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CASE REPORT OPEN ACCESS

WELLENS' SYNDROME: A CASE REPORT

[1,*]Abdelmajid Bouzerda, [1]Mohamed Sabry, [1]Zouheir Lakhal, [2]Laila Bendriss and [2]Ali Khatouri

[1]Military Hospital of Instruction Mohammed V, Department of Cardiology, Rabat, Morocco
[2]Military Hospital of Avicenne, Department of cardiology, Marrakech, Morocco. University Cadi ayyad, Faculty of Medicine and Pharmacy of Marrakech

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ABSTRACT

Wellens' syndrome is a condition in which electrocardiographic (ECG) changes indicate critical proximal left anterior descending artery (LAD) narrowing occurring during the chest pain-free period. Due to the severity of the obstruction, if such cases are managed by early invasive revascularisation therapy, a major threat in the form of a massive myocardial infarction or sudden death may be averted. We report the case of a 56-year-old patient admitted to an emergency department, because of a thoracic pain suspicious for angina pectoris. Although the patient had become asymptomatic on admission, his electrocardiogram presented abnormalities (T wave inversion in V1 to V6) suggestive of Wellens' syndrome. This was confirmed by an immediate

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INTRODUCTION

Interpretation of electrocardiographic abnormalities is of critical importance to the Emergency Physician (EP). Wellens' syndrome is a pattern of precordial T-wave abnormalities, first described by Wellens and his group in 1982, representing critical left anterior descending (LAD) coronary artery stenosis (de Zwann *et al*., 1982). These electrocardiographic abnormalities alone, regardless of the presence or absence of ongoing chest pain, are an indication for emergent cardiac catheterization. Medical therapy in the absence of revascularization is insufficient in the majority of cases to prevent large anterior wall myocardial infarction (de Zwann *et al*. ,1982). We describe a patient with Wellens' syndrome. In view of the large area of myocardium at risk, the importance of recognizing the significance of this ECG pattern is of critical importance for the emergency physician, especially those involved in the evaluation of patients at emergency department chest pain centers. Wellens' syndrome, the criteria for diagnosis, and a discussion of its implications are presented.

Corresponding author: **Abdelmajid Bouzerda,**
Military Hospital of Instruction Mohammed V, Department of cardiology, Rabat, Morocco.

Case report: A 56-year-old man with no significant medical history presented to the emergency department (ED) after the acute onset of sharp, left-sided chest pain. The pain woke the patient from sleep and was accompanied by shortness of breath. The episode lasted approximately 30 minutes before the patient arrived to the ED. There was no family history of heart disease, and the patient did not smoke cigarettes or use illicit drugs. Vital signs in the ED showed blood pressure of 150/49, heart rate of 70, respiratory rate of 14, and O2 saturation of 100% on room air. Physical examination revealed the patient to be in no acute distress, with a normal cardiopulmonary examination result. There were no other gross abnormalities on the remainder of his physical examination. electrocardiography (ECG) obtained during pain-free interval revealed T-wave inversion in V1 to V6. Progression of R-wave in chest leads, ST segment was isoelectric in the absence pathological Q-waves. This pattern is typical of Wellens' syndrome (Figure1). Interestingly, the patient was free of chest pain since admission. Initial cardiac enzymes were negative, Troponin-T (<0.01 ng/ml), CK-MB 3 ng/ml. Total cholesterol was 174 mg/dl and LDL was 132 mg/dl. Random blood glucose was 110 mg/dl and serum creatinine was 0.7 mg/dl.

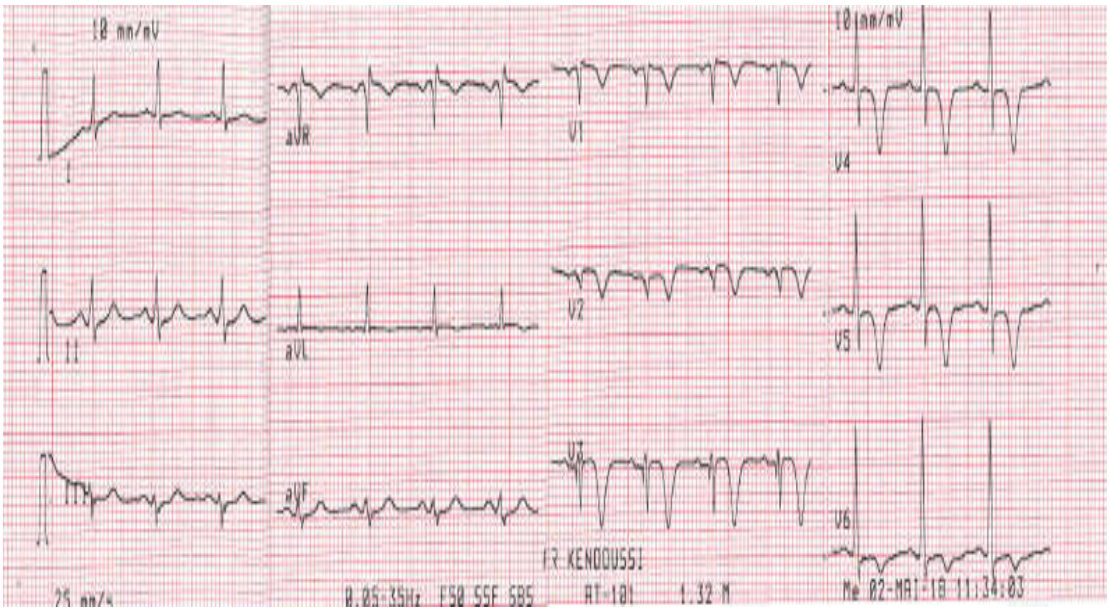

Figure 1. Electrocardiography performed on admission, showing deep, symmetrical T-wave inversion in leads V1 – V6 (typical of type 2 Wellens' syndrome)

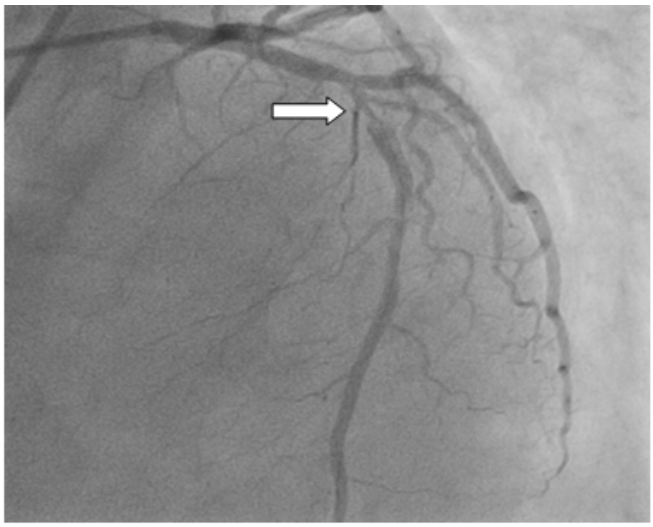

Figure 2. Cardiac catherization with a stenosis of 95–99% at the proximal left anterior descending coronary artery pre-intervention

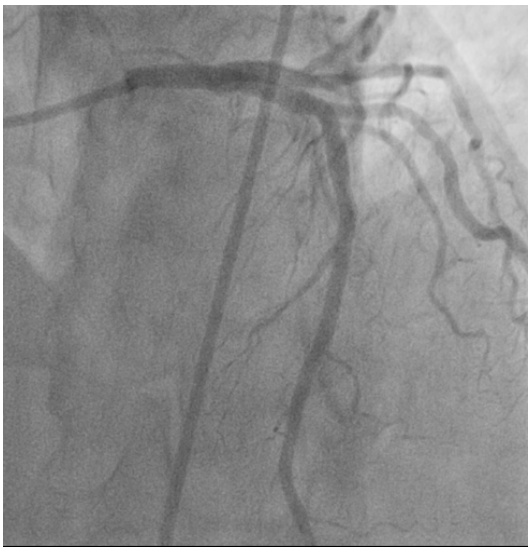

Figure 3. A drug-eluting- stent was placed successfully with improvement of the left anterior descending artery stenosis

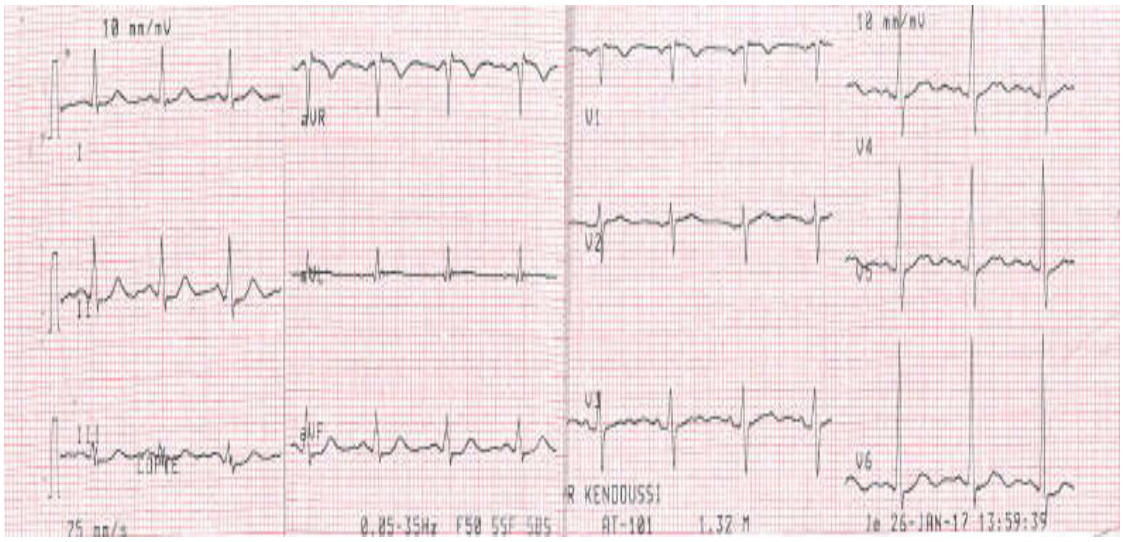

Figure 4. ECG 1 day after stent placement, normalisation T-wave in leads V2-V6.

He was stabilized with the following medications: heparin, clopidogrel, aspirin, tenormin, zocor, and ramipril. A bedside 2D-echocardiography showed normal left ventricular chamber size and function. Left ventricular ejection fraction measured 65% in absence of regional wall motion abnormality. Evaluation of valves and pericardium was unremarkable. Coronary angiography performed 12 hours after presentation showed severe stenos is of 95- 99% in proxiaml left anterior descending artery (Figure 2), which was successfully treated with a drug-eluting stent (Figure 3). ECG 1 day after stent placement, shows normalisation T-wave in leads V2-V6 (Figure 4). The patient did not suffer any post procedure complications and was discharged in stable condition. And a cardiac rehabilitation referral. Six months after his presentation, the patient has resumed daily activities and does not report any further episodes of chest pain.

DISCUSSION

Wellens syndrome, first reported in 1982 by de Zwaan *et al* (de Zwann *et al*., 1982), is typically described by a pattern of characteristic ECG changes implicating significant obstruction and critical stenosis of the proximal LAD (Rhinehard *et al*. ,2002). The criteria for Wellens syndrome are a prior history of angina, minimal or no elevation of cardiac enzymes, minimal or no ST-segment elevation (b1 mm), no loss of precordial R-wave progression, no pathologic precordial Q waves, and biphasic or deeply inverted T waves in leads V2 and V3, but sometimes found in V1, V4, V5, and V6 (de Zwann *et al*., 1989). More specifically, Wellens syndrome can be classified into 2 types. Type 1 Wellens constitutes roughly 24% of cases and is identified by biphasic T waves in leads V2 through V3 (Kardesoglu *et al*., 2003) Such T-wave inversions are recognized by distinctive upsloping followed by a sharp downslope pattern, which is noticeably different from T-wave inversions of other etiologies. The more common type 2 Wellens accounts for the remaining 76% of cases and is identified by deep, symmetrically inverted T waves in precordial leads V2 and V3 (Tandy *et al*. 1999). In the first detection of Wellens syndrome in 1982, de Zwaan *et al* (de Zwann *et al*., 1982) identified a unique ECG pattern in a subgroup of patients with unstable angina who had stenosis of the proximal LAD.

Twenty-six of 145 patients (18%) had the biphasic or deeply inverted T waves predominantly in precordial leads V2 and V3. Moreover, among 16 of the 26 patients who had these findings and also did not undergo coronary revascularization, 12 (75%) ultimately developed extensive anterior wall myocardial infarction within a fewweeks of admission despite symptomatic relief (de Zwann *et al*., 1982). In a second study, 180 (14%) of 1260 patients presenting with unstable angina demonstrated Wellens syndrome; and urgent coronary angiography was performed. Here, all 180 patients were found to have 50% to complete obstruction of the proximal part of the LAD (de Zwann *et al*., 1989). Early detection of Wellens is extremely important because characteristic ECG changes can develop even if the patient is not suffering from angina. Furthermore, the abnormalities of the ST segment/T wave either develop into ST-segment elevation or normalize during chest pain attacks (Rhinehard *et al*., 2002). The mechanism of Wellens' syndrome remains unclear.

It is considered a pre-infarction stage of coronary artery disease as the T-wave changes in the syndrome usually occur during a pain-free interval. However, it is postulated that the changes in the EKG account for reperfusion of the ischemic myocardium due to alleviation of spasm of proximal LAD artery (Sheng *et al*.,). Because Wellens patients are at high risk of myocardial infarction and sudden death, early detection of the distinct Wellens ECG patterns will enable physicians to intervene and help prevent extensive anterior wall infarctions from occurring. For example, physicians in EDs may report patients with biphasic T waves and negative cardiac markers to have "nonspecific" ECG changes. Such diagnoses have resulted in stress imaging tests that induce acute myocardial infarctions (Rhinehard *et al*. ,2002) and delay obtaining themost needed therapies. In clinical practice, stress testing might be performed due to lack of typical ST segment elevation in Wellens' syndrome to obtain more evidence of cardiac ischemia. However, increasing cardiac demand during stress testing may result in acute myocardial infraction, and even fatal dysrhythmia and death (Tandy *et al*., 1999). Therefore, it is essential to consider the coronary angiogram as the initial diagnostic modality instead of other conservative examinations in patients with ECG patterns, indicating a possibility of Wellens' syndrome. Patients with Wellens syndrome are at high risk for suffering an acute myocardial

infarction; and thus, the timely recognition of this presentation can truly be a life-saving diagnosis.

Conclusion

Wellens' sign is an important ECG pattern for clinicians to recognize and may occur even in asymptomatic patients. Clinicians should be well aware about the subtle y*et al*arming ECG changes of Wellens' syndrome and its association with critical left anterior descending coronary artery stenosis. It is a pre-infarction stage and may progress to an ill-fated myocardial infarction when unintervened. Thus it requires an early invasive approach with coronary angiography in view to consider revascularization. Patients with Wellens' syndrome should not be subjected to stress test.

Consent: Written informed consent was obtained from the patient for publication of this case report and any accompanying images

Competing Interest: The authors declare that they have no competing interests.

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