Case Report

'Action potential-like' ST elevation following pseudo-Wellens' electrocardiogram

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ABSTRACT

Coronary artery vasospasm is an important cause of chest pain syndromes that can lead to myocardial infarction, ventricular arrhythmias, and sudden death. In 1959, Prinzmetal et al described a syndrome of nonexertional chest pain with ST-segment elevation on electrocardiography. Persistent angina is challenging, and repeated coronary angioplasty may be required in this syndrome. Calcium antagonists are extremely effective in treating and preventing coronary spasm, and may provide long-lasting relief for the patient. Whereas the Wellens' syndrome is characterized by symmetrically inverted T-waves with preserved R waves in the precordial leads suggestive of impending myocardial infarction due to a critical proximal left anterior descending stenosis, the pseudo-Wellens' syndrome caused by coronary artery spasm has also rarely been reported in literature. We present a pseudo-Wellens syndrome as a cause of vasospastic angina, and a diffuse ST segment elevation on electrocardiogram resembling the Greek letter lambda, called also 'action potential-like' ECG in a patient with vasospastic-type Prinzmetal angina.

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1. Introduction

Resting angina is one of the most frequent symptoms of patients in the emergency room.1 Many patients with chest pain who have no obstructive stenoses at diagnostic angiography are misclassified as having noncardiac chest pain.2 Variant angina is a form of angina pectoris that shows transient ST-segment elevation on electrocardiogram during an attack of chest pain. Ischemic episodes of variant angina show circadian variation and often occur at rest from midnight to early morning.3 The demonstration of either spontaneous or provoked coronary spasm proves coronary hypercontractility and thus the diagnosis of variant angina.4 Coronary spasm could be documented in nearly 50% of the suspected ACS in emergency departments.5 Risk stratification of patients with recurrent chest pain and normal coronary angiogram is a relevant but still definitely unsolved clinical problem.6 Although coronary artery vasospasm can be suspected clinically, proof cannot usually be obtained by non-invasive means but is easily available during cardiac catheterization. Patients with vasospastic angina are repeatedly exposed to this invasive procedure as most cardiologists suspect a coronary lesion (requiring intervention) as the cause of the patient’s resting angina.7

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2. Case report

A 33-year-old man presented with substernal, crushing chest pain that lasted intermittently for 60 min. He had none of the classical risk factors for coronary artery disease, and did not use alcohol, tobacco, or cocaine. He had a history of emergency coronary angiogram one month before due to similar chest pain. An electrocardiogram (ECG) revealed sinus rhythm, biphasic T waves with preserved R waves in V1–V4 precordial leads (Fig. 1); but these appearances were similar to those noted before his recent discharge from the hospital (Fig. 2). We decided to follow the patient. However, in the early morning hours about 5.5 h after admission, his severe chest pain restarted, and his ECG showed lamda-like diffuse ST segment elevation with reciprocal ST depression in aVR and V1 (Fig. 3). Immediately after starting intravenous nitroglycerine, his diffuse 'action potential-like' ST segment elevation returned to baseline (Fig. 4), concomitant with his pain subsiding. Since we did not have the previous angiogram data; we planned to perform immediate coronary angiography, which showed normal coronary arteries except mild lesion at mid left anterior descending coronary artery. The next day, his previous coronary angiogram taken one month earlier revealed that the angiographic findings were similar to those noted before his recent discharge from the outside hospital. The clinical and ECG pictures pointed to the diagnosis of a vasospastic-type Printzmetal angina, and the patient responded well to calcium blockers and long-term nitroglycerin therapy and remained symptom-free throughout a 6-month follow-up.
3. Discussion

Coronary artery spasm is a hyper-contraction of coronary smooth muscle triggered by an increase of intracellular Ca\(^{2+}\) in the presence of an increased Ca\(^{2+}\) sensitivity.\(^8\) Electrocardiographically, the attacks of coronary spasm are associated with either ST segment elevation or depression, or negative U wave on ECG.\(^8\) Whereas the Wellens’ syndrome is characterized by symmetrically inverted T-waves with preserved R waves in the precordial leads suggestive of impending myocardial infarction due to a critical proximal...

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Fig. 3 – The 12 lead ECG during severe chest pain shows the diffuse ST segment elevation resembling the Greek letter lambda, called also 'action potential-like' shape or ‘tomb shape-like’ in precordial leads.

Fig. 4 – The 12 lead ECG immediately after starting intravenous nitroglycerine showing the his diffuse ST segment elevation returned to baseline.
left anterior descending stenosis, the pseudo-Wellens’ syndrome caused by coronary artery spasm has also been rarely reported in literature. Complete or near-complete occlusion of LAD causes severe myocardial ischemia and angina. When coronary flow is restored, reperfusion injury related repolarization abnormalities will be seen in precordial leads with inverted or biphasic T-waves. Absolute recovering of stunned myocardium results in normalized T-waves. Not only severe stenosis, any cause of coronary flow interruption, including spasm, could cause characteristic T-wave changes. Our patient had a pseudo-Wellens sign on ECG at first admission, and a diffuse ST segment elevation on electrocardiogram resembling the Greek letter lambda, called also ‘action potential-like’ shape or “tomb shape-like” in precordial leads (Fig. 3). This specific ST segment elevation was accompanied by an upsloping ST segment depression in aVR and V 1. ACS patients without culprit lesion and proof of coronary spasm have an excellent prognosis in terms of survival and coronary events after 3 years compared with patients with obstructive ACS; however, persistent angina represents a challenging problem in these patients, leading in some cases to repeated coronary angiography.

Conflicts of interest

The authors have none to declare.

References