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CASE REPORT

Rare Wellen's Rare Presentation

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ABSTRACT

Wellens syndrome is a distinct electrocardiographic (ECG) finding that reflects critical narrowing of the proximal left anterior descending (LAD) coronary artery. First identified in 1982, this pattern typically appears in patients with a history of unstable angina, especially during pain-free intervals. Recognizing this syndrome is vital, as it signals a high risk for imminent anterior wall myocardial infarction if not treated promptly. The syndrome is classified into two types based on T wave morphology. Type A, which features biphasic T waves most prominently in leads V2 and V3, can be particularly challenging to identify. These subtle ECG changes are often overlooked, especially when the patient is not actively experiencing chest pain, which increases the risk of misdiagnosis or delayed treatment. Such oversight can lead to rapid deterioration and serious cardiac complications, including extensive myocardial infarction. In this case report, we present a patient with type A Wellens syndrome, where the early ECG showed only mild biphasic T wave abnormalities that were initially missed. This diagnostic oversight delayed appropriate cardiologic intervention and emphasized the importance of vigilance in evaluating atypical or transient ECG changes. Early identification of Wellens syndrome, even in asymptomatic phases, is critical to guiding appropriate management and preventing life-threatening outcomes. Clinicians, particularly in emergency medicine settings, should be aware of these subtle yet significant ECG markers.

KEYWORDS

• Wellens syndrome • Myocardial infarction • Vigilance • Abnormalities

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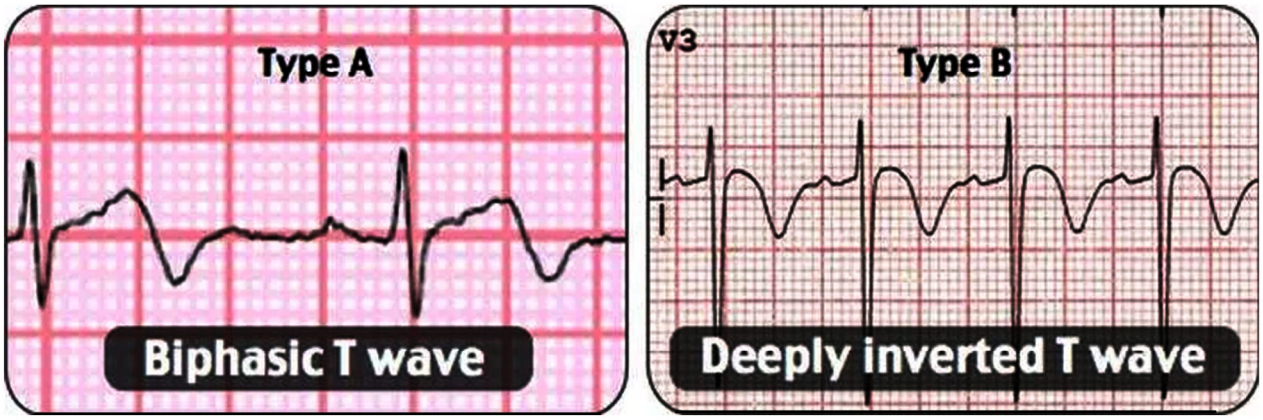
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INTRODUCTION

Wellens syndrome, also referred to as LAD T-wave inversion syndrome, is a distinct electrocardiographic (ECG) pattern that strongly suggests significant stenosis of the proximal left anterior descending (LAD) coronary artery.[1] It was first reported in 1982 in a group of patients presenting with unstable angina. The importance of this ECG finding lies in its association with a high risk of anterior wall myocardial infarction if not promptly recognized and managed.[1,2]

There are two recognized ECG patterns in Wellens syndrome (image).



Type A: Biphasic T waves, a rare entity, highly critical; **Type B:** Deeply inverted T waves, commonly seen, less critical than type A (***Source: Image Courtesy:*** ROSH Review of Emergency Medicine, Google Search Engine).

Type A, though less commonly observed, is more specific and is characterized by biphasic T waves primarily in leads V2 and V3.[2] Type B is more frequent, presenting as deep, symmetrical T wave inversions in the anterior precordial leads. While Type B is relatively easier to identify due to its pronounced nature, Type A poses a diagnostic challenge because of its subtle appearance.[3]

Timely recognition of these ECG patterns particularly Type A is essential for preventing adverse outcomes. These changes often occur during pain-free periods and may be mistaken for nonspecific repolarization abnormalities, especially in patients without conventional cardiovascular risk factors. Failure to recognize Wellens syndrome may delay appropriate management, increasing the likelihood of significant myocardial damage.[4]

In this case we discuss a young patient with no known coronary artery disease risk factors presented with very subtle Type A Wellens ECG changes. These findings were initially overlooked, leading to a delay in accurate diagnosis and treatment. This case underscores the need for heightened clinical suspicion and careful ECG interpretation, even in low-risk individuals, to prevent potentially life-threatening cardiac events.

CASE

A 35-year-old male presented to the Emergency Department (ED) of our hospital with complaints of retrosternal chest pain radiating to his left side of jaw with associated palpitations, nausea and diaphoresis lasting for few minutes with spontaneous resolution since last 1 week. The patient was seen by two local doctors and was advised anxiolytics, but his symptoms did not improve. His initial vitals in the ED were: pulse rate – 74/min, blood pressure – 130/80 mmHg, Oxygen saturation – 98% on room air, Random Blood Sugar – 105 mg/dL, GCS – E4V5M6 (15/15), pain score (out of 10, 10 being the highest).[2]

The patient was physically active without any underlying co-morbidities. The patient did not have any significant past medical history. His physical examination on presentation was unremarkable. His electrocardiogram (ECG) showed (image).

The first-set of troponin level was 0.04 ng/mL and the subsequent troponin levels were undetectable over the next 4 hours. The patient was given tablet ecospirin (aspirin) 325 mg, tablet atorvastatin 80mg, tablet clopidogrel 300mg and sublingual sorbitrate 5mg in the ED. He had mild improvement after the medications. Suddenly the patient developed bradycardia ranging from 38-45/min with

normal sinus rhythm in the monitor. It was decided to admit the patient for overnight monitoring. His repeat ECG was same. A quick 2D-echocardiography was performed bedside and revealed an estimated ejection fraction of 50–55% with no wall motion abnormalities.

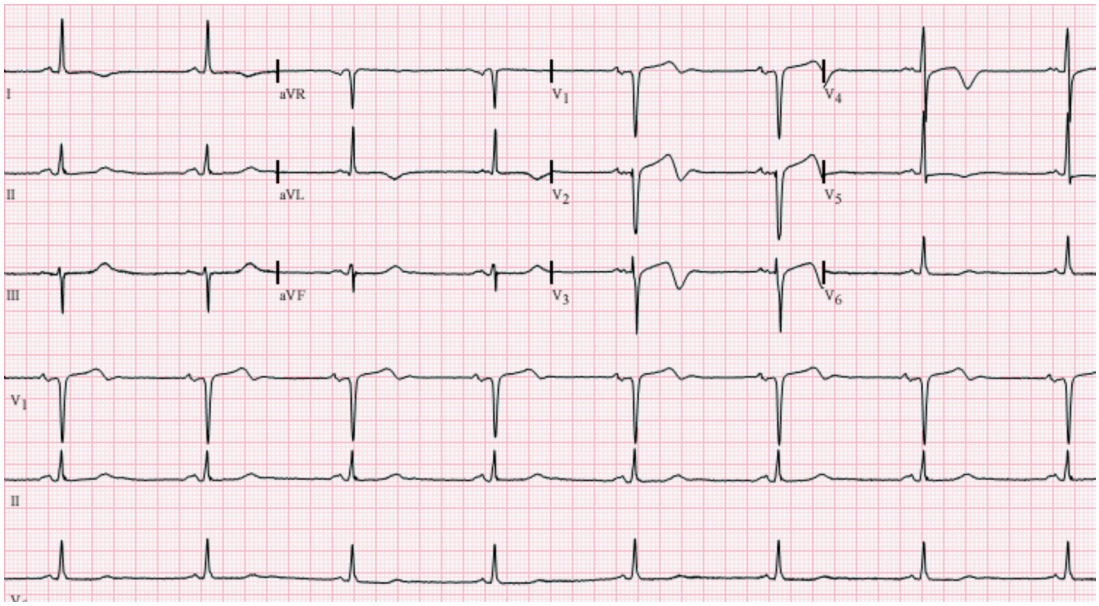


Image Courtesy: Department of Emergency Medicine, Max SSH, SHBG.

*Note the changes in lead V1-V3

After 3 hours, on further discussion with the emergency team and cardiology team and taking references from the emergency and cardiology textbooks, it was noticed that the patient ECG was having subtle abnormality. An urgent cardiology consultation was done, who ordered a stress test for the patient. The patient underwent cardiac stress testing, during which he had recurrence of his symptoms and developed 5.5mm ST-elevation in the anteroseptal leads. The test was immediately terminated and the patient was urgently shifted to the cath lab.

Emergency coronary angiography was done which showed 95% obstruction in the proximal LAD that was successfully treated with a Drug-Eluting Stent (DES) (image).

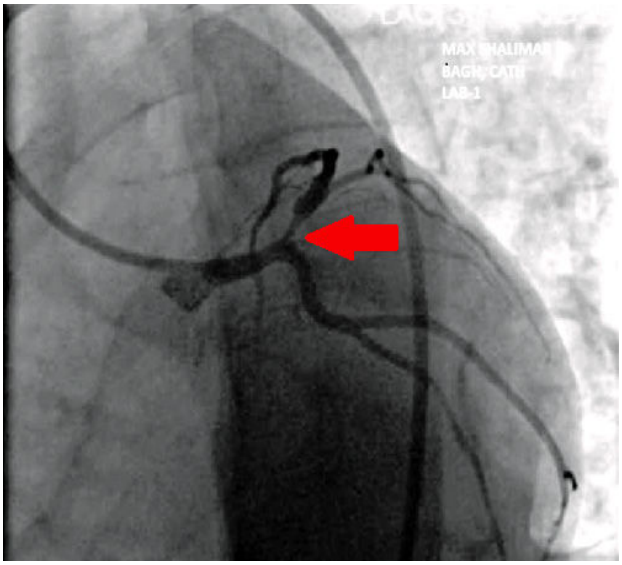


Image Courtesy: Department of Cardiac Science, Max SSH, SHBG. Arrow mark indicating the occuldad LAD territory in the coronary vessel

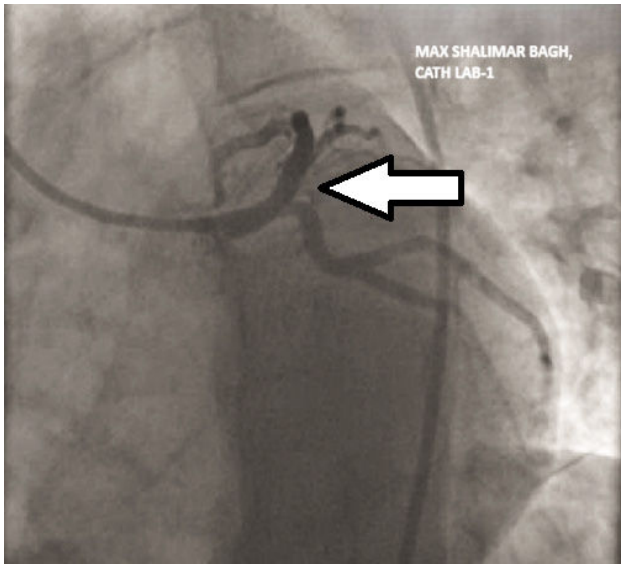


Image Courtesy: Department of Cardiac Science, Max SSH, SHBG. Arrow mark indicating the opened LAD territory in the coronary vessel after placing the DES

The patient was discharged home on optimal medical therapy after 3 days of hospitalization. The post discharge follow-up's were asymptomatic at regular intervals for next 6 months and the patient recovered well from this hidden underlying g rare but critical clinical entity.

DISCUSSION

Electrocardiogram (ECG) interpretation remains a cornerstone in the initial evaluation of patients presenting with suspected myocardial ischemia, following a thorough clinical history and physical examination. The ability to identify ECG patterns indicative of ongoing or imminent cardiac ischemia is critical in initiating timely and appropriate intervention.[5] Among these patterns, Wellens syndrome is particularly important, as it reflects a pre-infarction state related to significant stenosis of the proximal left anterior descending (LAD) artery.

First characterized by Wellens and colleagues in the 1980s, the syndrome was identified in approximately 14–18% of patients admitted with unstable angina in their original studies.[6] Alarmingly, 100% of these patients had significant LAD lesions, and those who did not undergo timely revascularization frequently developed massive anterior myocardial infarctions.[6] In contrast, more recent studies suggest a lower prevalence of Wellens patterns in patients with non-ST elevation myocardial infarction (NSTEMI), with some cases lacking a definitive LAD culprit lesion.[7]

The classical diagnostic criteria for Wellens syndrome include a history of transient chest discomfort, normal or minimally elevated cardiac enzymes, and specific ECG changes. These changes typically involve isoelectric or slightly elevated ST segments, absence of significant Q waves in precordial leads, and either biphasic or deep, symmetrical T wave inversions in leads V2 and V3.[2,5,7] These are categorized as type A and type B patterns, respectively, with some confusion in terminology across different case reports. For consistency, the original classification by Wellens biphasic T waves as type A and inverted T waves as type B is preferred.

A unique feature of Wellens syndrome is that its characteristic ECG changes typically manifest during asymptomatic periods.[8] This poses a diagnostic challenge but also presents an opportunity: early recognition can lead to life-saving interventions. Misinterpreting these subtle changes or proceeding with stress testing instead of immediate angiographic evaluation can provoke severe complications, including ventricular arrhythmias or full-blown myocardial infarction.[9] Therefore, when Wellens syndrome is suspected, urgent coronary angiography should be prioritized over provocative testing.

In this presented case, there were dangers of underrecognizing Wellens syndrome. Subtle T wave changes were missed by the emergency team initially, leading to delay in the final treatment commencement. Fortunately, the patient did not experience a catastrophic outcome, but the situation illustrates the necessity of heightened vigilance and education regarding this ECG pattern.

Moreover, clinicians must recognize that Wellens-like ECG changes can occasionally be observed in non-ischemic contexts commonly referred to as pseudo-Wellens syndrome.[10] Conditions such as congenital myocardial bridges, stress-induced cardiomyopathy (Tako-Tsubo), acute abdominal pathology, and the use of certain substances (e.g., cocaine or cannabis) have been linked to these mimic patterns.[10] In such cases, myocardial oedema detected by cardiac MRI may play a role, suggesting that anti-inflammatory therapies could be beneficial, though more research is needed to explore this hypothesis.[11]

In conclusion, the accurate and prompt identification of Wellens syndrome is critical in preventing adverse cardiac events. All healthcare providers, especially emergency and intensivist, should be adept at recognizing even the most nuanced ECG signs of this condition and respond with appropriate, often invasive, diagnostic measures.

CONCLUSION

Wellens syndrome represents a critical warning sign of significant stenosis in the proximal left anterior descending (LAD) coronary artery and indicates a high risk for impending anterior myocardial infarction. Early recognition of its characteristic ECG patterns especially during asymptomatic periods is essential to avoid serious complications such as ventricular arrhythmias, extensive myocardial damage, or

even sudden cardiac death. Misinterpretation or delayed diagnosis may lead to inappropriate management, such as stress testing, which can provoke life-threatening events. Therefore, prompt referral for coronary angiography is crucial when Wellens syndrome is suspected. Healthcare professionals, particularly those in emergency and intensivists, should maintain a high index of suspicion for Wellens ECG patterns, even when clinical symptoms are minimal or absent. Additionally, awareness of pseudo-Wellens presentations due to non-ischemic causes is important to avoid unnecessary interventions. Overall, early detection and appropriate management can significantly reduce morbidity and mortality associated with this high-risk cardiac condition.

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