Wellens syndrome and the value of the T-wave

Síndrome de Wellens: la importancia de la onda T

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Wellens syndrome (WS) is a medical emergency described in the context of non–ST-segment elevation acute coronary syndromes (NSTEACS).[1] Diagnosis is based on the presence of inverted or biphasic T waves in precordial leads V_2 and V_3, representing an electrocardiographic sign of a critical or total stenosis in the proximal portion of the left anterior descending coronary artery (LAD).[2] Total occlusion of a major coronary artery usually presents as an ST-segment elevation myocardial infarction (STEMI); however, other high-risk electrocardiographic patterns that do not show the classic ST-segment elevation can occur,[3] such as in WS, which reflects acute coronary artery occlusion and is associated with a poor prognosis if not identified and treated promptly.[4] We present 2 case reports consistent with the diagnosis of WS.

Case report #1

A 63-year-old man with a past medical history of hypertension presented to the emergency department with a 12-hour history of typical intermittent chest pain. On arrival, the patient was asymptomatic. Vital signs and physical examination were normal. Laboratory tests revealed a mild elevation in high-sensitivity troponin I (hs-TnI). The electrocardiogram (ECG) (Figure 1A) showed sinus rhythm at 84 beats per minute (bpm) and biphasic T waves in leads V_2–V_4. Emergency coronary angiography revealed a critical stenosis in the proximal LAD (Figure 1B), which was treated with implantation of a drug-eluting stent (DES) with good final angiographic results (Figure 1C). The diagnosis was consistent with type A WS. The patient was discharged on dual antiplatelet therapy (aspirin and ticagrelor) and high-intensity statin therapy with atorvastatin.

Case report #2

An 81-year-old woman with dyslipidemia presented to the emergency department with a 48-hour history of self-limiting episodes of typical chest pain. On arrival, she was asymptomatic, with normal vital signs and physical examination. High-sensitivity troponin I was within normal limits. The ECG showed sinus rhythm at 70 bpm with deep, symmetric, inverted T waves in leads V_2–V_4 (Figure 2A). Urgent coronary angiography revealed a complete occlusion of the mid-LAD segment (Figure 2B), which was treated with implantation of 3 DESs, achieving good final angiographic results (Figure 2C). The diagnosis was consistent with type B WS. The patient was discharged on dual antiplatelet therapy (aspirin and clopidogrel) and atorvastatin.

WS was first described in 1982 by de Zwaan and Wellens[5] in a subgroup of patients admitted with unstable angina who shared a common ECG pattern characterized by symmetric, deeply inverted T waves in precordial leads V_4 and V_6 during pain-free periods. Among patients who were not revascularized, 75% developed a large anterior acute myocardial infarction (AMI) despite optimcal medical therapy. In a prospective study published in 1989,[6] all patients with unstable angina and the characteristic ECG pattern had at least a 50% stenosis in the proximal LAD, with complete occlusion in 59% of cases, confirming that T-wave inversion in V_2–V_3 represents critical LAD disease—with a sensitivity, specificity, and positive predictive value of 69%, 89%, and 86%, respectively.[7]

The incidence rate of WS has been reported between 9% and 14%, although more recent series, such as that by Li Zhou *et al.*, estimate it at 5.7% of all patients admitted with acute coronary syndrome (ACS).[8] The risk factors associated with WS are similar to those of other ACS (hyperlipidemia, obesity, chronic kidney disease), though diabetes and hypertension are the most frequently observed.[9]

T-wave morphology defines 2 types of WS: type A, which accounts for 25% of cases and is characterized by biphasic T waves in V_2–V_3, and type B, which represents 75% of cases and shows deeply inverted T waves in the same leads.[1] Occasionally, these findings may extend to leads V_4–V_6. The ECG pattern must be present during pain-free periods, and during anginal episodes, T-wave abnormali-

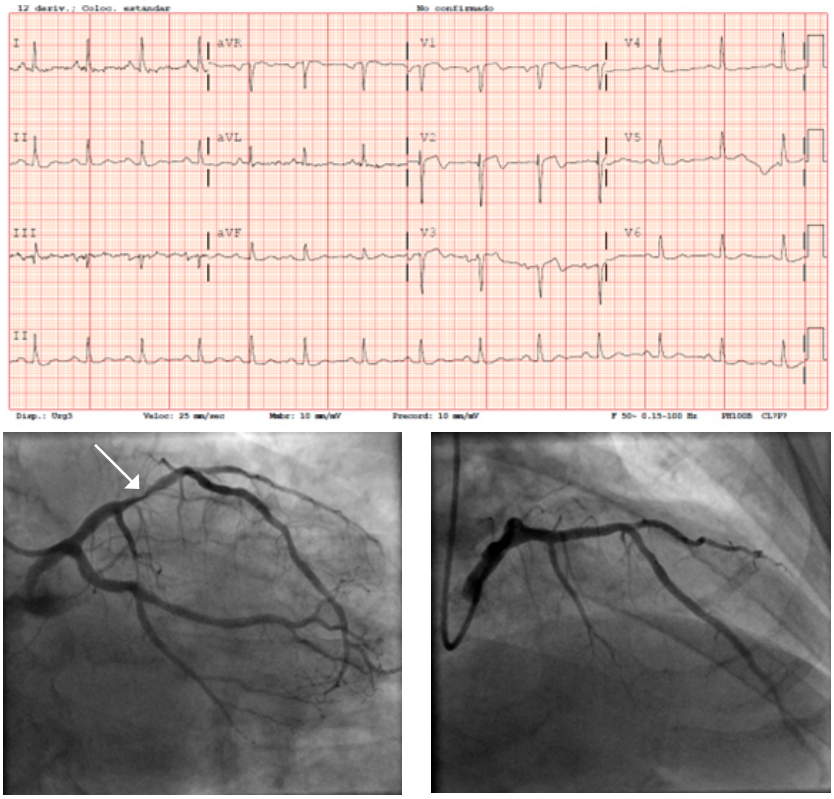

Figure 1. 1A: ECG showing sinus rhythm at 84 bpm and biphasic T waves in V_2–V_3. B: Coronary angiogram showing critical proximal LAD stenosis (arrow). C: well perfused LAD after stent implantation.

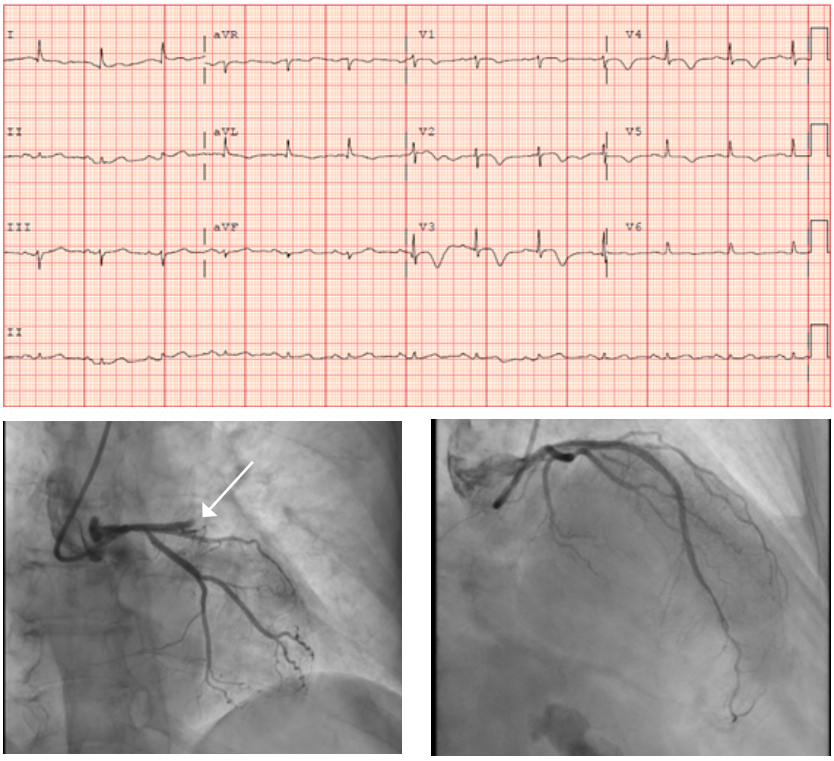

Figure 2. 2A: ECG showing sinus rhythm at 70 bpm and deep, symmetric, inverted T waves in V_2–V_4. B: Coronary angiogram showing complete occlusion of the mid-LAD segment (arrow) with no distal flow. C: well perfused LAD after stent implantation.

ties typically normalize.[10] Diagnostic ECG criteria also include an isoelectric or minimally elevated ST segment (< 1 mm), absence of Q waves in precordial leads, and normal or minimally elevated cardiac biomarkers.[2] T-wave inversion is not pathognomonic of WS and may appear in myocarditis, left ventricular hypertrophy, Takotsubo syndrome, pulmonary embolism, cerebrovascular accident, or digitalis toxicity.[11]

The main pathophysiologic mechanism underlying WS is partial or complete interruption of blood flow in the LAD due to intraluminal rupture of a vulnerable atheromatous plaque, as in other ACS. Plaque rupture exposes highly thrombogenic lipids, triggering an inflammatory cascade that leads to platelet aggregation, thrombus formation, and coronary occlusion. WS has also been reported secondary to vasospasm[12] and cocaine use.[13]

The mechanism responsible for the T-wave inversion in WS remains uncertain. It has been proposed that it results from brief ischemic episodes without significant necrosis, producing stunned or hibernating yet viable myocardium.[14] Cardiac magnetic resonance imaging modalities in patients with the Wellens pattern have identified myocardial edema as the underlying substrate.[15]

Management of WS is not yet clearly defined. Approximately one-quarter of patients with NSTEACS have a totally occluded artery,[4] making recognition of ECG patterns (in the absence of ST elevation) that may indicate complete coronary occlusion critically important. WS should therefore be treated as a high- or very high-risk NSTEACS, requiring early or immediate reperfusion therapy according to established clinical practice guidelines.[1] Optimization of medical therapy with antiplatelet, lipid-lowering, and antianginal agents alone is insufficient to prevent progression to a large anterior AMI,[5] making reperfusion the main therapeutic goal. It is essential to remember that the characteristic ECG pattern appears during pain-free intervals, and serial ECGs are crucial for diagnosis. Exercise stress testing is contraindicated, as it may precipitate sudden death.[10]

WS is a high-risk condition due to critical or total LAD obstruction that may progress to extensive anterior AMI if not promptly recognized and revascularized. Early recognition of this pattern by emergency and prehospital care providers is essential to prevent morbidity and mortality associated with delayed diagnosis and treatment.

Note of the editors: This is a BOWMAN-generated English translation of the officially indexed Spanish-language article, which should be cited as Rev Esp Urg Emerg. 2024;3:246-248. In this translated version, the editors have supervised the process; however, it cannot be ruled out that some errors resulting from the artificial intelligence translation process may have gone unnoticed.

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