



Vasospastic angina with J waves formation in patients with sudden loss of consciousness

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Abstract

Vasospastic angina is caused by sudden occlusive vasoconstriction of a segment of an epicardial artery, which can present with a wide spectrum of clinical scenario. We report the cases of two patients diagnosed with vasospastic angina, with one of which presenting with sudden cardiac arrest, while the other presenting with a relatively benign syncope. But both of them have J waves formation on ECG during active ischemia. The diagnosis and management of vasospastic angina, as well as the proposed clinical significance of J waves during coronary spasm are discussed.

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Keywords: Angina; Consciousness loss; J wave; Osborn wave

1 Introduction

Vasospastic angina (VSA) is a form of angina caused by coronary artery spasm, which consists of a sudden occlusive vasoconstriction of a segment of an epicardial artery, resulting in a significant reduction of coronary blood flow.^[1] J wave, or Osborn wave, is donated by a notch or slur at the terminal part of the QRS complex, which presents in a variety of clinical situations including ischemia.^[2] We report two cases presenting with sudden loss of consciousness related to VSA and J waves formation.

2 Case Report

2.1 Case 1

A 57-year-old male rushed to emergency room (ER) with sudden rest retrosternal burning pain. During the encounter, he lost the consciousness with no carotid pulse palpated. Cardiopulmonary resuscitation was initiated immediately and electrocardiograph (ECG) showed ventricular fibrilla-

tion. He was successfully resuscitated after 5 min and then ECG revealed intraventricular block without J waves (Figure 1). Past medical history revealed atrial fibrillation managed by metoprolol and propafenone, and sick sinus syndrome on permanent pacemaker. A 30 pack-year smoking history was noted. But there was no significant past medical history, similar episode, or chest pain in the past. We couldn't get a baseline ECG before the encounter. Laboratory test, including troponin, creatine kinase-MB (CK-MB), total CK, and echocardiogram were unremarkable. Coronary angiography showed 20%–30% stenosis of the left main coronary artery (LMCA) and 70%–80% stenosis of the posterior descending coronary artery (Figure 2). Intravascular ultrasound revealed non-stenotic plaques in the middle portion of the anterior descending coronary artery (ADCA) and the LMCA (Figure 3). During the hospitalization, he had resting chest pain and then quickly went into sudden cardiac arrest again. The resuscitation lasted for 30 min, and was successful as before. ECG during chest pain showed ST segment elevation was noted in all V2–V5, aVR and aVL, with further II, III, aVF depression, as well as J waves in V2–V6 (Figure 4), followed by ventricular fibrillation. He was presumably diagnosed with VSA treated with IV diltiazem, followed by oral formulation and atorvastatin. Heavily counseling on smoking cessation was also performed. An implantable cardioverter-defibrillator (ICD) was implanted before discharge.

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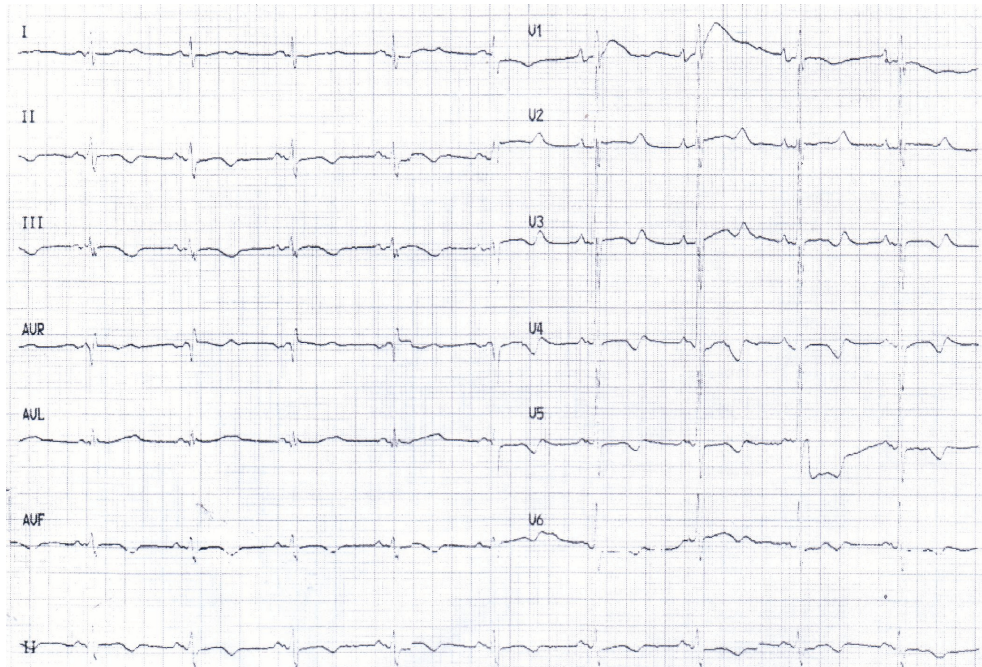


Figure 1. ECG after resuscitation.

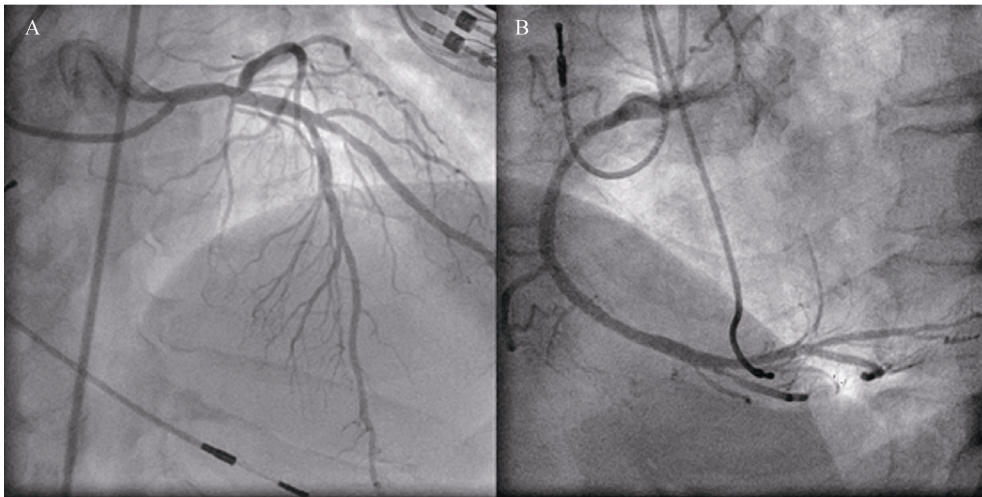


Figure 2. Left (A) and right (B) coronary artery angiography. Coronary angiography showed 20%–30% stenosis at the end point of left main coronary artery and 70%–80% stenosis at the beginning of the posterior descending coronary artery.

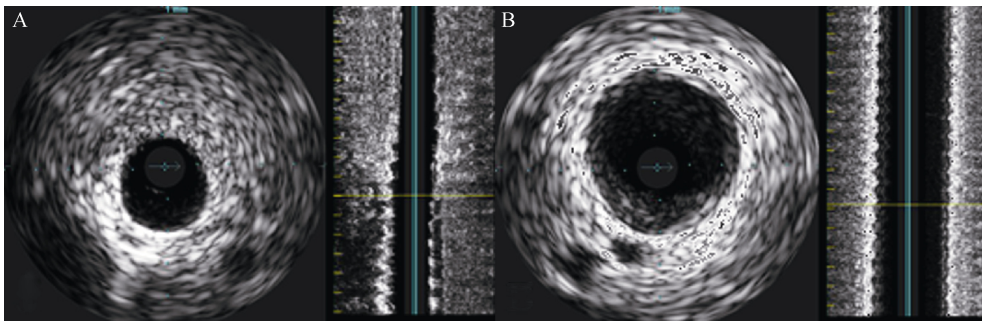


Figure 3. Intravascular ultrasound revealed non-stenotic plaques in the middle portion of the anterior descending (A) and the left main coronary artery (B).

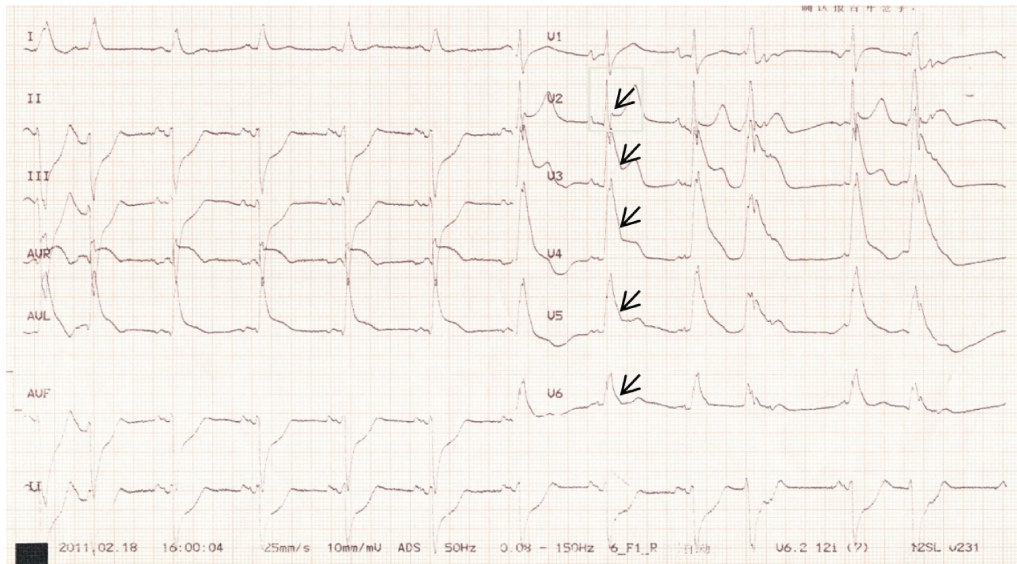


Figure 4. ECG showed ST-segment elevation with J wave formation and QRS widening in V2-V6 leads, and ST-segment depression in II, III, aVF leads. Arrows point to ischemic J waves.

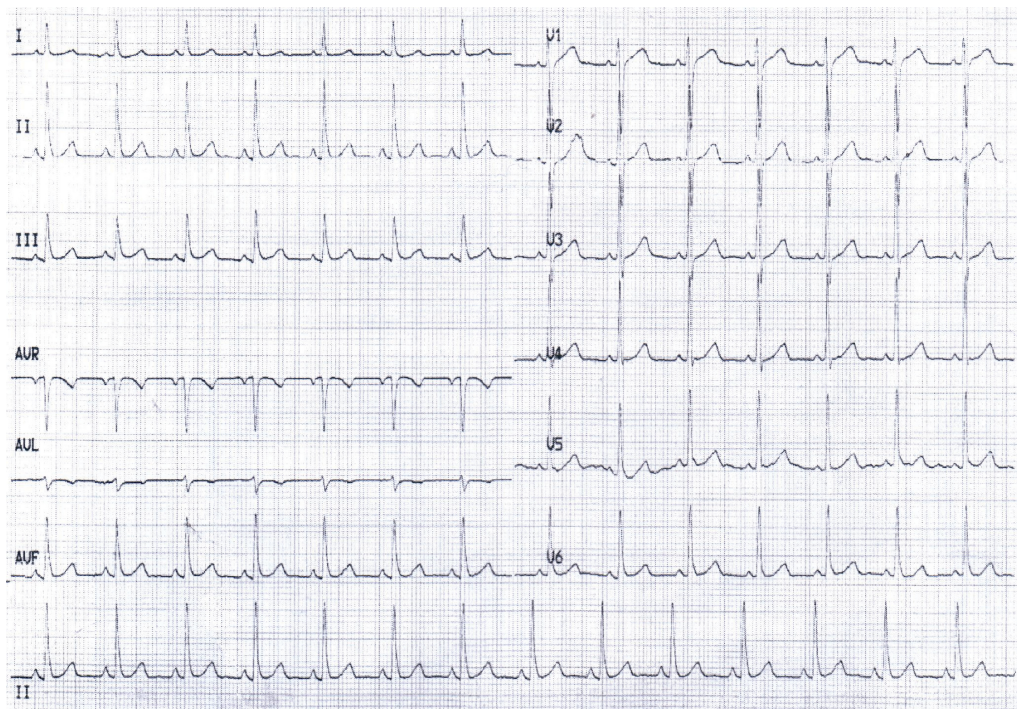


Figure 5. Baseline ECG.

2.2 Case 2

A 53-year-old male had a 2-year history of progressive rest throat and chest tightness in the morning. Each episode lasted for 3 min and resolved spontaneously. One day, he suddenly lost his consciousness for 2 min, then regained it spontaneously, and was taken to the ER. Personal history revealed a 60 pack-year smoking history. Baseline ECG (Figure 5) was unremarkable without J waves. Laboratory

test showed no abnormalities except for mild hyperlipidemia. Echocardiogram failed to show any dysfunction. He was treated with aspirin, clopidogrel and rosuvastatin. He complained rest chest pain in the morning once during hospitalization. ECG showed widening QRS complex, ST segment elevation in I, aVL, V1-V6 leads, with J waves in these leads except V1 lead, and episodes of premature ventricular complex (Figure 6). The symptom also resolved spontaneously after a couple of minutes with resolution of

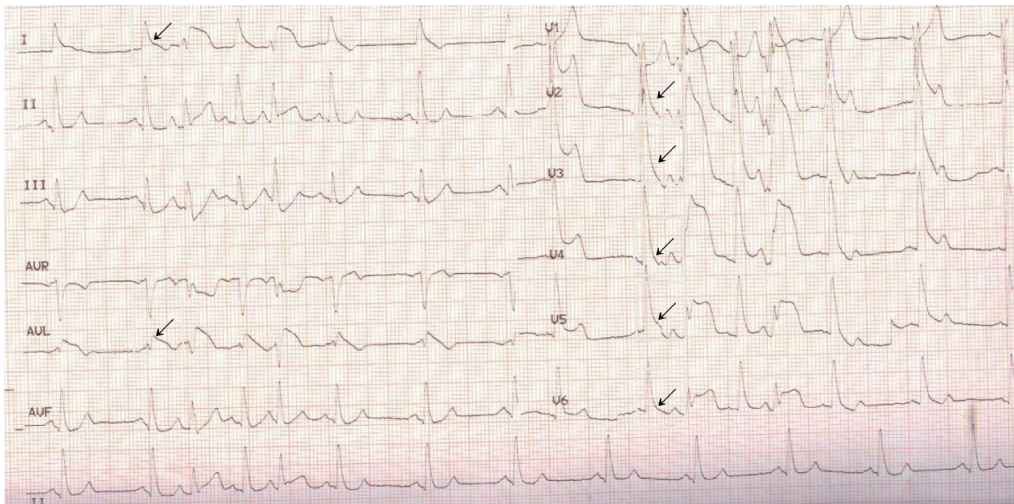


Figure 6. ECG showed ST-segment elevation in I, AVL, V1-V6 leads with J wave formation and QRS widening in these leads except V1 lead. Arrows point to ischemic J waves.

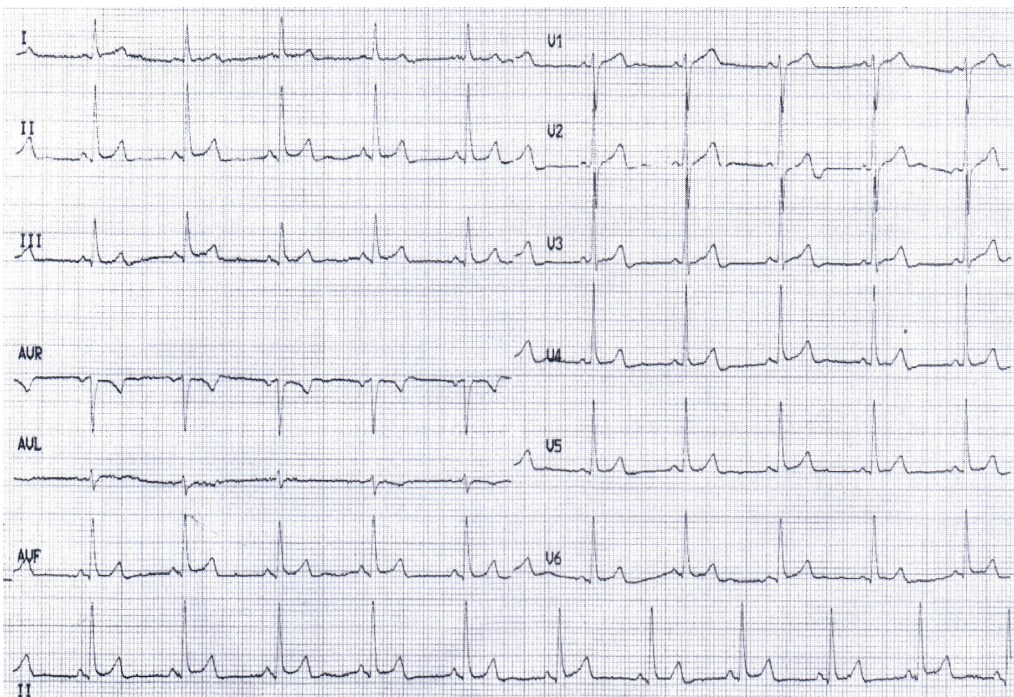


Figure 7. ECG after the resolution of resting chest pain shows.

ECG (Figure 7). Angiography demonstrated 25%–50% stenosis of the left ADCA, 70% stenosis of the left circumflex coronary artery, and 50% stenosis of the right coronary artery (Figure 8). VSA was diagnosed. He was also counseled extensively on stopping smoking and treated with diltiazem, isosorbide mononitrate and rosuvastatin.

3 Discussion

Coronary artery spasm is defined as a condition in which a relatively large coronary artery running on the surface of

the heart transiently exhibits abnormal contraction. According to the 2013 Japanese Circulation Society Guideline, the diagnosis of vasospastic angina (VSA) can be made based on the documentation of transient ST-segment elevation during resting angina episodes and normal or non-obstructive plaques on coronary angiogram, without conducting a provocative test.^[1] In our cases, patients both presented with rest chest pain and transient ST segment elevation with non-obstructive plaques on coronary angiogram. The ECG pattern during the attack indicates possible either multivesel, left main artery disease or proximal left anterior descend-

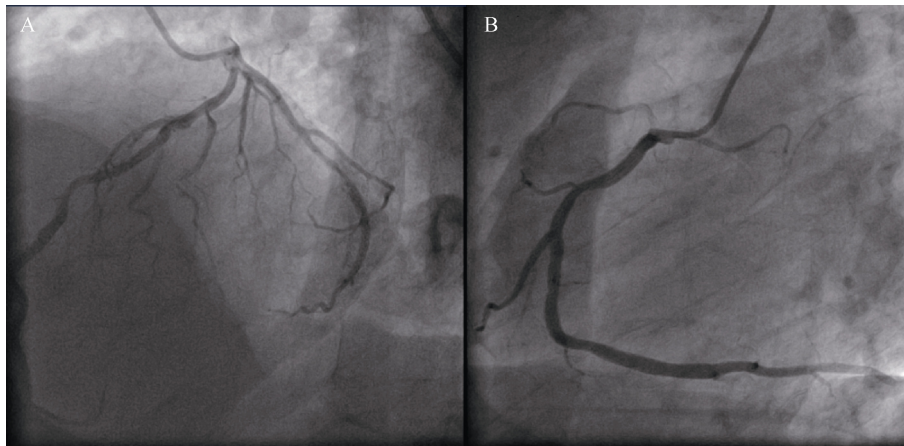


Figure 8. Left (A) and right (B) coronary artery angiography. Coronary angiography showed 25%–50% stenosis of the left anterior descending coronary artery, 70% stenosis of the left circumflex coronary artery, and 50% stenosis of the right coronary artery.

ing involvement.^[3] However, the extent and degree of coronary obstruction during angiography cannot explain. Normal cardiac enzyme and echocardiogram make myocardial infarction and other structural heart disease unlikely. Therefore, the loss of consciousness is highly likely from coronary spasm.

In our cases, J waves with QRS widening in the anterolateral leads coincide with ST-segment elevation during coronary spasm. It is suggested that J-wave pattern recorded in inferolateral leads is a sign of underlying electrical vulnerability that increases the risk of life threatening ventricular arrhythmias (LTVA) under favorable pathological conditions,^[4] and it is reasonable to hypothesize that it also applies when J-wave pattern with QRS widening appeared in the anterolateral leads under severe coronary spasm. But one of our patients presents with sudden cardiac arrest, while the other patient only has syncope, suggesting that J wave causing loss of consciousness and arrhythmias may represent a wide spectrum of risk. The differential diagnosis of J wave and proposed pathophysiology of J waves in LTVA development are discussed in supplemental file 1.

The mainstay treatment for VSA is calcium channel blockers. Smoking cessation is also mandatory. Concomitant nitrate and statin can also be used. But unlike atherosclerotic coronary artery disease, beta-blocker should be avoided and there is no evidence that aspirin is beneficial.^[1] The role of ICDs in patients with VSA is not clear. It is suggested that history of out-of-hospital cardiac arrest (OHCA), smoking, angina at rest alone, significant organic stenosis, multivessel spasm, ST elevation and beta-blocker use are poor prognostic markers of VSA, with OHCA being the most important.^[5] Our case one patient has five of them, and questionable multivessel disease, with risk of major adverse cardiac event at least 13.3%,^[5] so ICD was implanted.

In conclusion, VSA can be diagnosed based on the do-

documentation of transient ST-segment elevation during a resting angina episode. Ischemic J waves can occur in VSA and may indicate a warning sign for LTVA. The standard medical treatment for VSA is calcium channel blockers. An ICD could be considered in patients with high risk features. The clinical significance of J wave in VSA needs further investigations.

Acknowledgements

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Supplemental Material

Differential diagnosis of J waves

The J wave, or Osborn wave, is denoted by a notch or slur at the terminal part of the QRS complex, which presents in a variety of clinical situations including Brugada syndrome, early repolarization syndrome, idiopathic ventricular fibrillation and J waves induced by hypothermia.^[1] It can also be induced by ischemia and accompany ST-segment elevation.^[2] Antzelevich, *et al.*^[1] propose the name “J wave syndromes” for the spectrum of disorders which share a similar mechanism to the J wave development.

Sudden loss of consciousness with J waves and ST segment elevation raise the suspicion of Brugada syndrome and early repolarization syndrome.^[1] However, the chest pain symptom during the episodes concomitantly with anterolateral leads involvement makes these syndromes unlikely.^[1] Also, both patients respond well to calcium channel blocker and nitrate treatment, which have not been documented to be effective to other J wave syndromes.^[3] Lastly, the theory that atherosclerotic disease alters the vasomotor tone and reactivity of the affected vessels, which can lead to a particular susceptibility to coronary spasm events in sites of organic stenosis, also favors vasospastic angina a reasonable diagnosis.^[4]

Pathophysiology of J waves in life threatening ventricular arrhythmia development

Experimental studies hypothesize that transient outward K⁺ current (I_{to})-mediated prominent action potential notch in the epicardium, but not endocardium, causes a transmural voltage gradient resulting in J waves on the surface ECG.^[5] It is also suggested that acute ischemia, which may cause opening of K_{ATP} channels and reduction calcium channel current, leads to an in-

crease in the magnitude of the epicardial action potential notch and partial or total loss of action potential dome at phase II. At the same time, heterogeneous loss of the epicardial action potential dome induced a marked increase in the dispersion of repolarization as well as phase 2 reentry, which can be responsible for sustained ventricular arrhythmias.^[6,7] Animal study also shows that J-wave pattern together with QRS widening in acute myocardial ischemia is predictive of impending ventricular fibrillation.^[8]

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