

Wellens Syndrome in a patient with a history of hypertension and chronic obstructive pulmonary disease: a case report

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Abstract

Wellens syndrome, an ST Elevation Myocardial Infarction (STEMI) equivalent, is also known as T-wave left anterior descending (LAD) coronary artery disease. Wellens syndrome is characterized by a unique electrocardiogram (ECG) pattern that suggests a significant stenosis in the left anterior descending coronary artery that warrants immediate intervention. Hereby, we present a case report of Wellens syndrome in a patient with a history of hypertension and chronic obstructive pulmonary disease (COPD) that may be potentially mistaken for pseudo-Wellens syndrome because the ECG pattern mimics left ventricular strain pattern (LVSP) in left ventricular hypertrophy (LVH). Thus, cautious examination of recent chest pain and ECG is important to differentiate Wellens syndrome and LVSP in patients with hypertension and COPD to perform early detection and aggressive intervention since they may help to lessen the adverse results.

Keywords: Coronary Artery Disease, Myocardial, Electrocardiography, Chest Pain, Hypertrophy, Arrhythmias, Cardiac

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Introduction

Approximately 15% of the population who suffers from unstable angina has Wellens pattern, a unique ECG pattern associated with a greater risk of developing extensive anterior wall infarction.^{1,2} This pre-infarction stage usually begins with a significant stenosis in the left anterior descending (LAD) coronary artery, which is characterised by Wellens ECG pattern that suggests a significant stenosis in the LAD coronary artery.^{1,3,4} If the patients with Wellens syndrome are not revascularized promptly, three-quarters of them will suffer from

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extensive anterior wall myocardial infarctions (MIs).¹ Wellens syndrome has significant diagnostic and prognostic value due to its greater risk for morbidity and mortality.¹ There are two types of ECG changes in Wellens syndrome: type A with biphasic T-waves and type B with deep negative T-waves, particularly in leads V2 and V3.³

Wellens syndrome shares all traditional risk factors for coronary artery disease.¹ Even though Wellens is easy to diagnose by noninvasive ECG criteria, doctors frequently face difficulties in determining its diagnosis.¹ Several common clinical conditions share the same characteristic ECG pattern, making it difficult to identify this ominous syndrome.^{1,2,4} Hereby, we present a case of Wellens syndrome in a patient with a history of hypertension and COPD that may potentially be mistaken for the pseudo-Wellens syndrome. This case was seen at Dr. Soetomo General Hospital, in Surabaya, East Java in February, 2022. Written consent from the patient was obtained for publishing his case.

Case Report

A 69-year-old patient came to the emergency ward of Dr. Soetomo General Hospital, in Surabaya, East Java in February, 2022, with a history of hypertension and Chronic Obstructive Pulmonary Disease (COPD). His chief complaint was recurrent typical chest pain for the last two days with increasing intensity. The patient was a heavy smoker and had quit since the last two months. He was on therapy for hypertension and COPD, and there was no history of illicit drug use. At the time of admission, he was chest pain-free and his vital signs were unremarkable. The electrocardiography (ECG) during a free pain state revealed deep symmetric inverted T-waves in leads V2–V5. Figure-1. Troponin I level was slightly increased and other laboratory findings were within normal limits. An x-ray of the anteroposterior thorax revealed emphysematous lung and a prominent heart with a cardiothoracic ratio (CTR) of 52%.

Figure-2. Echocardiography results showed trivial mitral regurgitation and tricuspid regurgitation, grade 1 diastolic dysfunction, normal regional wall motion, and a

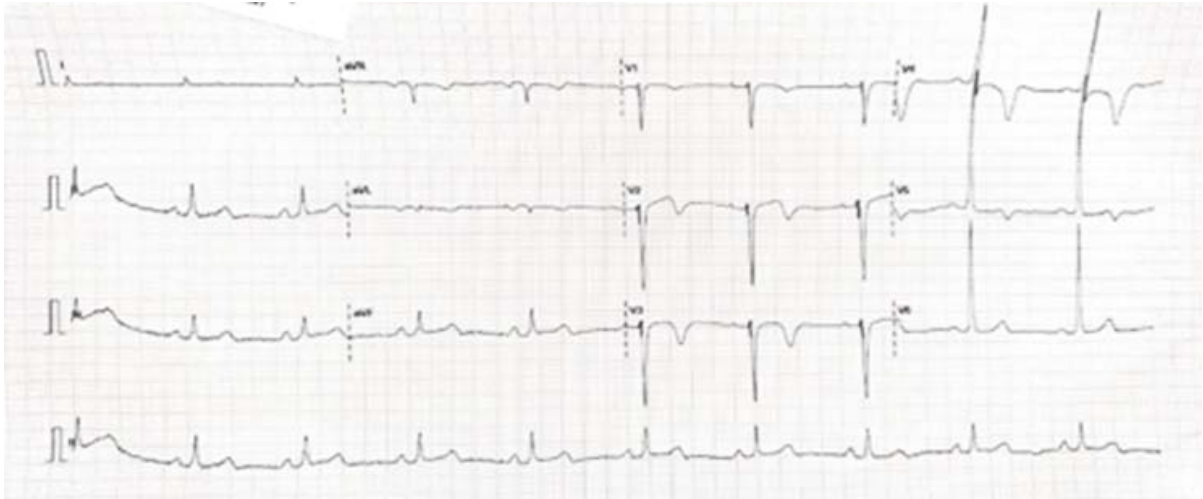


Figure-1: Admission ECG showed deep symmetric inverted T-wave in leads V2– V5 (Wellens type B).



Figure-2: X-ray of the anteroposterior thorax revealed emphysematous lung and a prominent heart.

62% ejection fraction by TEICH.

T-wave abnormalities in this case may be mistaken for LVSP or pseudo-Wellens syndrome due to LVH. However, based on the symptoms of recurrent angina and pathognomonic electrocardiogram pattern in a pain-free condition that are consistent with Wellens type B. The patient was then planned for cardiac catheterization. Coronary angiography confirmed total occlusion in mid-LAD, Figure-3, and was followed by stenting with a drug-eluting stent. Post-stenting, TIMI 3 LAD flow was observed. After the procedure, the patient was discharged two days later in good condition with isosorbide dinitrate, atorvastatin, beta-blocker, angiotensin receptor blocker, aspirin and clopidogrel.

Discussion

Wellens syndrome, also known as LAD coronary

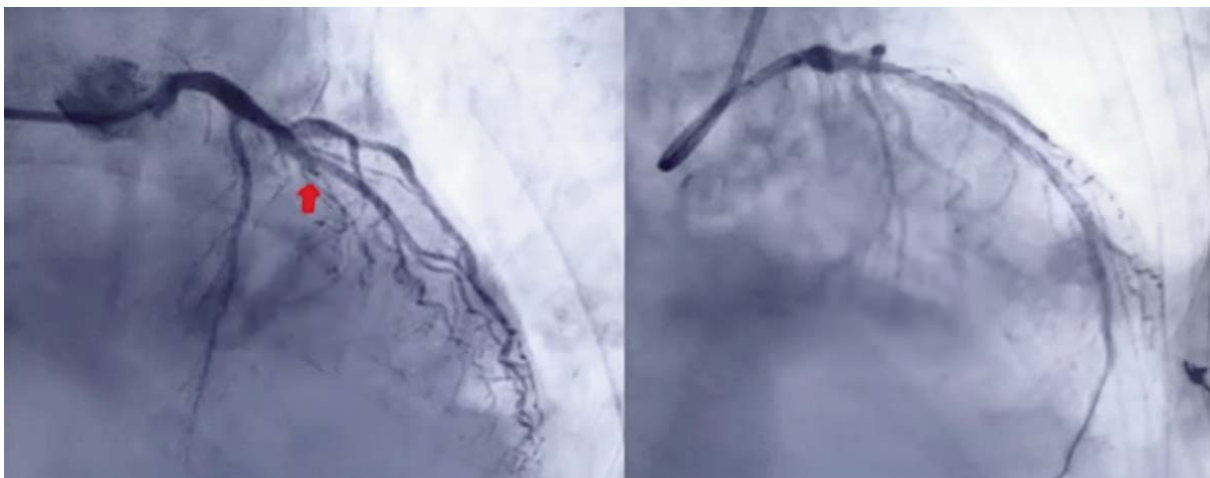


Figure-3: Coronary angiography revealed total occlusion in mid-LAD (red arrow).

syndrome or widow maker, was initially seen in a subset of patients with unstable angina during a pain-free phase described in 1982 by de Zwaan et al.⁵ It was linked to more than 90% proximal LAD artery stenosis, and 75% of patients experienced anterior wall myocardial infarction within a few days to weeks of presenting.⁵⁻⁷

There are two types of Wellens syndrome: type A and type B.⁵ Biphasic T-waves in leads V2 and V3 are a sign of type A, but profound T-wave inversions are a sign of type B in those same leads.⁵ Both of these ECG abnormalities, however, can extend to include any pericardial chest leads,⁵ as was the situation in this case. While finding ECG changes that match the Wellens criteria is important, it should be highlighted that inverted T-waves that are significant for LAD stenosis have a 69% sensitivity, an 89% specificity, and an 86% positive predictive value.² This indicates that ECG alterations suggestive of Wellens syndrome do not always indicate the presence of the condition and should be considered in the differential diagnosis for the pseudo-Wellens syndrome.² The term "pseudo-Wellens syndrome" refers to comparable distinctive ECG abnormalities resulting from sources other than LAD stenosis.⁶

Several clinical conditions might have been linked to pseudo-Wellens ECG patterns, such as acute pericarditis, persistent juvenile T-wave inversion, Takotsubo cardiomyopathy, intracerebral haemorrhage, acute pulmonary embolism, Prinzmetal angina, early repolarization, hypokalaemia, complete right bundle branch block, Wolff-Parkinson-White pre-excitation, and left ventricular hypertrophy.^{1,2,4}

The establishment of Wellens syndrome includes several criteria, including the following: history of anginal chest pain; normal or minimal (1 mm) ST-segment elevation; normal or slightly elevated cardiac enzyme levels; normal precordial R-wave progression; absent precordial Q-waves; and deeply inverted or biphasic T-waves in the leads V2 and V3, which occasionally may extend to V1–V6, taken during a pain-free period.^{2,5,6}

This patient had a deep symmetric inverted T-wave in leads V2–V5 and was diagnosed with Type 2 or B Wellens, which accounts for 75% of cases,⁶ as noted in this case. The mortality rate is comparatively higher in type B Wellens syndrome.⁶ Type 1 or A Wellens syndrome, which is represented by biphasic T-waves mostly in V2 and V3, accounts for the remaining 25%.⁶

In this case, T-wave abnormalities may be mistaken for LVSP due to LVH.² However, LVSP should be observed in lateral leads (V4-6, I, and aVL), while Wellens syndrome is

typically seen in anterior leads (V2-4).² The ECG diagnosis of LVH based on voltage criteria requires the presence of prominent electric forces in the QRS complexes of leads V1 to V6 for LVH.⁴ Additionally, the Electrophysiologists (EP) must be aware that leads I, aVL, V5, and V6 are typically the only ones experiencing this repolarization change in left ventricular hypertrophy.⁴

When assessing individuals with anterior T-wave abnormalities on ECG, LVH secondary to hypertension should be taken into consideration because it can mimic Wellens syndrome and the ST-T segment alterations in LVH are caused by aberrant repolarization of the left ventricle's hypertrophied muscle.² It is worthy to note that evidence also showed the association between COPD and LVH,⁸ making the diagnosis of Wellens syndrome more challenging.

This patient had a complete blockage of the left anterior descending artery, although no ST-elevation was visible on the ECG.^{9,10} An ECG of the Wellens type demonstrated how the progression of the T-waves might signify significant stenosis.^{9,10} Early identification of this patient's Wellens characteristic ECG led to an early choice to employ an invasive technique, which eventually produced positive results.^{7,9-10}

Conclusion

This case report showed that distinct ECG patterns of Wellens syndrome often go unrecognized and can also be mistaken with Pseudo-Wellens syndrome, particularly LVSP in this case. Early detection and aggressive intervention of Wellens syndrome are also crucial since they may help to lessen the adverse effects.

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Conflict of Interest: None.

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Consent for publication was obtained from the patient for promotion of science.

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