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Case Report

Atypical presentation of the Wellens electrocardiographic pattern associated with coronary bifurcation lesion

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ABSTRACT

We present a patient who was admitted to the emergency room due to unstable angina, with an initial electrocardiogram without signs of acute ischemia and a favorable clinical evolution. During hospitalization, she developed the Wellens electrocardiographic pattern, noted in the literature as an infrequent, poorly identified finding and with an ominous prognosis. This electrocardiographic pattern is described in precordial derivatives, suggesting a significant lesion of a principal epicardial artery; our patient had similar electrocardiographic alterations in the high lateral leads, in whom the coronary bifurcation lesion not previously described in this scenario was confirmed.

Keywords: Myocardial infarction; Electrocardiography; Myocardial reperfusion (source: MeSH NLM).

Introduction

The electrocardiogram (ECG) has been widely used for diagnosis and prognosis in acute coronary syndrome (ACS). In ST-elevation myocardial infarction (STEMI), the injury wave of the ECG correlates with the ischemic territory and the culprit injury. Nevertheless, in non-ST-elevation ACS (NSTE-ACS) the culprit lesion is not clearly defined by ECG.

In the 1980s Wellens *et al*, reported T wave abnormalities known as the "Wellens pattern" (WP), which were present in 14% of patients with unstable angina. This electrocardiographic feature implies a significant obstruction in the coronary tree with fatal consequences [1].

In this case report, we describe the evolution and electrocardiographic characteristics of the WP. Our patient had an WP with an unusual presentation of a coronary bifurcation lesion. Finally, an association between electrocardiographic and angiographic findings is discussed.

Case report

A 71-year-old woman was diagnosed with hypertension, dyslipidemia, and myocardial infarction 10 years ago. She was admitted to the emergency room for typical angina lasting more than 30 min and normal vital functions. The physical examination did not provide meaningful data. The initial ECG showed sinus rhythm, a necrosis pattern on the inferior leads, and nonspecific ST segment and T wave alterations **(Figure 1A).** Initial management included isosorbide 5 mg sublingual, with pain relief 20 min after the medication.

The Transthoracic echocardiogram (TTE) showed hypokinesia in the territory of the left anterior descending artery (mid and apical anterior, mid anteroseptal, and apical septal wall of the left ventricle), with no compensatory contraction. The wall thickness and left ventricle ejection fraction (LVEF) were normal (LVEF:55%). The ultrasensitive troponin T at 6 h after the onset of angina was 0.657 ng / mL (normal: <0.014 ng / mL), consistent with recent myocardial necrosis. The patient received doubled antiplatelet therapy and anticoagulation.

An invasive coronary angiography was indicated according to NSTE acute myocardial infarction (NSTEMI). The GRACE and TIMI scores were 100 and 2, respectively. After 24 hours of symptoms´ onset, the patient remained asymptomatic. Despite this, the control ECG showed repolarization disorders, as can be seen in **Figure 1B**. According to these new electrocardiographic findings, the need for a Cath lab was urgent. However, it was done on the fourth day of hospitalization due to delays in the health system.

Coronarography showed right dominance and a bifurcation lesion in the left anterior descending artery (LADA) with a Medina classification of 1-1-1 **(Figure 2A)**. The right coronary artery had moderate stenosis (60%) in the middle segment, while the circumflex artery had no lesions. Percutaneous coronary intervention (PCI) was performed in the LADA with successful implantation of a 3.0 x 22 mm medicated stent **(Figure 2B).** Later, the patient remained asymptomatic with isolated changes in the T wave **(Figure 1C)** until discharged without complications.

Discussion

Wellens and De Zwaan detailed the transition of two electrocardiographic patterns as an evolutionary wave [2]. Pattern A, the less common (20%). It consists of an isoelectric or minimally elevated (1 mm) takeoff of the ST segment from the QRS complex, and a concave ST-segment passing into a symmetrical inverted T wave at an angle of 60 to 90 degrees at V2-V3 lead. Pattern B includes an isoelectric or minimally elevation (1 mm) of the ST segment from the QRS complex, and a straight ST segment passing into a negative and symmetrical T wave in V2-V3 leads. There can be similar abnormalities that extend to V1, V4-V6 leads [1,3]. In fact, our patient developed ST and T wave alterations from V2 to V4, similar to pattern B, and additionally in V5 and V6. It is important to emphasize that the original studies excluded patients with secondary T wave disorders like ventricular hypertrophy and complete left or right bundle branch block, with Q waves or poor R wave progression in the precordial leads.

Classically, Wellens waves were considered an expression of ischemia due to critical injury of the proximal LADA [1-3]. However, the presence of WP has been observed in other clinical pictures of ischemic and non-ischemic left ventricular dysfunction, demonstrating by the magnetic resonance that it is not an indicator of ongoing ischemia, it might represent myocardial edema due to coronary reperfusion (spontaneous or pharmacological) after intermittent occlusion of the LADA (occlusion-perfusion cycle) [4]. De Luna *et al.*, Argue that acute ischemia does not manifest itself with isolated inversion of the T wave [5], as in our case, but rather in association with ST depression.

De Zwaan *et al.*, Described the relief of angina by beta-blockers and nitroglycerin, some without complete remission [1,3]. This angina history associated with the described T wave alterations is known as "Wellens syndrome." This favorable response observed in our patient to medication does not indicate a benign course, highlighting the prognostic value of the electrocardiographic pattern [1–3].

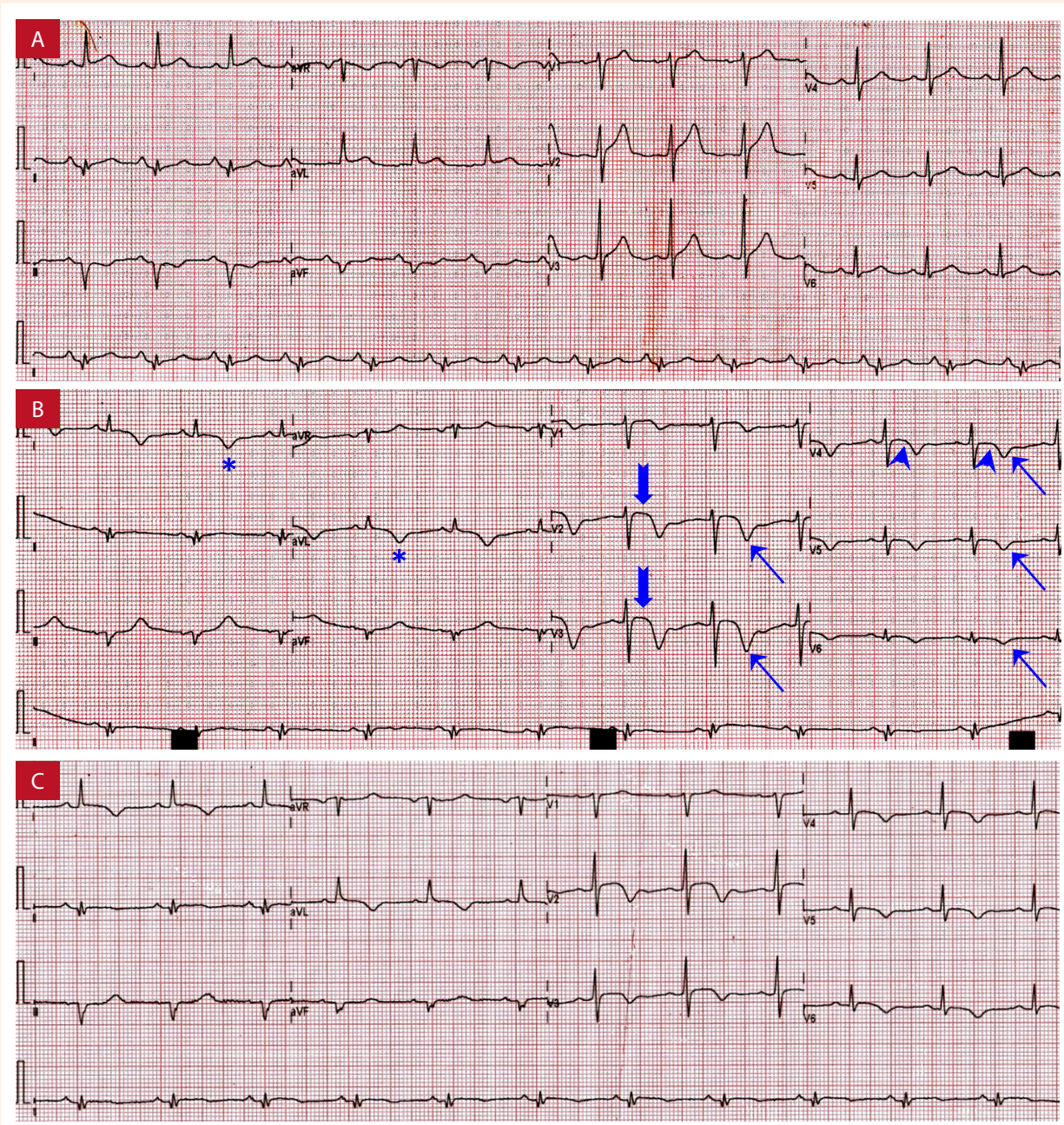

Figure 1. Electrocardiographic series (10 mm / mV; 25 mm / s). **A.** At arrival. Note the 80 ms QRS; Q waves in DII, DIII, and aVF; elevated J point in V2 and V3 (<1.5 mm), DI and aVL (<1 mm); high T waves in V1 and V2, and biphasic in DIII and aVF. **B.** Control at 24 h. Negative and symmetric T waves can be seen in the precordial leads (thin arrows) at V2-V3 are deep (> 5 mm) and associated with elevation of the J point <1 mm with rectified ST (thick arrows). In V4, the angle between the ST segment and the descending branch of the T wave was 120 ° (arrowhead); the findings are consistent with Wellens' B pattern. The alteration of the T wave described from V2 to V4 occurs in a similar pattern in DI and aVL, the T wave being of less depth (asterisk). **C.** Control on the fifth day. In V2, the elevated J point (1 mm) with an upward slope of the ST segment is shown. Negative T waves are of lower voltage, being the deepest (4 mm) in V2. No new Q waves were formed.

Initial trials included patients with unstable angina and minimal biomarker elevations; however, if high-sensitivity troponin measurement had been available in those studies, the proportion of acute myocardial infarction (AMI) had reached 64% [6]. In cohorts with NSTEMI, the incidence and performance of PW are similar to the initial studies [7].

Severe lesions with stenosis ≥ 70% of the LADA are more frequent in patients with WP compared to the control (86% vs. 26%; p <0.0001) [2,7]. Lesions with stenosis > 50% are located mainly between the first and second septal branches [3]. The most severe lesions (≥ 90%) are proximal to the first septal branch [1]. Initially, these lesions were considered proximal lesions; however, there can

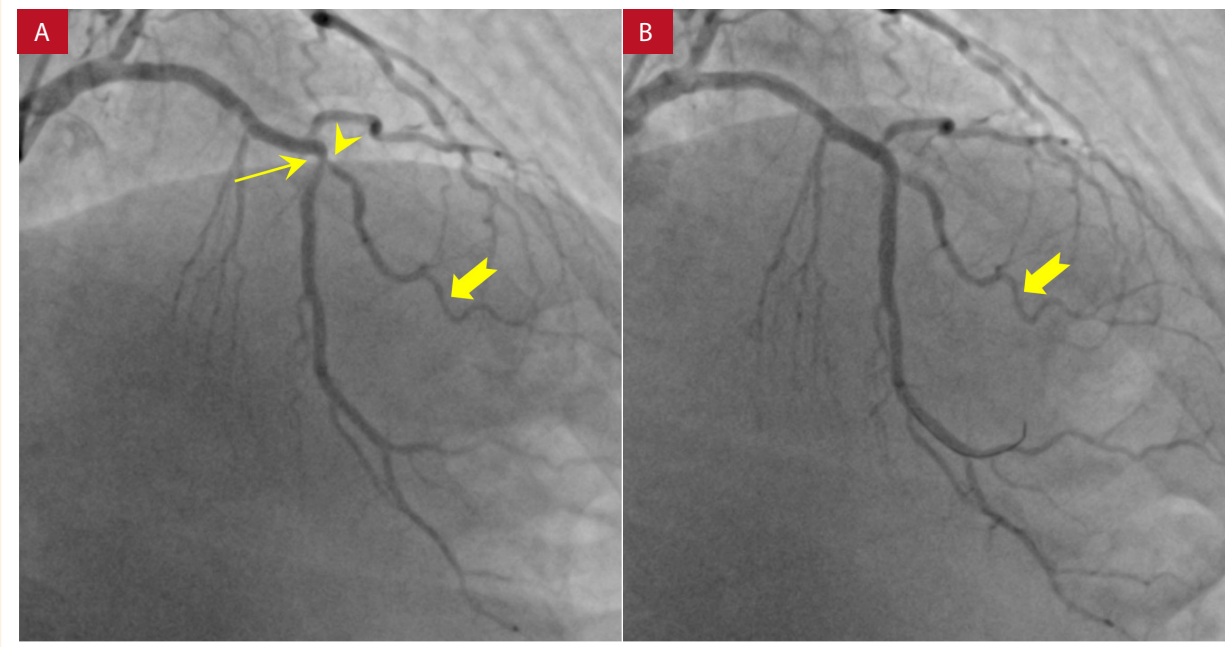

Figure 2. Coronary angiography. **A.** Right anterior oblique projection cranial view. Severe stenosis (80%) is seen in the middle segment of the anterior descending artery (thin arrow) and a non-extensive ostial lesion in the second diagonal (arrowhead) with normal TIMI flow (thick arrow). **B.** Final result: We observed TIMI 3 distal flow and absence of residual lesion in the anterior descending artery, without significant flow changes in the second diagonal (thick arrow).

be lesions with stenosis ≥ 70% at the "middle segment" (SYNTAX defines the "middle segment" as the coronary portion distal to the first major septal) [8]. Severe lesion (≥70%) of the diagonal branch is rare (7%), and we did not find publications where it constitutes a bifurcation lesion [9].

In our case, besides the typical WP and the stenosis in the LADA, we noted the involvement of the second diagonal in the presence of Wellens waves in DI and aVL leads. This association has not been explored previously. It might allow predicting the involvement of a potentially complex lesion. Likewise, WP in inferior leads has high specificity (96%) to predict severe injury in the circumflex and right coronary arteries [2,10].

In NSTE-ACS, total occlusion of a coronary artery requiring primary PCI occurs in 26%, and it is recognized before the Cath lab in less than 5% of cases [11]. The early identification of WP is important. Since the absence of early revascularization results in evolution to extensive AMI in 63% (LADA territory) or the need for late revascularization, in the context of related complications (heart failure), or death in 25% of cases [1,3].

So, even when our patient was asymptomatic on the fourth day of evolution, the Cath lab was urgently indicated because of Wellens's syndrome. Furthermore, we defer functional stress tests because they are associated with myocardial infarction and fatal ventricular arrhythmias [12].

The strategy of choice for bifurcation lesions is the provisional stent technique to the secondary branch [13], even in acute settings [14]. Only in specific complex circumstances will they benefit from an initial strategy with two stents [15]. Thus, our patient presented a bifurcation lesion (Medina 1-1-1) with a second diagonal of 2 mm in diameter. The absence of extensive disease (<10 mm from the carina), severe stenosis (<90%), or moderate-severe calcification facilities the use of the provisional technique with a stent in this patient.

Conclusions

Wellens patterns in patients admitted for unstable angina with or without acute myocardial infraction include ST-T alterations with T-wave inversions. The syndrome identifies patients at imminent risk for extensive AMI, heart failure, and death. Therefore, patients in this context should undergo coronary angiography with the subsequent angioplasty as soon as possible.

Wellens waves in the high lateral leads (DI and aVL) could signify the involvement of a second lesion in a lateral branch. There is a need for more information to validate this hypothesis.

Contributions

All authors participated in the writing, revision of the manuscript, and approval of the final version.

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