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## **WELLENS SYNDROME: THE “CHAMELEON” IN THE ELECTROCARDIOGRAPHIC DIAGNOSIS OF CORONARY SYNDROME. CASE REPORT**

**SÍNDROME DE WELLENS: EL “CAMALEÓN” EN EL  
DIAGNÓSTICO ELECTROCARDIOGRÁFICO DEL  
SÍNDROME CORONARIO. REPORTE DE CASO**

**Marcos Ivan Tienda Pimentel Msc**

Dep. Emergency of Playa del Carmen General Hospital, Quintana Roo, México.

**Sergio Esqueda Vargas**

Dep. Emergency of Playa del Carmen General Hospital, Quintana Roo, México.

**Frida Denisse Álvarez Ríos**

Private practice

**Graciela Guadalupe Andrade Vázquez**

Private practice

## Wellens Syndrome: The “Chameleon” in the Electrocardiographic Diagnosis of Coronary Syndrome. Case Report

**Marcos Ivan Tienda Pimentel Msc<sup>1</sup>**

[marcos.711@hotmail.com](mailto:marcos.711@hotmail.com)

Dep. Emergency of Playa del Carmen General  
Hospital, Quintana Roo, México.  
Universidad Autónoma de Yucatán

**Sergio Esqueda Vargas**

Dep. Emergency of Playa del Carmen General  
Hospital, Quintana Roo, México.

**Frida Denisse Álvarez Ríos**

Private practice

**Graciela Guadalupe Andrade Vázquez**

Private practice

### ABSTRACT

**Objective:** To describe the electrocardiographic characteristics and clinical relevance of Wellens syndrome, a condition associated with critical proximal stenosis of the anterior descending artery, through a clinical case. **Material and Methods:** A systematic literature review was conducted in 20 high-evidence articles published since 2009. Additionally, we report the case of a 70-year-old male with cardiovascular risk factors and clinical symptoms consistent with Wellens syndrome. **Results:** The patient presented with chest pain, deeply inverted T waves in precordial leads (V1–V5), and minimal elevation of cardiac enzymes. Coronary angiography confirmed a critical lesion in the anterior descending artery. Immediate percutaneous coronary intervention was performed with favorable evolution. **Discussion:** The author of this next recommended performing a rapid analysis that includes the chronic history of the patient, as well as the current symptoms, without ignoring the paraclinics of the patient under study, without ruling out the possibilities of this syndrome, before a normal electrocardiogram but with chronic symptoms. **Conclusions:** Wellens syndrome remains a diagnostic and therapeutic challenge. Early identification of its ECG pattern is crucial for timely intervention, which significantly reduces the risk of massive anterior myocardial infarction. Physician training and awareness are key to improving clinical outcomes.

**Keywords:** wellens syndrome, electrocardiogram, coronary angiography, myocardial infarction, percutaneous coronary intervention

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<sup>1</sup> Lead author

Correspondence: [marcos.711@hotmail.com](mailto:marcos.711@hotmail.com)

# Síndrome de Wellens: El “Camaleón” en el Diagnóstico Electrocardiográfico del Síndrome Coronario. Reporte de Caso

## RESUMEN

**Objetivo:** Describir las características electrocardiográficas y la relevancia clínica del síndrome de Wellens, una condición asociada con la estenosis crítica proximal de la arteria descendente anterior, a través de un caso clínico. **Material y Métodos:** Se realizó una revisión sistemática de la literatura en 20 artículos de alta evidencia publicados desde 2009. Además, se reporta el caso de un paciente masculino de 70 años con factores de riesgo cardiovascular y síntomas clínicos consistentes con el síndrome de Wellens. **Resultados:** El paciente presentó dolor torácico, ondas T invertidas de forma profunda en las derivaciones precordiales (V1–V5) y una mínima elevación de las enzimas cardíacas. La angiografía coronaria confirmó una lesión crítica en la arteria descendente anterior. Se realizó una intervención coronaria percutánea inmediata con evolución favorable. **Discusión:** El autor de este artículo recomienda realizar un análisis rápido que incluya la historia clínica del paciente, así como los síntomas actuales, sin ignorar los estudios paraclínicos del paciente en cuestión, sin descartar la posibilidad de este síndrome, incluso si el electrocardiograma es normal pero con síntomas crónicos. **Conclusiones:** El síndrome de Wellens sigue siendo un desafío diagnóstico y terapéutico. La identificación temprana de su patrón electrocardiográfico es crucial para una intervención oportuna, lo que reduce significativamente el riesgo de un infarto de miocardio anterior masivo. La formación de los médicos y su conciencia sobre esta condición son clave para mejorar los resultados clínicos.

**Palabras clave:** síndrome de Wellens, electrocardiograma, angiografía coronaria, infarto de miocardio, intervención coronaria percutánea

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## INTRODUCTION

The syndrome was first identified in 1982 by Hein J. Wellens and his team in patients who had unstable angina. Currently, it is recognized as a clinical entity well defined that usually indicates the presence of a critical stenosis in the proximal segment of the artery left anterior descending (AID), which implies a high risk of imminent myocardial infarction and demands urgent medical intervention.<sup>1</sup>

This pathology is a rare presentation of acute coronary syndrome (ACS) that indicates a critical obstruction in the left anterior descending artery. It is recognized by certain patterns specific in the T waves of the electrocardiogram (ECG), described below. However, it cannot be detected by some doctors due to ignorance of these characteristic findings in the ECG. <sup>2</sup>

Wellens syndrome is usually caused by a transient obstruction in the artery left anterior descending (AD), caused by the rupture of an atherosclerotic plaque that leads to its block. This can trigger the dissolution of the clot or other changes that reduce in a way critical blood flow, which can culminate in a massive infarction of the anterior wall of the myocardium. The risk factors associated with this syndrome are the same as those of the common coronary disease, such as dyslipidemia, high blood pressure, diabetes, physical inactivity, obesity, tobacco use and metabolic syndrome.<sup>3</sup>

Epidemiologically, it responds approximately to 14–18% of cases of unstable angina and is characterized in the electrocardiogram (ECG) by two distinctive patterns:

- Type 1: biphasic T waves, most frequently observed in the V2 and V3 leads (Present in 24% of cases).
- Type 2: deeply inverted and symmetrical T waves, mainly in V1 to V4, being thiis the most frequent pattern. <sup>4</sup>

The diagnosis is usually a clinical challenge because the symptoms usually correspond to a picture of acute coronary syndrome, characterized by oppressive chest pain, with possible



Irradiation to the neck, jaw or shoulder. However, at the time of the evaluation in the emergency service, patients are often asymptomatic. The physical exam it can be non-specific or reveal subtle signs such as mild diaphoresis, similar to those observed in the acute myocardial infarction. <sup>5</sup> For the aforementioned, the doctor must rely on studies imaging such as the electrocardiogram where you can observe the Wellens pattern, which is a finding that shows biphasic or deeply inverted T waves in the V2 and V3 derivations, the which suggests ischemia in the anterior wall and classically reflects a critical stenosis of the artery left anterior descending (DAI). This pattern indicates a pre-infarct state that can progress to a massive and fatal anterior myocardial infarction (MI).<sup>6</sup>

When an electrocardiogram (ECG) is recorded during the initial episode, waves can be observed T inverted in the derivations V4 to V6, as well as elevation of the ST segment in aVR and V1, being greater in aVR than in V1, a characteristic pattern of left coronary trunk stenosis (LMCA) The exact pathophysiological mechanism is not yet fully elucidated. Some researchers relate it to the phenomenon of myocardial stunning (ischemic stunning), in the which is the ischemia of the anterior wall of the left ventricle, secondary to a critical stenosis of the anterior descending artery (LAD), alters the ventricular repolarization process. This alteration is manifests as inversion or biphasic morphology of the T wave, which tends to normalize once that the contractile function of the myocardium is recovered.<sup>7</sup>

During the interpretation of the ECG it is important to take into account that there are various conditions that can imitate the characteristic electrocardiographic pattern of Wellens syndrome, known as "pseudo-Wellens". These include congenital myocardial bridge, induced cardiomyopathy by stress (takotsubo) and the consumption of psychoactive substances.<sup>8</sup> In addition, other pathologies such as brain injuries, left ventricular hypertrophy, pulmonary embolism, right branch block and hypertrophic cardiomyopathy can also cause T-wave inversion. It's fundamental differentiate the genuine Wellens syndrome from these simulator entities, since the realization of a stress test in the presence of a critical stenosis could precipitate an acute infarction of myocardium.<sup>9</sup>

Clinical, laboratory, and electrocardiographic criteria for Wellens syndrome include:



History of angina, minimal or no elevation of cardiac enzymes, minimal or no elevation of the ST segment ( $<1$  mm), absence of precordial pathological Q waves and biphasic T waves in V2 and V3 (Type 1) or deep, symmetrical and inverted T waves in V2 and V3, occasionally in V1, V4, V5 and V6 (Type 2).<sup>[10](#)</sup>

Acute coronary syndrome (ACS) should be considered and ruled out as a diagnosis before the presence of acute chest pain. While cardiac biomarkers and TIMI score are useful tools for risk stratification, in situations such as Wellens syndrome immediate intervention may be required, even when the markers are negative and the risk score is low. Recognizing this syndrome early is key to avoiding heart attacks of extensive myocardium.<sup>[11](#)</sup>

As mentioned above, patients with Wellens syndrome have a high risk of suffer an extensive myocardial infarction in the anterior region of the heart, so it is essential perform an early coronary revascularization.<sup>[12](#)</sup> Some research has suggested that intervening early with revascularization in patients with Wellens syndrome can

significantly improve your chances of survival. Acting quickly allows you to restore adequately cardiac reperfusion, which minimizes damage to cardiac tissue and reduces the appearance of cardiovascular complications.<sup>[13](#)</sup>

It is estimated that approximately 75% of patients treated only with medical measures progress to an acute myocardial infarction. Therefore, it is strongly recommended to carry out an immediate coronary intervention as a definitive treatment. The combined use of antiplatelet platelets, such as acetylsalicylic acid and clopidogrel, along with measures such as thrombolysis, the control of blood pressure and glucose, as well as the exclusive use of statins, do not achieve significantly reduce morbidity or mortality related to events such as left ventricular dysfunction, anterior infarction or sudden death.<sup>[14](#)</sup>



It should be noted that in patients with Wellens syndrome, stress tests should not be carried out under any circumstances. Although at the beginning they can show an apparent improvement with medical treatment, in the long term the conservative approach is insufficient, so it is essential to implement revascularization interventions. For this reason, it is essential that the medical personnel correctly identify the characteristic electrocardiographic findings of this syndrome, in order to establish adequate management in time and thus reduce the risk of severe complications such as acute myocardial infarction and death.<sup>15</sup>

## **MATERIAL AND METHODS**

A systematic review was conducted on 20 scientific articles and textbooks from 2009 to 2025 with high evidence levels, focusing on electrocardiographic patterns associated with LAD stenosis. Additionally, we present the case of a 70-year-old male with risk factors including type 2 diabetes, hypertension, and obesity, who presented with chest pain and fatigue. Diagnostic tests included ECG, cardiac enzymes, and coronary angiography, biochemical analysis serves as a follow-up criterion for these cases. Ethical procedures were respected, and patient consent was obtained. Institutional protocols were followed under the approval of the hospital's ethics committee.

## **RESULTS**

The patient presented initially to private and later to public hospital services with symptoms of chest pain and dyspnea. ECG revealed deeply inverted T waves in V1–V5, normal QRS and PR intervals, and no signs of myocardial necrosis. Cardiac enzymes were minimally elevated. Based on clinical findings, a diagnosis of Wellens syndrome type II was made. Urgent coronary angiography revealed critical stenosis of the proximal LAD. A percutaneous intervention with stent placement was performed successfully. The patient recovered in the ICU and was discharged five days later with a favorable prognosis.

## **Discussion**

This case emphasizes the importance of early recognition of the electrocardiographic signs of Wellens syndrome, especially in patients presenting without classic signs of ST-elevation myocardial infarction. The patient's timely diagnosis and treatment prevented extensive myocardial infarction. This reinforces the value of structured protocols such as the heart attack code and highlights the necessity of continuous



training for emergency care personnel. Previous studies have shown a high rate of infarction within days if revascularization is delayed. The limitations of this study include reliance on a single case, but it reflects the real-world utility of ECG in guiding urgent intervention. Future studies may expand to evaluate long-term outcomes post-revascularization in Wellens syndrome.

## Conclusions

Wellens syndrome represents a diagnostic challenge due to its subtle ECG presentation. Timely recognition and intervention are essential to reduce mortality. Training of medical personnel and institutional protocols play a fundamental role in the early identification and management of this high-risk condition. We recommend performing a biochemical analysis as part of the patient's follow-up to ensure the success of the intervention.

Keywords: Wellens; Electrocardiography; Stenosis; Revascularization; Myocardial infarction.

## CASE:

It is a 70-year-old male patient with only the following important medical history:

Type 2 diabetes for a month of diagnosis in treatment, do not remember what medications you use, systemic arterial hypertension of 1 year of diagnosis in treatment with losartan at unremembered dose.

He reports having started with symptoms on February 14, 2025, characterized by dyspnea of medium efforts, fatigue, fatigue which initially went to get effort, intensifying to medium, reports that in the face of this increase in symptoms on March 14, 2025 he decides to go to a consultation with cardiology, where a diagnosis of stable angina of low risk is made without receiving treatment, the symptoms were intensifying, appearing oppressive precordial pain, without irradiation, on an intensity scale of 6/10. On March 24, 2025, without improvement of the condition, he decides to go to a private hospital where after paraclinics (image 1, table 1), admission to his unit is indicated with the following diagnoses: Acute coronary syndrome without elevation of the ST segment + essential hypertension (primary) + Diabetes mellitus type II, dyslipidemia + obesity. Treatment is indicated: Acetyl salicylic Acid 100 mg PO every 24 hours + atorvastatin 40 mg PO every 24 hours + clopidogrel 75 mg PO every 24 hours + enoxaparin 20 mg SC every hours, with general care measures. It should be noted that no note with vital signs or physical exploration was found.



On March 25, 2025, for personal reasons, the patient decides to discharge from the private hospital voluntarily at 12:04 hours, the same day at 9:00 pm he goes for evaluation to the emergency service of the General Hospital of Playa del Carmen, State of Quintana Roo, Mexico, on March 25, 2025, where after performing a thorough assessment with the physical examination in normal parameters, where the paraclinics performed in the external hospital were included, adding institutional counterparts (image 2, table 1) timely protocol of heart attack code is executed at 21:30 hours on March 25, 2025, with the following diagnoses: Wellens syndrome type 2 + diabetes type 2 in treatment + systematic arterial hypertension in treatment with the following vital signs: BP: 110/80 mmHg, HR: 75 beats, RF: 19 rom, T: 36 C, SaO2: 97% Glasgow non traumatic: 15.

The electrocardiogram shows a heart rate of 78 lpm with sinus rhythm and an approximate axis of +90. Deep and symmetrical inversion of the T wave from v1 to v5 is observed, without left ventricular hypertrophy data according to the Peguero- Lo Presti criterion (2.4), and without sings of injury or necrosis. The PR and QRS intervals were 0.16 s and 0.08 s, respectively. These findings are compatible with Wellens syndrome type 2 (figure 1).

On March 25, 2025, at 9:45 pm, the patient is evaluated and accepted urgently for percutaneous coronary interventional cardiology service of hospital. At 10:00 pm, the patient's intervention is performed percutaneously with the placement of stents in coronary, without accidents or incidents. The following practices are taken after the surgical procedure (figure 2, table 1).

The patient is admitted to the intensive care unit after the interventional procedure to be discharged on March 30, 2025. To follow up on the patient's condition, new biochemical tests are taken, to evaluate the effectiveness of the surgical treatment by finding the following results. (table 2).

El paciente fué evaluado posterior a 15 días con una exploración clínica completa, la cual se encontraba dentro de parametros normales, así como una evolución bioquímica y enzimática completa (table 3)

## **Discussion**

It should be noted that the success in the treatment of this patient is due to the clinical expertise of those involved in this case, to carry out the initial and definitive approach of the patient, taking into account the effective response time of the heart attack code of the General Hospital of Playa del Carmen.



The author of this next recommended performing a rapid analysis that includes the chronic history of the patient, as well as the current symptoms, without ignoring the paraclinics of the patient under study, without ruling out the possibilities of this syndrome, before a normal electrocardiogram but with chronic symptoms.

Last but not least, we want to emphasize the importance of carrying out a heart attack or coronary disease protocol in each hospital or medical research center, in order to treat each patient as effectively as possible, without neglecting the integrative possibilities incipient coronary pathologies. This syndrome, as well as some others, can occur in younger patients without comorbidities.

The lack of training of each service within each institution becomes a key piece to achieve effective treatment in each individual.

## **CONCLUSION**

It is a diagnostic and therapeutic challenge for each doctor that is part of the coronary approach protocol in each institution. The training of each individual and clinical experience become the cornerstone for this condition. The general recommendation that is held by the most difficult and strange without demeaning the obvious, because this always allows us to investigate and diagnose the most complex pathologies with greater greed. We recommend performing a biochemical analysis as part of the patient's follow-up to ensure the success of the intervention.

## **Gratitudes:**

Marcos Tienda Pimentel, MD: To my father and mother for stronghold and aploment at every stage of my life, to my old and teachers who are in my training. To the Autonomous University of Yucatan for joining its ranks.

Sergio Esqueda Vargas, MD: To my wife Ivonne Montero Parra, MD, who gives me her support and unconditional love, to my son Santiago Esqueda Montero, with love.

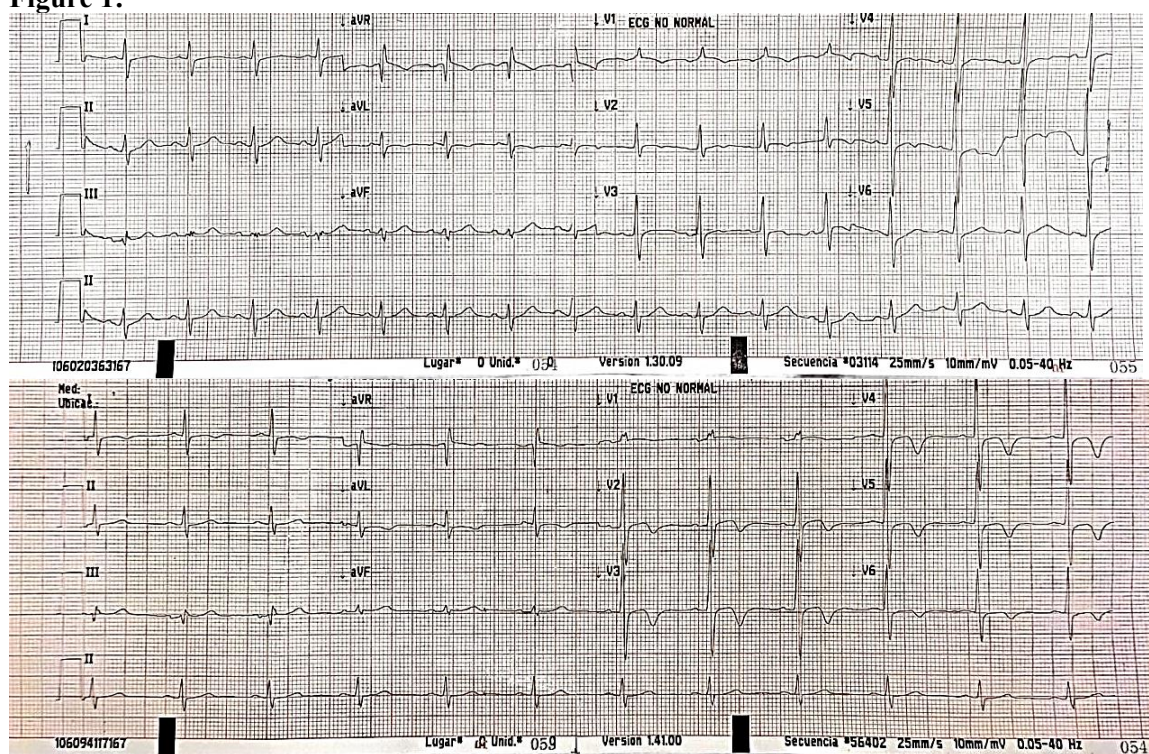
Graciela Guadalupe Andrade Vázquez, MD: To God for his company, and to myself, for my determination.

Frida Denisse Alvarez Rios, MD: To my mother, for her unconditional love, strength and constant example of dedication and to my sister, for her company, support and trust in every step of my professional path.



## Annexes:

**Figure 1:**



**Figure 3:**



Table 1

**Report of Paraclinics on Patient Admission**

Patient: Patien XXX Age: 70 years Sex: Male

| Parameter          | 24.03.26 (16:48 hrs) | 24.03.26 (22:51 hrs) |
|--------------------|----------------------|----------------------|
| Creatinine         | 0.96                 |                      |
| Glucose (GLU)      | 143                  |                      |
| Urea (URE)         | 19.3                 |                      |
| BUN                | 9                    |                      |
| Sodium             | 137                  |                      |
| Potassium          | 4.2                  |                      |
| Chlorine           | 104                  |                      |
| Total bilirubin    | 0.61                 |                      |
| Direct bilirubin   | 0.14                 |                      |
| Indirect bilirubin | 0.47                 |                      |
| Troponin           | 3.10                 | <1.5                 |
| CK                 | 232                  |                      |
| CKMB               | 22                   | 17.9                 |
| DHL                | 196                  |                      |
| TGO                | 49                   |                      |
| TGP                | 52                   |                      |



Table 2.

**Post-Angioplasty Laboratory Results**

Patient: Patien XXX Age: 70 years Sex: Male

Date: March 26, 2025 Hour: 10:15 HRS

| Parameter                 | Result |
|---------------------------|--------|
| <b>Blood chemistry</b>    |        |
| Creatinine                | 0.89   |
| Glucose (GLU)             | 124    |
| Urea (URE)                | 17.5   |
| BUN                       | 8.2    |
| <b>Serum electrolytes</b> |        |
| Sodium                    | 138    |
| Potassium                 | 4.4    |
| Chlorine                  | 103    |
| <b>Bilirubines</b>        |        |
| Total bilirubin           | 0.58   |
| Direct bilirubin          | 0.13   |
| Indirect bilirubin        | 0.45   |
| Troponin I                | 1.4    |
| <b>Muscle enzymes</b>     |        |
| CK                        | 198    |
| CKMB                      | 12.3   |
| DHL                       | 165    |
| TGO (AST)                 | 42     |
| TGP (ALT)                 | 46     |

*Biochemical laboratory results after angioplasty. Improvement is observed in cardiac markers, such as troponin I and muscle enzymes, although they are not yet fully normalized. Renal and hepatic values remain within acceptable limits, reflecting a clinical evolution favorable.*



Table 3.

**Laboratory Results- 15 Days Post Angioplasty**

Patient: Patien XXX Age: 70 years Sex: Male

Date: April 10, 2025 Hour: 09:20 HRS

| Parameter                 | Result |
|---------------------------|--------|
| <b>Blood chemistry</b>    |        |
| Creatinine                | 0.92   |
| Glucose (GLU)             | 108    |
| Urea (URE)                | 16.1   |
| BUN                       | 7.6    |
| <b>Serum electrolytes</b> |        |
| Sodium                    | 139    |
| Potassium                 | 4.3    |
| Chlorine                  | 102    |
| <b>Bilirubines</b>        |        |
| Total bilirubin           | 0.59   |
| Direct bilirubin          | 0.12   |
| Indirect bilirubin        | 0.47   |
| Troponin I                | 0.7    |
| <b>Muscle enzymes</b>     |        |
| CK                        | 174    |
| CKMB                      | 8.9    |
| DHL                       | 152    |
| TGO (AST)                 | 36     |
| TGP (ALT)                 | 40     |

*Biochemical results at 15 days after angioplasty. At 15 days after angioplasty, a clear trend towards the normalization of biochemical parameters is observed. The values of cardiac and muscle enzymes such as CK, CKMB and Troponin I have decreased with respect to acute stages, indicating an improvement in tissue damage. Likewise, serum electrolytes and liver tests show stability and kidney function remains adequate.*



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