



# WELLENS' SYNDROME: A DANGEROUS HEART INFREQUENCY

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## SUMMARY

*Wellens syndrome is an acute coronary syndrome with a high risk of life-threatening myocardial ischaemic disease. It should be emphasised that it is not always an acute process, and may manifest itself over periods of weeks and result in an Impending Acute Myocardial Infarction of the anterior wall.*

*The aim of this research lies in the importance of detecting this pathology in a timely manner, because, although it is infrequent, it has a high mortality rate in patients who present it and do not have an early detection.*

**KEY WORDS:** *wellens syndrome. inverted t-waves. coronary revascularisation. acute myocardial infarction.*

## INTRODUCTION

Wellens syndrome is the presence of chest pain, plus T wave changes in the precordial leads; minimal or no changes in markers of myocardial damage, absence of Q waves and R wave progression in the precordial leads; along with minimal

or no ST segment elevation.

Briefly, electrocardiographic changes can manifest, even without chest pain; in addition, there are 2 patterns in the electrocardiogram that can be identified: the most frequent is the presence of negative T waves, known as type 1, with a



frequency of 76%, and evident in V2 - V3 in most cases of Wellens Syndrome; the other electrocardiographic pattern is type 2, where isodiphasic T waves are found<sup>(1)(2)</sup>. This syndrome correlates with severe proximal stenosis of the Anterior Descending Artery.<sup>(3)(4)(5)</sup>

This pathology is an antecedent period of Acute Myocardial Infarction associated with obstructions in the Left Anterior Descending Artery, in the proximal segment<sup>(6)(7)</sup>.

The causes of Wellens syndrome are similar to any condition that causes heart disease, such as:

- Atherosclerotic plaque
- Coronary artery vasospasm
- Hypoxia
- Increased cardiac demand<sup>(8)</sup>.

The risk factors for Wellens Syndrome are identical to those for acute coronary syndrome, such as: diabetes mellitus, hypertension, advanced age and family history of coronary heart disease, hypercholesterolemia, hyperlipidaemia and metabolic syndrome<sup>(9)(10)</sup>.

### DEVELOPMENT OF THE DISEASE

Wellens syndrome results from a temporary obstruction of the anterior descending coronary artery, usually caused by rupture of an atherosclerotic plaque leading to occlusion of the anterior descending coronary artery, with subsequent clot lysis or other disruption of the occlusion before a complete myocardial infarction has occurred. The electrocardiogram changes in Wellens syndrome are presumed to be generated by coronary artery spasm. Another hypothesis is caused by repetitive transmural ischaemia-reperfusion leading to myocardial oedema.

The spectrum of this pathology is very broad, and knowledge and high clinical suspicion for its diagnosis, especially in its rare biphasic T-wave presentations, is the key to avoid catastrophic consequences. Criteria for diagnosis are: previous history of chest pain, no elevation of cardiac markers or elevation 2 times the normal limit of reference values, isoelectric or slightly elevated ST segment, no precordial Q waves, no loss of R waves in the precordial leads and symmetrically inverted T waves or, less commonly, biphasic T waves in the precordial leads<sup>(1)</sup>.

### DIAGNOSIS AND THERAPEUTIC MANAGEMENT

The clinical manifestations may resemble unstable angina, accompanied by dyspnoea, diaphoresis, nausea/vomiting, presyncope/syncope; however, the likelihood of this pathology increases when the patient has cardiovascular risk factors<sup>(12)</sup>. In the electrocardiographic pattern, T-wave abnormalities may be evident in the anterior leads, inverted, deep and symmetrical in leads V2 and V3; although occasionally it may be found in leads V1, V4, V5 and V6, or biphasic T-waves in leads V2 and V3 and slightly elevated or isoelectric ST segment<sup>(13)</sup>. Early diagnosis and intervention leads to good outcomes<sup>(14)(15)(16)</sup>.

Pharmacological management of this pathology is not sufficient, as 75% of patients with this syndrome will develop an acute anterior wall myocardial infarction. An important point is not to take the patient to a stress test, as this may lead to an acute anterior wall myocardial infarction<sup>(17)</sup>.

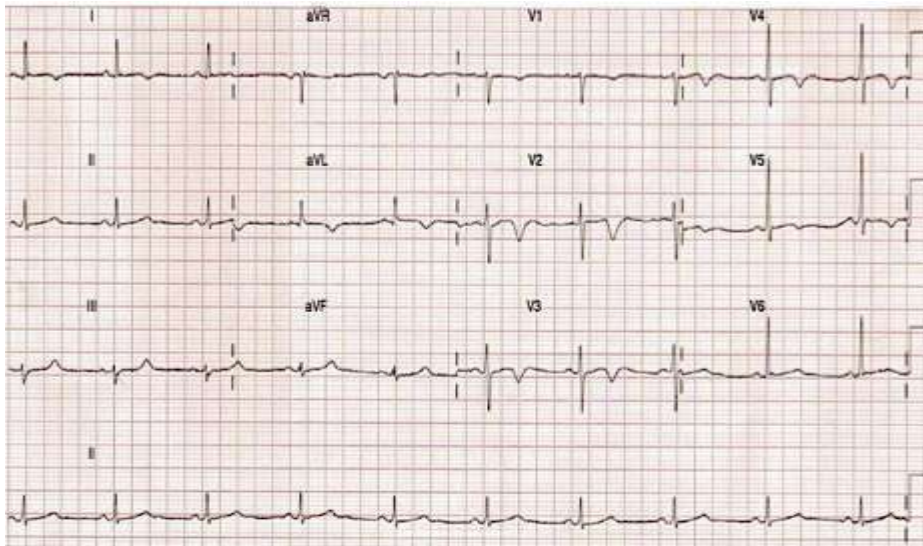
Once this syndrome is recognised, general measures such as the use of nitrates, beta-blockers and the use of antiplatelet therapy with P2Y12 receptor inhibitors should be initiated urgently, coordinated with Cardiology for treatment with invasive coronary angiography and followed by coronary revascularisation. Although T-wave abnormalities in patients with Wellens syndrome are seen in asymptomatic patients, and although they respond adequately to pharmacological treatment, they ultimately require revascularisation. If significant occlusion of the anterior descending artery is evident, percutaneous coronary intervention or coronary artery bypass surgery should be performed to prevent acute anterior myocardial infarction. When treated with pharmacological therapy alone, 75% of patients with this syndrome develop anterior wall infarction within one week<sup>(18)(19)</sup>.

### CONCLUSIONS

This pathology has a significant impact on patients with the presence of comorbidities, and also has an anterior presentation with not so subtle electrocardiographic changes, so it is imperative to consider the admission of patients with the presence of unstable angina and the performance of an electrocardiogram, since with this, the clinical manifestations and the presence of comorbidities, this pathology can be diagnosed and treated in a timely manner; therefore, both clinical and electrocardiographic diagnosis will define early diagnosis and appropriate and timely management.

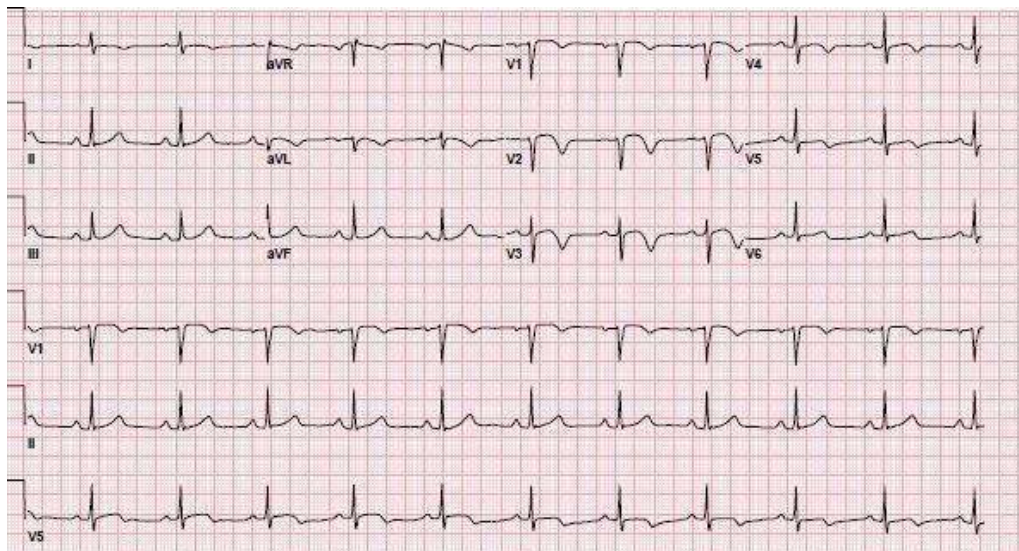
## IMAGES TO CONSIDER

Electrocardiogram of Wellens syndrome, type 1, with T wave inversion in V2, V3, V4 and V5.



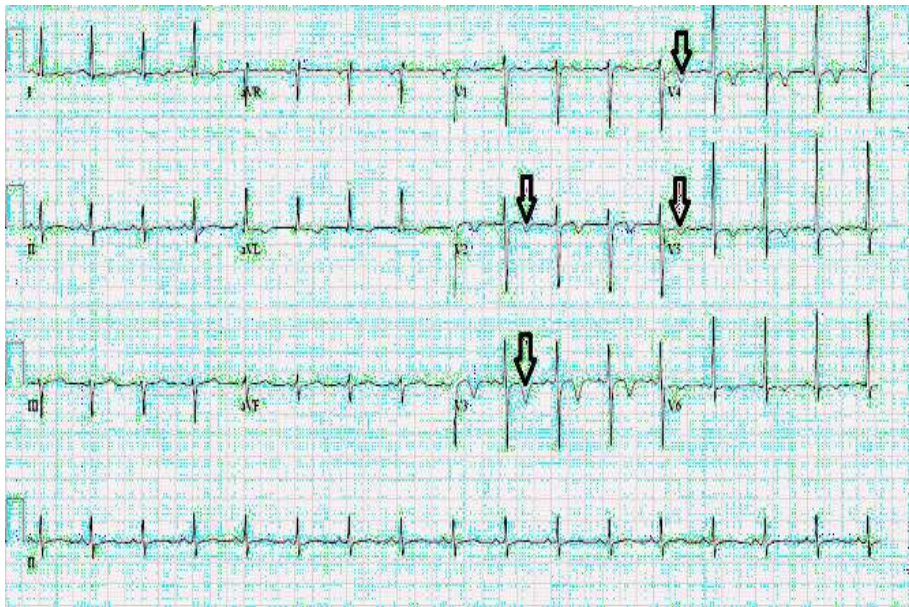
Source: Martínez-Losas, Viana-Tejedor, Freitas-Ferraz, & Ruiz-Mateos. Wellens syndrome (1)

Figure 1. Electrocardiogram demonstrating the Wellens Syndrome pattern in leads V1 - V6, with greater depth in V2 and V3.



Source: Miner B, Hart EH. StatPearls Publishing (8).

Figure 2. Electrocardiogram with demonstration of T-wave inversion from V2 to V5.



Source: Udechukwu N, Shrestha P, Zeeshan M, Donato A. Wellens' syndrome: a close call (20).

## FINAL STATEMENT

This review is based on an article by Santiago Vintimilla called “Síndrome de Wellens, una infrecuencia de peligro”, whose author authorized the translation and rewriting from the Spanish language version to the English language version.

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