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Wellens' syndrome: report and review of a case

Síndrome de Wellens. Reporte y revisión de un caso

Oswaldo Aldana Varela,* Giuliana Verónica Chacón Juárez,* Marco Antonio Hernández-Mercado,‡ Froylán Fernando López Reyes,§ Norma Eloisa Morales-Bernal‖

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Palabras clave:
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ABSTRACT

Wellens' syndrome is a clinical entity characterized by an important occlusion of the left anterior descending artery (10-15% of incidence) in more than the 80% of the cases in the proximal segment, that is diagnosed with an electrocardiogram with the presence of precordial T wave inversion (V1-V5) in the 76% of the cases (type B) and in the 24% of the cases in V2-V3 leads (type 1). We present a patient evaluated in the emergency department for intermittent chest pain and T-wave inversion of the Wellens syndrome type B pattern, who was treated with percutaneous coronary intervention where a critical lesion was demonstrated in the medial anterior descending. This case shows how timely diagnosis prevents progression to extensive anterior acute myocardial infarction.

RESUMEN

El síndrome de Wellens es una entidad clínica caracterizada por una oclusión importante de la arteria descendente anterior izquierda (10-15% de incidencia) en más de 80% de los casos en el segmento proximal, que se diagnostica con electrocardiograma por la presencia de inversión de onda T precordial (V1-V5) en 76% de los casos (tipo B) y en 24% de los casos en derivaciones V2-V3 (tipo 1). Presentamos a un paciente evaluado en el Servicio de Urgencias por dolor torácico intermitente e inversión de la onda T del patrón del síndrome de Wellens tipo B, que fue tratado con intervención coronaria percutánea, en la que se demostró una lesión crítica en la descendente anterior media. Este caso muestra cómo el diagnóstico oportuno previene la progresión a infarto de miocardio agudo anterior extenso.

* Médico Residente del Servicio de Cardiología y Cirugía Cardiovascular del Centro Médico ISSEMYM «Lic. Arturo Montiel Rojas», Toluca. Estado de México, México.
‡ Investigador y Asesor Clínico. Jefe de Servicio de Cardiología y Cirugía Cardiovascular del Centro Médico ISSEMYM «Lic. Arturo Montiel Rojas», Toluca. Estado de México, México.
§ Médico adscrito al Servicio de Cardiología y Cirugía Cardiovascular del Centro Médico ISSEMYM «Lic. Arturo Montiel Rojas», Toluca. Estado de México, México.

INTRODUCTION

Chest pain is a common reason for seeking care in emergency departments. Cardiovascular disease can be present in up to 20%, but only 5.5% represents a life-threatening condition. The low to moderate risk stratification could lead to an early discharge, it has been shown that from 2% to 13% of patients discharged from the emergency services and who came for chest pain presented undiagnosed acute myocardial infarction.[1] The interpretation of the ECG usually follows the evaluation of patients with suspected myocardial ischemia after medical history and physical examination, so it is vitally important to recognize any pattern that suggests myocardial ischemia taking into account that 10 to 25% of patients with acute coronary syndrome requiring urgent reperfusion therapy, may have atypical ECG patterns.[2,3]

Wellens Syndrome (WS) is a preinfarction stage, characterized by an electrocardiographic pattern of changes in the T wave associated with severe stenosis of the anterior descending artery.[4] It is currently not considered an equivalent pattern of acute coronary syndrome,[5] despite that without timely diagnosis and intervention, extensive acute myocardial infarction of the anterior wall can occur in an average time of 8.5 days[6] and possibly sudden death, so they must undergo percutaneous coronary intervention. We present a clinical case from the Hemodynamics Service of our institution, in order to analyze the characteristics of Wellens syndrome, emphasizing timely and differential diagnosis.

CASE REPORT

A 62-year-old male, without a family history of cardiovascular disease. With a personal pathological history of positive smoking from 42 years of age with an 8 pack/year smoking history, type 2 diabetes mellitus and systemic

‖ Investigador y Asesor Metodológico. Investigador y Data Manager ConsulMed, México. Estado de México, México.

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hypertension of two years of diagnosis without adequate control and chronic kidney disease KDIGO 5, recently diagnosed, without renal function replacement treatment.

He was admitted to a second-level hospital with 6-hour history of sudden and intermittent oppressive chest pain at rest intensity 7/10, with irradiation to the left thoracic limb lasting two minutes, associated with dyspnea, improvement with nitrate intake. Braunwald class IIIA unstable angina was diagnosed and was referred to our hospital for evaluation.

At admission, the patient was asymptomatic with vital signs of blood pressure 150/80 mmHg, SO_2 94%, temperature 36 °C, heart rate 83 beats per minute. Neck without jugular venous distention, precordium without abnormal heart sounds, lung fields with vesicular murmur, painless abdomen, edema-free extremities with symmetrical pulses.

Initial electrocardiogram showed sinus rhythm, heart rate 75 beats per minute, P wave 80 ms, PR interval 190 ms, QRS complex 80 ms, electrical axis + 30°, no Q waves, symmetric and deep T wave inversion (up to 1 mV) from V2 to V6, and shallow (up to 0.2 mV) in I, II, aVL *(Figure 1)*.

The cardiac biomarkers were increased: troponin I 1.54 ng (0-0.40 ng/mL) and CK-MB 53 mg/dL (0-24 mg/dL). The enzymatic curve did not show any increase, while the electrocardiogram recorded during a period of pain revealed pseudo normalization of the T wave *(Figure 2)*. The patient was pharmacologically treated with atorvastatin, aspirin, clopidogrel and enoxaparin from admission.

The typical angina, the result of the highly sensitive troponin, as well as the high-risk electrocardiographic pattern are nosological characteristics of Wellens syndrome type B. Therefore, it was decided to submit the patient to coronary angiography that revealed a critical lesion in the medial portion of the anterior descending with distal TIMI 3 flow that required the placement of 2 drug-eluting stents. Angiography also showed a 90% lesion in the marginal obtuse and no significant disease in the right coronary artery *(Figure 3)*.

The patient was discharged stable and improved six days later, with a pharmacological indication of double antiplatelet therapy for 12 months. At 6 months follow-up in the outpatient clinic, he was clinically asymptomatic.

DISCUSSION

There are many elements described in the universal literature as case report for Wellens syndrome. Wellens syndrome is a clinical entity, didactic due to its acute symptoms and documented by means of electrocardiograms, but there is important evidence of ischemia in resting nuclear medicine, given that in many cases it is persistent angina.[7]

We present a male patient with multiple cardiovascular risk factors and chest pain; with a first diagnosis to be ruled out: acute coronary syndrome with or without ST segment

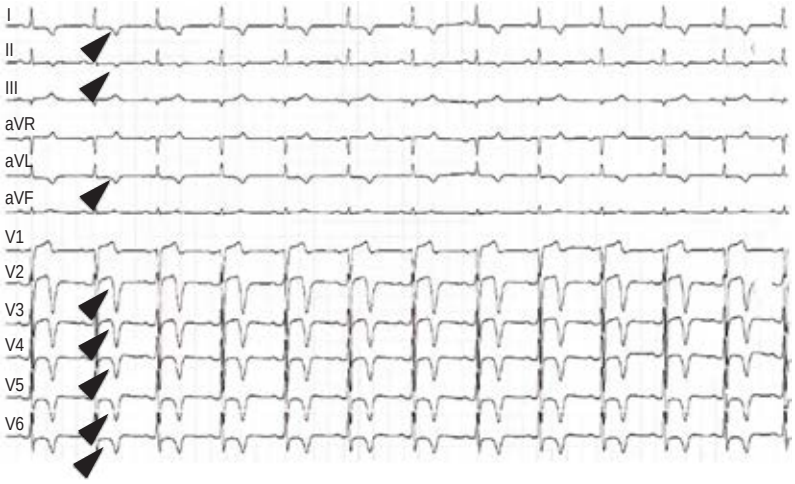

Figure 1:
12-lead electrocardiogram, shows symmetrical and deep T-wave inversion (up to 1 mV) from V2 to V6 and lower depth (up to 0.2 mV) in I, II, aVL.

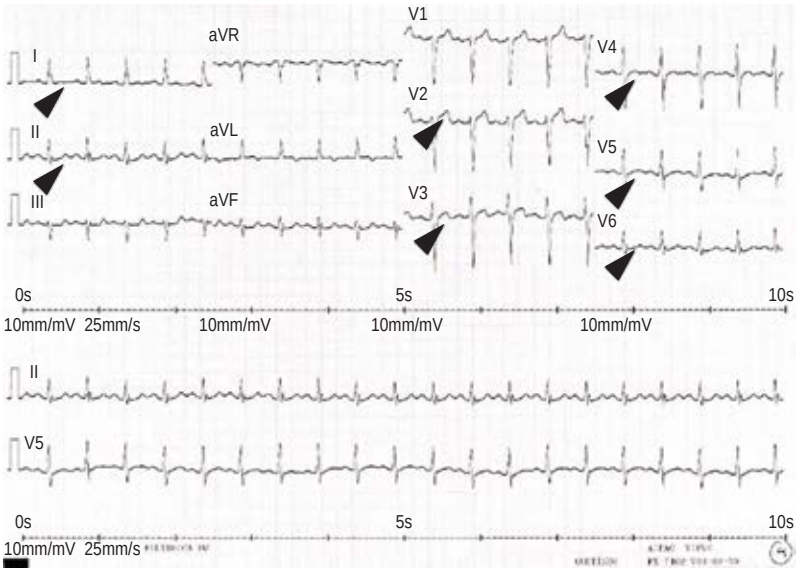

Figure 2:

Pseudo-normalization of T-waves.

elevation, being essential to establish risk, the measurement of highly sensitive troponins and the electrocardiogram. The latter was taken in a period without pain and showed a typical pattern of Wellens syndrome type B,[8] which allowed the patient to be defined as high risk,[9] conditioning an early invasive strategy[4] without performing other types of ischemia-inducing tests, reducing the morbidity and mortality.[10,11] The patient presented a critical obstruction in the middle portion of the anterior descending artery, although it has been controversial whether these patients should really be diagnosed as Wellens syndrome,[12,13] since the original study refers to the proximal portion of the descending anterior[4,9] we are convinced (based on multiple studies and case reports in the medical literature) that this may be a variant or subgroup of Wellens syndrome, but more studies and more debate on the subject will be required.

Wellens syndrome or anterior descending artery syndrome, in an electrocardiographic pattern with changes in the precordial T waves, was described by Zwaan and Wellens in 1982[4] in 14% to 18% of admitted patients with unstable angina and associated with critical stenosis of the proximal anterior descending. Kobayashi and collaborators, reported in 2019 a retrospective analysis of 274 patients with non-ST-segment elevation myocardial infarction, presenting Wellens pattern 24 (8.8%) of the patients, of these, 16 had a culprit lesion of the anterior descending (8 proximal, 8 mid), 2 patients had a culprit lesion not of the anterior descending artery and 6 patients had non-obstructive coronary artery disease.[14] Therefore, despite the association that was initially described, the same electrocardiographic pattern may be in critical obstructions of the mid anterior descending artery, also in normal coronary arteries with spasm induced by substances such as cocaine called pseudo Wellens syndrome,[15,16] coronary angiography being the method of choice to confirm this latter diagnosis, since precordial pain as well as elevation of cardiac enzymes is also present in cocaine users.

The incidence of WS is 10-15% of all acute coronary syndromes[17] with an etiology similar to any other condition that causes coronary heart disease: atherosclerotic plaque, coronary arterial vasospasm, hypoxia, and increased cardiac demand. Its risk factors include a family history of premature coronary heart disease, type II diabetes mellitus, metabolic syndrome, hypertension, smoking, hyperlipidemia, work stress, advanced age[18] (55 ± 9 years), cases associated with HIV, probably due to premature coronary disease[19] and also in patients without cardiovascular risk factors.[20]

It is classified into type A and B (1 and 2 respectively).[8] Type A constitutes 24% of cases,

it is less common but more specific and less recognized, it presents biphasic T waves in V2 and V3. Type B is the most common and least specific,[9] represents 76% of cases and is characterized by symmetrical and deep inverted T waves in V1 to V4.[17] Schears MR and collaborators, describe the evolution of these patterns starting with biphasic T waves (pattern A) that are deeply and symmetrically reversed (pattern B). Then it extends to V4, then to V5 and finally to V6, not documented in all cases by lack of serial electrocardiograms,[21] without defining the time in which one pattern can change to another. Atypical cases have been described where the type B pattern changes to type A.[22] It should be emphasized that changes in the T wave occur during periods without precordial pain and pseudo normalized during the precordial pain episode, and may persist for hours or weeks[7,23] leading to errors in diagnosis and inadequate risk stratification, so the ability to recognize these patterns is extremely important, since ECG changes can be subtle[8-24] There are atypical presentations in which there is no precordial pain, only syncope, but the electrocardiographic pattern meets WS criteria.[25] In the case presented, the fact that the patient was asymptomatic at the time of admission to the emergency department, and the taking of an initial electrocardiogram as part of the cardiology evaluation, showed the inversion of T waves in precordial leads from V2 to V6, leading to timely diagnosis confirming it with pseudonormalization during an episode of precordial pain, which occurred spontaneously without maneuvers to cause ischemia.

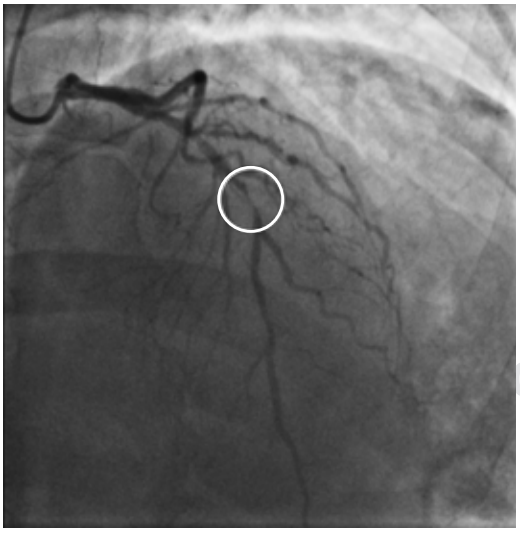

Figure 3: Coronary angiography showing critical lesion in the middle portion of the anterior descending.

The pathophysiology of ECG changes is unclear, local edema or lightheadedness due to intermittent or destabilized blood flow in the anterior descending coronary artery with critical obstruction has been proposed.[26] These changes have been demonstrated through cardiac magnetic resonance imaging.[27]

Although the patient's physical examination was normal, a protodiastolic murmur has been described in the third intercostal space above the left clavicular midline, probably caused by turbulence of blood flow immediately distal to the stenosed segment, known as Dock's murmur.[28]

The criteria for Wellens syndrome were described by Rhinehardt and collaborators, as follows:[8] deeply inverted T waves in leads V2 and V3 (can also be seen in leads V1, V4, V5 and V6) or biphasic T waves (with initial positivity and terminal negativity) in V2 and V3, recent history of precordial pain, absence of Q waves without loss of R waves in precordials, without significant ST segment elevation (< 1 mm), normal or minimally elevated cardiac markers, and changes in the T wave (biphasic or inverted) in the precordial leads in a pain-free state. The positive predictive value of WS to detect significant proximal anterior descending coronary artery stenosis is 86%, with a sensitivity of 69% and specificity of 89%.[2,29]

Differential diagnosis of precordial T-wave inversion includes pulmonary embolism,[30] cerebral hemorrhage, left ventricular hypertrophy, cocaine or morphine-induced coronary vasospasm, chronic thromboembolic pulmonary hypertension, interruption of transient left bundle branch block, or Wolff-Parkinson-White pattern, persistent juvenile T wave pattern, late stages of pericarditis, digitalis effect and Takotsubo cardiomyopathy.[31-33] Under a suitable clinical context, each one can be ruled out.

It is possible to confirm the diagnosis with coronary angiotomography, in hemodynamically stable patients[7,13] or with doubt in the initial approach since it is the most accurate

and reliable non-invasive modality to rule out significant coronary artery stenosis. It also helps to rule out conditions that cause the syndrome of pseudo Wellens such as pulmonary embolism, cannabis and phencyclidine poisoning, use of crack and vasospastic angina,[34] avoiding unwarranted cardiac catheterization.

Stress ischemia was not performed in this patient using a stress test since it is not indicated because it increases the demand for myocardial oxygen, producing adverse effects such as ST elevation and/or ventricular tachycardia with fatal results.[10,11]

Treatment with double antiplatelet (acetylsalicylic acid and clopidogrel), thrombolysis, control of blood pressure and glucose, as well as statin therapy alone do not decrease morbidity and mortality (left ventricular dysfunction, anterior myocardial infarction or sudden death),[8,35] 75% of patients progress to myocardial infarction only with medical treatment.[4] Therefore, immediate coronary intervention is highly recommended as a definitive therapeutic strategy.

CONCLUSIONS

The clinical presentation is the cornerstone for the suspected diagnosis in every chest pain to guide the correct diagnosis (typical and atypical angina). Wellens syndrome is characterized for electrocardiographic changes with right precordial T wave inversion V1-V5, with an occlusion of the proximal segment of the left anterior descending artery in the majority of cases, but in some cases the medial or distal segment of the left anterior descending coronary artery is occluded. Cardiac biomarkers are normal or may be slightly increased. The ischemic changes can be documented by myocardial perfusion gammagraphy or coronary angiography by computed tomography.

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Correspondence to:

Oswaldo Aldana Varela
Médico Residente del Servicio de Cardiología y Cirugía Cardiovascular del Centro Médico ISSEMYM «Lic. Arturo Montiel Rojas», Toluca.
Av. Baja Velocidad Km. 57.5,
Carretera México-Toluca Núm. 284,
Col. San Jerónimo Chicahualco, 52170,
Metepec, Estado de México, México.
E-mail: doc_os_var@hotmail.com