5). The patient denied tobacco, alcohol, and recreational drug abuse as

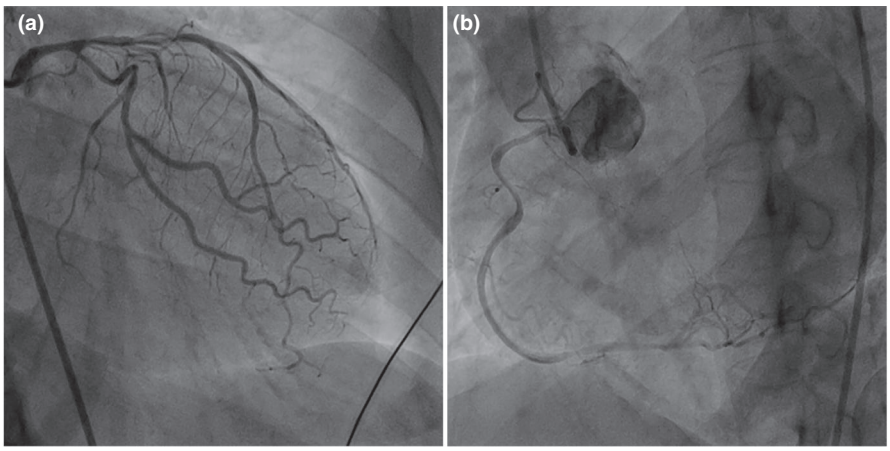

FIGURE 2 The emergency coronary angiogram demonstrates diffuse spasms of multiple coronary arteries. Panel (a). The left coronary artery. Panel (b). The right coronary artery

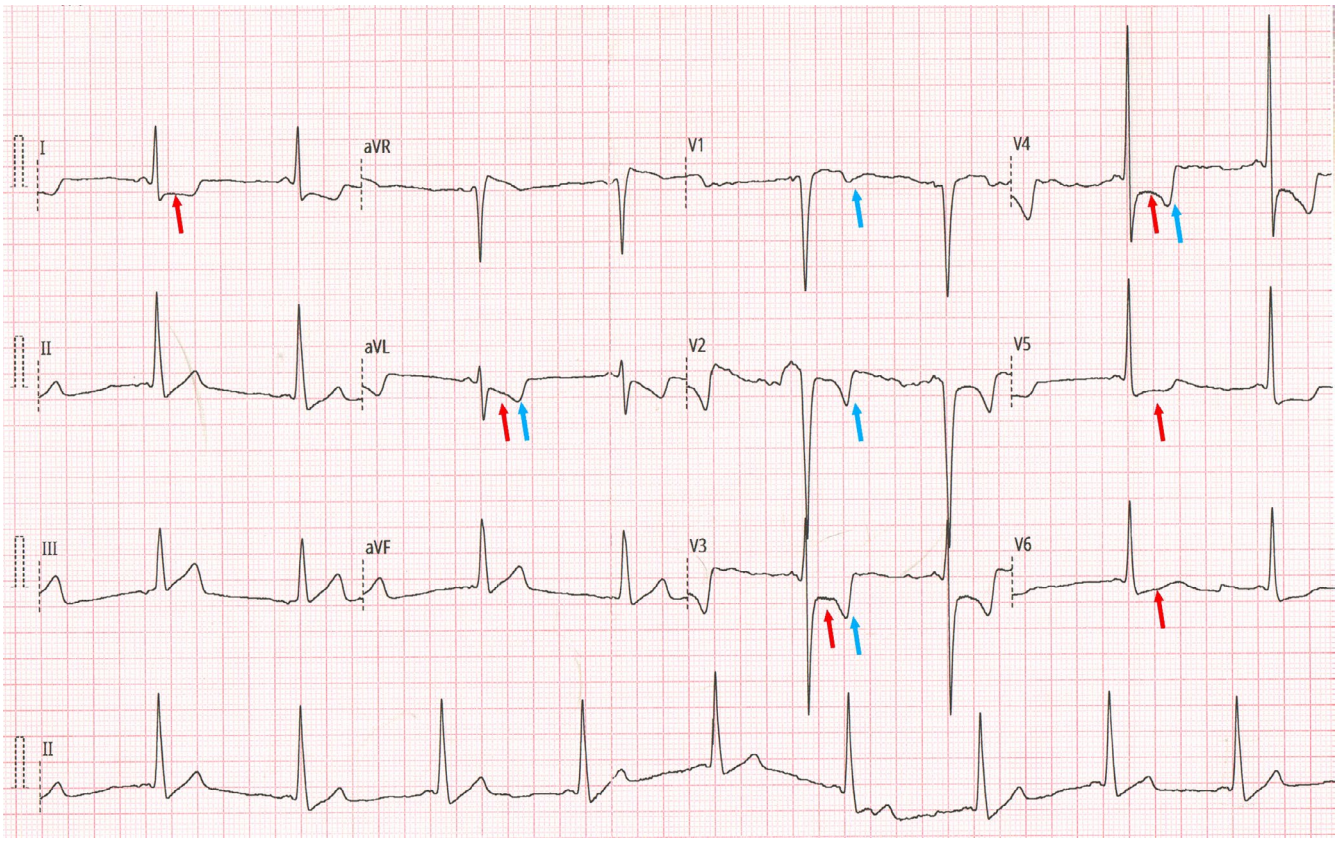

FIGURE 3 The ECG of the patient on the next day, when she recurred chest pain. (red arrows: ST-segment depression in leads V3–V6, I, aVL; blue arrows: T-wave inversion in leads V1–V4, I and aVL)

well as emotional stress. She was discharged with oral vasodilators and diltiazem. During follow-up 6 months later, she reported no episodes of chest pain with the oral drugs.

3 | DISCUSSION

CAS is recognized as an important mechanism involved in the pathogenesis of ACS but is not very common in clinical settings. CAS is caused by focal or diffuse spasm of the smooth muscle layer of the epicardial coronary artery wall (Kaski et al., 1986), which leads to severe stenosis. In many patients, transient myocardial ischemia can cause angina pectoris, and persistent spasms can cause myocardial infarction (Prinzmetal et al., 1959, 1960). The hyperresponsiveness of coronary vascular smooth muscle is considered to be the core of the pathogenesis of vasospasm angina (Kaski et al., 1986; Prinzmetal et al., 1959). In a retrospective observational study including 645 suspected ACS patients, Satoh et al. reported that 11% of those patients (70/645) were confirmed to have CAS. Using the acetylcholine test, diffuse spasms were provoked in 42 patients, and multivessel spasms were provoked in 40 patients (Satoh et al., 2013).

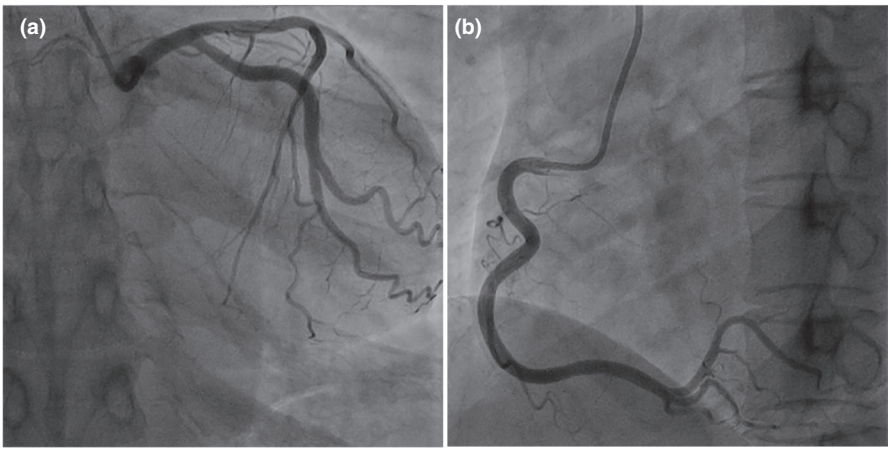

FIGURE 4 The coronary angiogram reveals completely normal epicardial coronary arteries. Panel (a). The left coronary artery. Panel (b). The right coronary artery

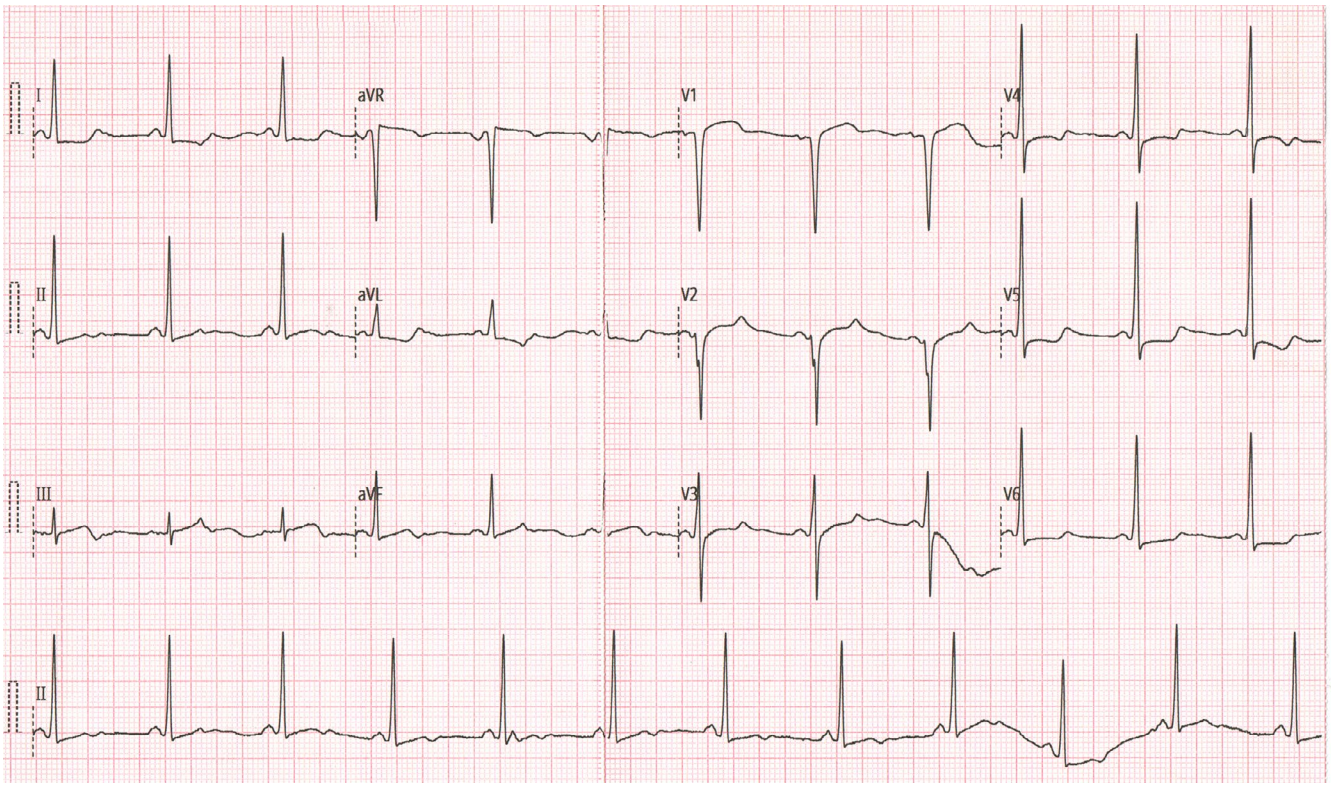

FIGURE 5 The ECG of the following days showed the ST-segment depression was improved and T-wave inversion returned to upright

The common risk factors for CAS include smoking, drug abuse, intoxication, or allergic reactions (Zhang et al., 2016). In addition, age, high-sensitivity c-reactive protein, and remnant lipoproteins are also significant risk factors for CAS (Yasue et al., 2019). However, the identified causes were not always found in many simultaneous CASs.

The ECG changes that occurred during the onset of CAS include ST-segment increase and/or depression with a peaking and/or increasing T-wave amplitude. The spasm completely or partially occludes the main coronary arteries, leading to the elevation of the ST segment in the lead, which represents the area of the myocardium supplied by the spasm artery. ST-segment elevation is typically accompanied by depression in the ST segment in the opposite lead. CAS may also cause ST-segment depression instead of elevation in the lead, representing the area of the spasm artery (Yasue et al., 2019).

In previous research, Kitano et al. demonstrated that yellow plaques containing lipids existed more frequently at the segments of focal CAS compared with the segments of diffuse CAS (Kitano et al., 2018). Given that focal CAS is caused by local hyperreactivity to a generalized constrictor stimulus in mild atherosclerosis, the researchers thought that the pathophysiology of diffuse CAS is limited to atherosclerotic lesions (Kitano et al., 2018). Occasionally, diffuse CAS can cause serious complications, such as cardiogenic shock, malignant arrhythmia (i.e., ventricular arrhythmias), and even death (Shimizu et al., 2018; Zhang et al., 2016). Early recognition and proper treatment are important. In general, ST-segment elevation in ACS

can be localized to a specific portion of the ventricle. Widespread ST-segment elevation indicating ischemia of multiple anatomical segments of the ventricle with blood supply delivered by different major coronary arteries is rare in clinical practice. In this case, the widespread ST-segment elevation, which can be localized to the anteroseptal and inferior portions of the left ventricle and the right ventricle, was a clue indicating diffuse coronary vasospasm.

CAS treatment seeks to reduce the frequency of symptomatic episodes and the incidence of serious complications. Although the attack may terminate spontaneously, sublingual administration of nitroglycerin can effectively shorten the duration of each attack. Nitroglycerin should be taken sublingually during each attack to reduce the duration of symptoms and ischemia. However, the first-line treatment for CAS is long-acting calcium channel blockers. These drugs can prevent coronary vasoconstriction and promote relaxation, thereby alleviating symptoms. It has been reported that statins suppress CAS and improve its prognosis; therefore, Yasue et al. (2008) statins are recommended for the treatment of CAS. Percutaneous coronary intervention (PCI) is not suitable as a routine treatment for patients with focal spasticity and mild obstructive diseases. However, if there is significant obstructive coronary artery disease that is considered a potential cause of focal spasm, PCI may help. PCI is an effective treatment for patients with mild-to-moderate coronary artery disease-related drug-refractory vasospasm, and the spasm vascular segment can be clearly identified (Gaspardone et al., 1999).

In this case, the main limitation is that when the first coronary angiography showed diffuse spasms of multiple coronary arteries, no nitrates or calcium antagonists were used to confirm the diagnosis. However, because the second coronary angiography revealed completely normal epicardial coronary arteries and the patient's symptoms were relieved after using drugs, such as vasodilators and diltiazem, it can be confirmed that the patient's symptoms and ECG changes were due to CAS. In this case, we highlighted that diffuse CAS can cause severe ACS and even cardiogenic syncope.

4 | CONCLUSION

ST-segment elevation in ACS can typically be localized to a specific portion of the ventricle. Widespread ST-segment elevation indicating ischemia of multiple anatomical segments of the ventricle with blood supply delivered by different major coronary arteries is rare in clinical practice. In this case, we present unusual ECG findings suggesting widespread ST-segment elevation of a patient with newly diagnosed diffuse coronary vasospasm as well as the unusual but classic coronary angiogram findings. These results serve as a reminder that diffuse CAS can cause severe ACS and even cardiogenic syncope.

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CONFLICT OF INTEREST

All authors have reported that they have no relationships relevant to the contents of this article to disclose.

ETHICAL APPROVAL

Additionally, the authors have obtained the patient's free informed consent for the publication of this case report per the journal's ethical guidelines.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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