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A Classic Pattern of Wellens Syndrome on ECG: A Case Report

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A Classic Pattern of Wellens Syndrome on ECG: A Case Report

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Abstract

Wellens syndrome is usually diagnosed in asymptomatic patients with normal or only slightly elevated cardiac enzymes. There are two different ECG patterns (Type A and Type B) described in the literature. Earlier studies demonstrated that the appearance of the Wellens pattern had a specificity of 89% and a positive predictive value of 86% for severe stenosis of the left anterior descending artery (LAD) hence a timely recognition and therapeutic approach may prevent fatal outcomes in the patients.

Here we are presenting a case of a 69-year-old gentleman with chest pain and Type A Wellens Syndrome pattern on ECG who was found to have LAD stenosis.

Keywords: Wellens syndrome, Coronary artery disease, Left anterior descending artery, Acute coronary syndrome

1. Introduction

Wellens syndrome was initially described in the 1980s among a group of patients presenting with unstable angina. The patient usually presents with normal or only slightly elevated cardiac enzymes including troponin, which may be falsely reassuring in patients with Wellens syndrome. Furthermore, ECG changes can develop over days to weeks, often when the patient is asymptomatic representing a diagnostic challenge. Timely recognition is imperative since it results from a temporary LAD obstruction, representing a pre-infarction state with a high risk of progression to an extensive myocardial infection due to the unstable nature of the coronary perfusion.[1] These patients require invasive intervention since medical management alone is associated with an increased risk of developing myocardial infarction.

There are two different ECG patterns described in the literature. The first pattern (Type A), which is seen in approximately 24% of the patients, is characterized by biphasic T waves in precordial lead V2 and V3. The second pattern (Type B), which is present in around 76% of the patients, is recognized by deep, and symmetrically inverted T waves in the anterior leads.[2]

Here, we are presenting a rare case of a patient who presented with symptomatic chest pain and was found to have a Type A Wellens Syndrome pattern on ECG.

2. Case presentation

This is a 69-year-old gentleman with a past medical history of coronary artery disease status post two stents placement, the first one in 2012 and the second one in 2016, who presented to the Emergency Room on 01/06/2022, complaining of chest pain for three days before admission. The patient complained of pressure-like, intermittent chest pain, 8–10 in intensity, located on the left-sided chest and radiated to the left arm and shoulder. Each episode had a duration of 30 min with 5 min between each episode. Initially, the pain was

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exacerbated upon physical activity and alleviated with rest, however, a few hours before presentation to the Emergency Room, the patient also experienced pain at rest.

He had not been compliant with his medication regimen for the last two years before the presentation and he chose to treat his medical conditions exclusively with ginger.

Pertinent negatives included diaphoresis, orthopnea, paroxysmal nocturnal dyspnea, lower extremity edema, dizziness, and syncopal episodes. His family history was remarkable for his father with cardiac disease status post stents placement.

In the Emergency Room, the patient was found to be hypertensive with a blood pressure of 154/83 mmHg, non-tachycardic or tachypneic, and saturating 97% on room air. The initial troponin I level was 0.12 [<0.5 ng/ml], but the follow-up troponin I was 0.51 [<0.5 ng/ml]. Initial ECG showed sinus rhythm, first-degree AV block, incomplete right bundle branch block, biphasic T-wave inversion in V2 and V3, and T-wave inversion in anterolateral leads.

In the setting of the patient's clinical presentation associated with troponin leakage and ECG changes concerning for ischemia, he was started on full acute coronary syndrome (ACS) protocol including loading dose of aspirin, and clopidogrel, therapeutic anticoagulation, and statin therapy along with monitoring ECG changes and troponin I levels.

The following day, the patient remained hemodynamically stable, and the chest pain was resolved. However, the repeat troponin I level was 0.80 [<0.5 ng/ml], and follow-up ECG showed sinus bradycardia with a heart rate of 55 beats per min with first-degree A-V block, incomplete right bundle branch block, and biphasic T waves in V2 and V3 which in that clinical setting was consistent with Type-A T waves seen in Wellens syndrome (Fig. 1). An echocardiogram was also performed, and it revealed a left ventricular ejection fraction of 50–55% and mildly hypokinetic changes on the apical septal and apical anterior segments.

The patient was taken to the catheterization laboratory for left heart catheterization, coronary angiography, and left ventriculography. During the intervention, it was found that the LAD had a proximal to mid stent and a distal stent in place. There was proximal 90% in-stent restenosis at the proximal edge of the first stent. There was also a 90% in-stent restenosis at the proximal edge of the second stent into the mid-segment, which was a tubular lesion. Also, there was a distal 70% discrete stenosis in the distal LAD.

Because of the above findings, a total of three stents were placed. The first stent was placed in the mid to distal LAD to cover the second 90% lesion. The second stent was placed to cover the proximal lesion and was placed at the proximal edge of the previous stent. A third stent was placed in the distal LAD at the level of the distal 70% discrete stenosis. The repeat angiographic shots showed excellent results with no residual angiographic stenosis and no dissection, perforation, or complications.

Further recommendations included aggressive guideline-directed medical therapy for coronary artery disease, including dual-antiplatelet therapy with aspirin and clopidogrel for minimally one year, a beta-blocker, and a statin. Furthermore, the patient was counseled extensively on medication compliance after stent placement.

3. Discussion

Wellens syndrome is a pattern of ECG changes characterized by biphasic or deeply inverted T waves in leads V2–V3. When present, it suggests an ischemic state from severe stenosis of the proximal left anterior descending artery (LAD).[3] The pattern was first described in 1979 as inverted U-waves, but then in the 1980s consensus shifted towards an inverted or biphasic T wave.[3,4] Many previous studies characterizing ischemia-induced inverted U-waves are referring to the same syndrome.[5] The significance of Wellens syndrome is its diagnostic value, as the pattern is usually seen with patients in an asymptomatic and painless phase of myocardial ischemia while suggesting a poor prognosis, as patients tend to be at high risk of anterior wall myocardial infarctions and other chronic ischemic events due to high-grade stenosis. Many also have poor collateral circulation along with the LAD.[3,6] These patients also have a poor prognosis with medical management alone. The other criteria for Wellens syndrome include a history of angina, normal R wave progression in precordial leads, lack of ST-segment elevation, lack of Q waves, and overall negative serum marker elevation in labs.[6]

There are 2 variations of Wellens syndrome. Type A is generally seen in 24% of patients and shows biphasic T-waves in V2 and V3, with an initial positive deflection and then ending with a negative deflection.[8,9] Type B is the more common presentation and is demonstrated in 76% of patients. It shows the deep and symmetrical inversion of the T wave in leads V2 and V3. While V2 and V3 are the major leads where the inversions will present, they can also appear in V1 and V4 in Type B, with V1 being involved in about 66% of patients and V4 in

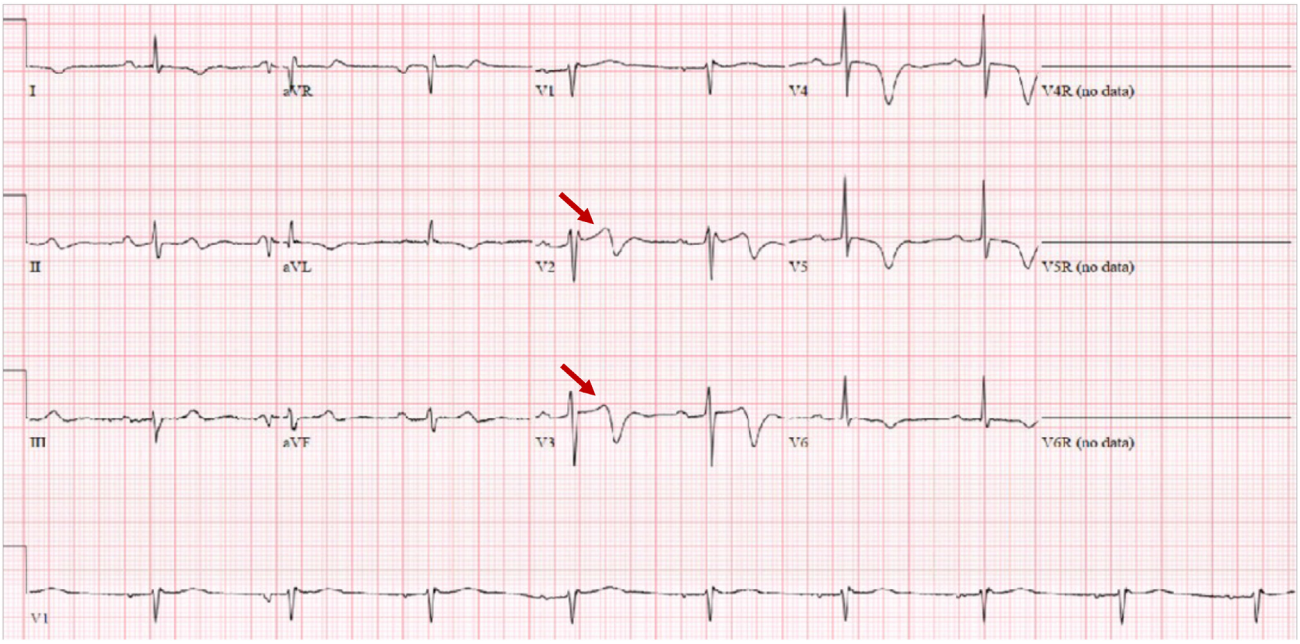

Fig. 1. EKG with arrows pointing toward biphasic T waves in V2 and V3 consistent with Type-A pattern in Wellens syndrome.

approximately 75%.[9] Inversions can also be present in V5 and V6 in rare cases. Once intervention for the severe stenosis is performed, the pattern may remain for hours to weeks, but then normalize over time.[6] The pattern may also be seen in patients with prior acute coronary syndrome and follows the appearance of ST-segment elevations in the natural ECG progression.

Thorough ECG interpretation is needed when assessing for the diagnosis of Wellens syndrome as there are many differentials for inverted or biphasic T-wave morphology. When assessing biphasic T waves, those that begin with a negative deflection and end in a positive deflection suggest a hypokalemic etiology. T wave inversions in multiple right precordial leads and inferior limb leads are specific for pulmonary embolism, particularly leads V1 and III.[10] Likewise, patients with severe LVH may also demonstrate a "Pseudo-Wellens" pattern with biphasic or inverted T-waves in multiple precordial leads, particularly in V5 or V6.[11] If seen in these patients, it is more likely a repolarization abnormality in patients with left heart strain, rather than critical stenosis of the LAD. T wave inversions in V1–V4 accompanied by QTc prolongation may also be a sign of Takotsubo syndrome, which can mimic ACS in clinical presentation.[12]

The diagnostic value of Wellens syndrome cannot be understated. Patients are usually asymptomatic, with negative to mildly elevated serum marker abnormalities, and have a general lack of other findings suggestive of acute coronary syndrome on ECG. Earlier studies demonstrated that the appearance of the Wellens pattern had a specificity of 89% and a positive predictive value of 86% for severe stenosis of the LAD.[3] This study also demonstrated that in 2 patients with the T-wave inversions in inferior leads, severe right coronary artery (RCA) disease was noted, suggesting that the pattern could be utilized in other scenarios if the proper criteria were met. These individuals must undergo revascularization via cardiac catheterization, as up to 75% of patients with Wellens syndrome managed with medical therapy alone develop anterior wall infarction.[7]

4. Conclusion

ECG changes can be very subtle, and they are often interpreted as non-specific T-wave changes by the computer and the reading physician. Timely recognition and therapeutic approach may prevent fatal outcomes in the patients since this pattern has a high specificity and positive predictive value for severe stenosis of the LAD.

A high degree of suspicion is warranted in asymptomatic patients, with normal or minimal elevated cardiac markers, and subtle T wave changes in precordial leads for preventing significant morbidity and mortality. Promptly after the diagnosis is made, patients should undergo revascularization via cardiac catheterization due to the

high risk of developing myocardial infarction in patients with Wellens syndrome.

Conflicts of Interest

There is no conflict of interest.

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