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CASE REPORT

Wellens' Syndrome with a proximal left anterior descending artery occlusion

Hongcheng Mai[1,#], XianWu Lan[2,#], Dan Lu[1,#], Yusheng Zhang[1], AiDong Zhang[2] & Tao Zhang[2]

[1]Department of internal neurology, Guangzhou Overseas Chinese Hospital, Guangzhou, Guangdong 510632, China
[2]Department of internal cardiology, Guangzhou Overseas Chinese Hospital, Guangzhou, Guangdong 510632, China

Correspondence
Tao Zhang, Department of internal cardiology, Guangzhou Overseas Chinese Hospital, Guangzhou, 510632 Guangdong, China. Tel: +8613016087802;
E-mail: heartdiease@126.com

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[#]These authors contributed equally to this work.

Key Clinical Message

The case is a 52-year-old male admitted to cardiology department with chest tightness. Admission ECG showed nontypical T-wave changes in V2–V4 leads in pain peroids, and increasing severe narrowing of proximal LAD. Cardiac enzymes were abnormal. Emergency coronary angiography showed severe stenosis (99%) in proximal LAD.

Keywords

Electrocardiographic, percutaneous coronary intervention, T-wave syndrome, Wellens' syndrome.

Introduction

Wellens' syndrome is an acute coronary syndrome that has a high risk for myocardial ischemic disease and threatens patients' life. According to electrocardiogram (ECG) patterns, Wellens' syndrome can be divided into two types: changes of symmetric, deeply biphasic T-waves classified as type 1 or inverted T waves classified as type 2 in precordial leads. These patterns are especially seen in V2–V3 leads during pain-free periods, and upright T-waves with possible elevated or isoelectric ST segments usually seen during pain episodes. Early diagnosis and appropriate intervention will carry good outcome [1]. We herein report a case of Wellens' syndrome with chest tightness and efficient PCI should be considered of these patients.

Case Report

A 52-year-old male smoker (20 cigarettes a day for 30 years) with a history of diabetes mellitus, presented to an outlying hospital with acute-onset squeezing chest tightness in precordium at rest for 24 h. With ECG indicative myocardial infarction, he was treated with oral aspirin 300 mg and clopidogrel 300 mg before chest tightness remission. Promptly hospitalized in the internal cardiology of our hospital, he had an initial diagnosis of acute ST segment elevation myocardial infarction with ST

segment elevation and biphasic T-waves in V1–V4 by ECG (Fig. 1A). Considering CK-MB 49 U/L and cTnI 2.09 ng/mL in laboratory studies, he was treated with oral aspirin 100 mg and clopidogrel 75 mg, subcutaneous low molecular-weight heparin 0.4 mL, oral perindopril 2 mg, and oral lipitor 20 mg. The next morning, his cTnI rechecked was 2.46 ng/mL at 6:00. He presented severe chest pain at 8:50. Urgent ECG revealed that T-waves showed biphasic laterally in V1 and inversed in V2–V4 (Fig. 1C), without ST segment changes. Emergency coronary angiography showed severe stenosis (99%) in proximal LAD at 10:25 (Fig. 1B), and immediately a Stent Helios 3.0 × 19 mm was successfully implanted (Fig. 1D). Vectorcardiogram at discharge showed sinus rhythm which made high agreement in diagnosis recanalization with angiography (Fig 1E and F). He was stable for 2 months, and has been in constant follow-up and not experienced angina again.

Discussion

In 1982, HJ Wellens and Zwaan described a subtype of unstable angina, which is characterized by precordial lead T-wave dynamic evolution without ST segment deviation and shown severe stenosis of left anterior descending in coronary angiography results. About mentioned clinical manifestations are also named anterior descending T-wave syndrome [2]. In clinic, these patients are diagnosed during unstable angina catabasis or the classic T-wave findings seen on an ECG taken

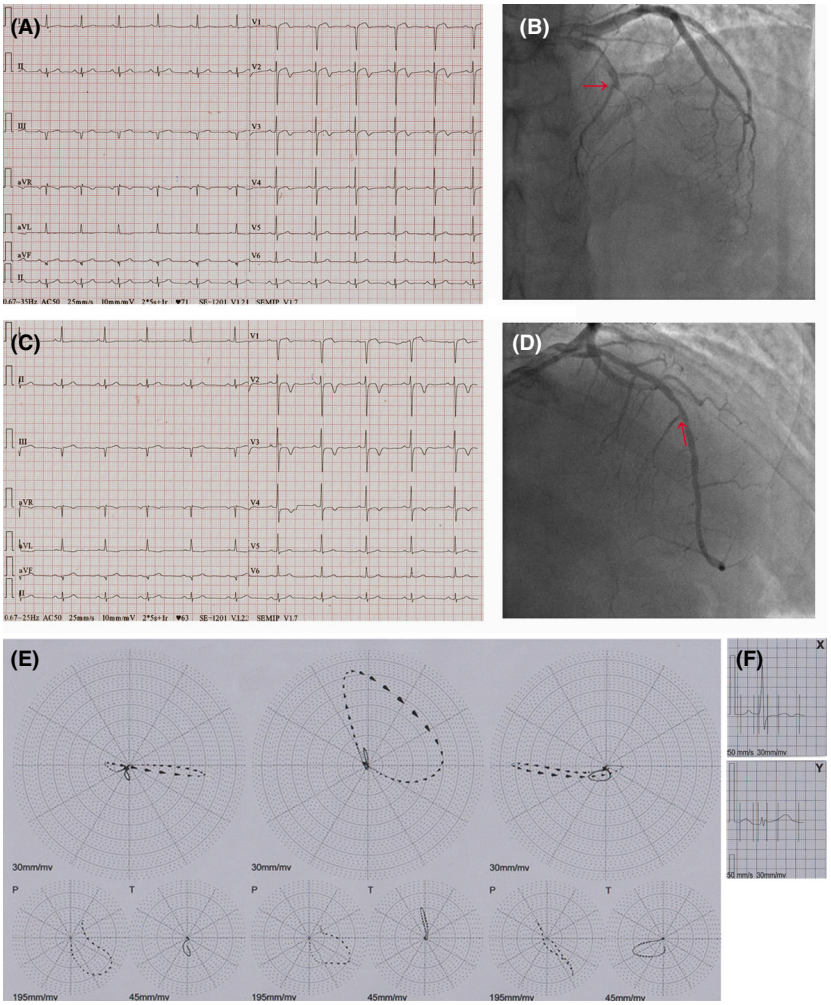

Figure 1. A. Initial ECG with ST segment elevation and biphasic T waves in V1–V4. B. Coronary angiography. Arrow indicates stenosis in LAD before stent implanting. TIMI = 0~1. C. ECG after the chest pain showed biphasic T waves in V1, T waves inversed in V2–V4 without ST segment elevation. D. coronary angiography after stent implanting. Arrow indicates stent in LAD. TIMI = 3. E and F Vectorcardiogram after stent implanting and the pain was relieved.

when pain free, with normal or slightly increased myocardial enzymes, of which electrocardiogram characteristics: characteristic inversed or biphasic T-wave found in V2–V3, sometimes in V1, V4 in the anterior leads with preserved R-wave progression and without pathologic Q-waves and ST-segment elevation [3, 4]. About 8.5 days later, these patients will develop myocardial infarction.

The complex and unanswered mechanisms of Wellens' Syndrome confuse the doctors to diagnosis and treatment, although there are some theories of coronary artery spasm and stunned myocardium by T-waves [5]. For this kind of patients, treatment with dual antiplatelet (oral aspirin and clopidogrel), antithrombosis, blood pressure and blood sugar control, and oral statin therapy [1] did not achieve good control effect. For avoiding the condition of developing myocardial infarction rapidly, we performed PCI reconstruction of coronary blood supply when the patient had alleviated chest tightness with T-waves changes and slightly nTNI elevation at hospital admission, but the onset of angina within 24 h and T-wave dynamic evolution.

Conclusion

Wellens' syndrome is crucial to recognize promptly. Early identification of characteristic ECG and clinical changes may provide critical clues for Wellens' syndrome. Once Wellens' syndrome has been diagnosed, the patients should be ideally allowed to carry out an emergency coronary angiography and plan the revascularization via PCI. Urgent intervention treatment is important to avoid evolution to acute anterior myocardial infarction and possible sudden death.

Conflict of Interest

None declared.

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