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Wellens Syndrome: impending acute myocardial infarction

Síndrome de Wellens: o infarto agudo do miocárdio iminente

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ABSTRACT – Wellens syndrome was described by Zwaan, Bär and Wellens, in 1982. On the electrocardiogram, T wave morphological alterations are observed in precordial leads, suggesting severe proximal stenosis of the left anterior descending coronary artery and an imminent risk of acute myocardial infarction and/or sudden death, with slight or no alteration in serum troponin. Its early recognition and management with early myocardial reperfusion avoid unfavorable outcomes. Percutaneous coronary intervention and surgical myocardial revascularization are the most used strategies for treatment. The authors describe two cases of types A and B Wellens syndrome treated with different reperfusion strategies.

Keywords: Myocardial infarction; Myocardial revascularization; Coronary artery disease; Electrocardiography; Syndrome; Angioplasty, balloon, coronary

RESUMO – A síndrome de Wellens foi descrita em 1982 por Zwaan, Bär e Wellens. No eletrocardiograma, observam-se alterações morfológicas da onda T nas derivações precordiais, sugerindo estenose proximal grave da artéria coronária descendente anterior que representa iminente risco de infarto agudo do miocárdio e/ou morte súbita, com discreta ou nenhuma alteração da troponina sérica. Seu reconhecimento antecipado e a abordagem com reperfusão miocárdica precoce evitam desfechos desfavoráveis. A intervenção coronária percutânea e a revascularização miocárdica cirúrgica são as estratégias mais utilizadas para tratamento. Os autores descrevem dois casos de síndrome de Wellens tipos A e B tratados por diferentes estratégias de reperfusão.

Descritores: Infarto do miocárdio; Revascularização miocárdica; Doença da artéria coronariana; Eletrocardiografia; Síndrome; Angioplastia coronária com balão

INTRODUCTION

Wellens syndrome (WS) is an acute coronary syndrome (ACS) with an electrocardiographic pattern, characterized by the presence of an inverted and/or biphasic T wave in precordial leads associated with normal and/or slightly altered troponin, which is highly suggestive of severe proximal lesion in the left anterior descending artery (ADA).[1] It has a prevalence of 15% of all ACS.[2]

This electrocardiographic pattern described exactly four decades ago, by de Zwaan et al., is especially seen in V2 and V3 leads, and is related to a poor prognosis due to the imminent risk of acute myocardial infarction (MI) and sudden death.[1] Wellens syndrome may present with or without pain, but its early recognition and management with an immediate reperfusion strategy is extremely important to avoid drastic consequences.[3]

The authors reported two cases of types A and B WS, early diagnosed and successfully treated with percutaneous coronary intervention (PCI) and surgical myocardial revascularization (CABG).

CASE REPORTS

Case 1

A 62-year-old Black female, smoker, hypertensive, diabetic for 5 years, coming from a private medical office, where she reported tightening oppressive chest pain, which

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was intermittent 72 hours before admission, and had been asymptomatic in the previous 24 hours. The electrocardiogram showed sinus bradycardia and biphasic T waves in the anterior wall (Figure 1A). The patient was not medicated there, due to the unavailability of diagnostic tests and a specific technical approach, and was transferred to our clinic, with the presumptive diagnosis of ACS. Upon arrival, she had no complaint of chest pain. A new electrocardiogram showed sinus bradycardia, biphasic T waves in V2-V3 (Figure 1B); troponin level was 1.51ng/mL (reference value: 0.00 to 0.50), and the patient was admitted to the intensive care unit, stable and without pain.

The following day, a coronary angiography was performed, which revealed a severe 99% proximal lesion of the left anterior descending artery (Figure 2A). Percutaneous coronary intervention was performed using a 3.5x23mm Xcience Sierra™ drug-eluting stent, with final Thrombolysis in Myocardial Infarction (TIMI) III flow (Figure 2B). The patient was discharged 2 days later with dual antiplatelet therapy (aspirin and clopidogrel) and remained asymptomatic.

Case 2

A 63-year-old male, Black, smoker, hypertensive, and diabetic patient. He sought the Emergency Department for tightening oppressive angina, which had been evolving for 2 weeks, and had intensified 6 hours earlier, associated with tiredness, without triggering factors. The electrocardiogram showed sinus bradycardia with negative T waves in V1-V5 (Figure 3A); troponin level was normal, 0.026ng/mL (reference value: 0.00 to 0.50). Emergency coronary angiography revealed severe 99% proximal stenosis of the left anterior descending artery, involving the ostium (Figure 3B). Given the complexity of the approach, a left internal mammary artery-anterior descending artery bypass graft was successfully performed. The postoperative echocardiogram showed preserved biventricular systolic function, without segmental changes. The patient had a satisfactory clinical course.

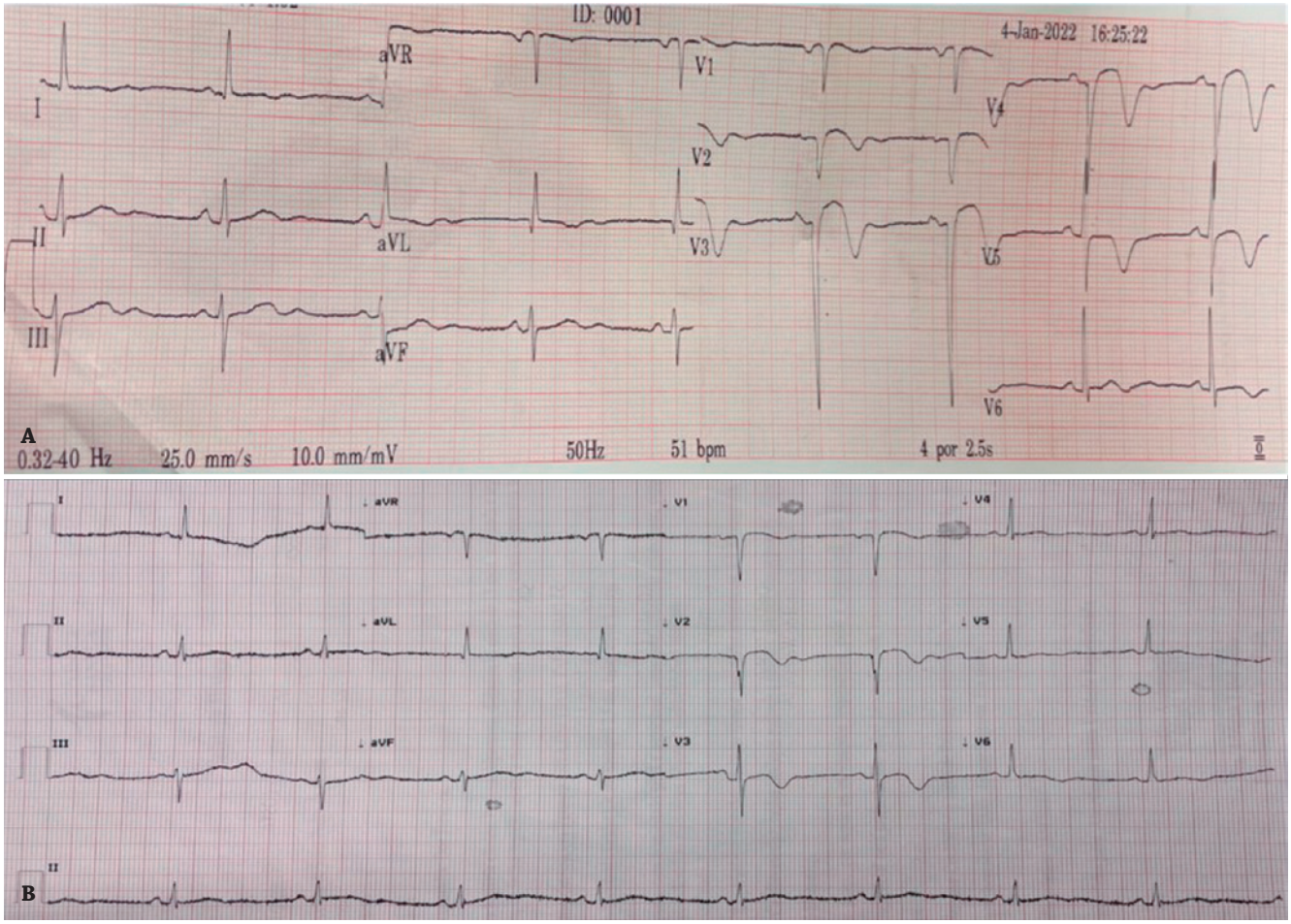

Figure 1. 12-lead electrocardiogram. (A) Sinus bradycardia, biphasic T waves (V2-V4). (B) Sinus bradycardia and biphasic T waves (V2 and V3) of type A Wellens syndrome.

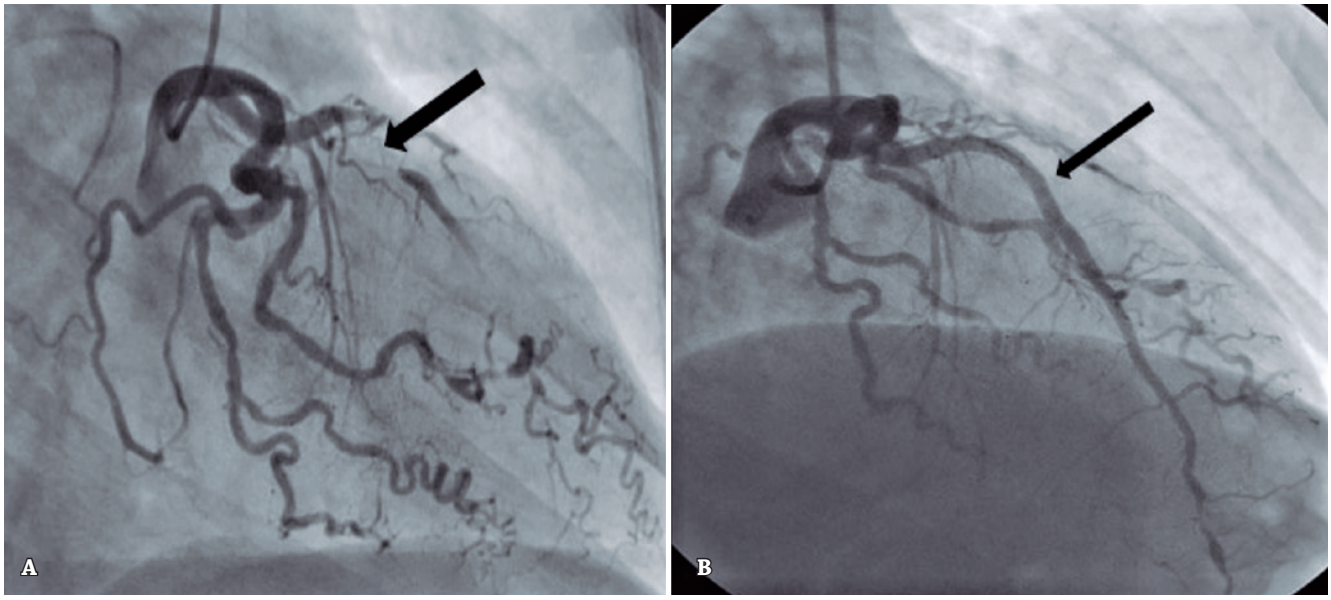

Figure 2. Coronary angiography performed by right transradial approach. (A) Right anterior oblique view at 37° and cranial view at 0.6°, where a 99% stenosis of the proximal left anterior descending artery is observed (arrow). (B) Right anterior oblique view at 38° with cranial view at 28°, after percutaneous coronary intervention with drug-eluting stent and restoration of coronary flow Thrombolysis in Myocardial Infarction III.

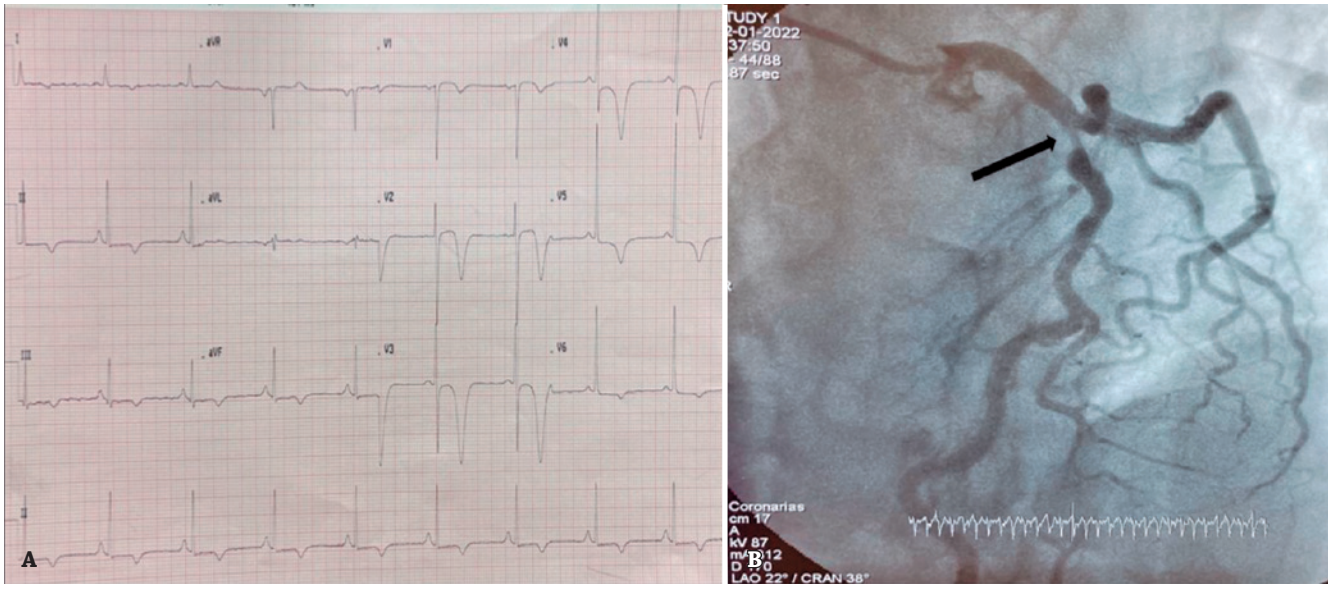

Figure 3. (A) Electrocardiogram showing sinus bradycardia and T wave inversion (V1-V6), of type B Wellens syndrome. (B) Coronary angiography performed by right transradial approach with left anterior oblique projection at 22° and cranial at 38°, in which a 95% stenosis of the proximal anterior descending artery is observed (arrow).

DISCUSSION

Since its first report four decades ago,[1] the WS electrocardiographic pattern suggestive of severe proximal anterior descending artery lesion remains a challenge frequently underestimated by emergency department physicians, especially when patients are asymptomatic at the time of admission,[4] which magnifies the risk of unfavorable outcomes, when the condition is not identified and treated with immediate myocardial reperfusion, even when patients are maintained in clinical treatment.[3,5]

Diagnostic criteria involve a history of angina, although there are reports of pain-free clinical presentation associated with mild or no changes in cardiac necrosis markers. The electrocardiogram may show a type A WS pattern (biphasic T waves in right anterior chest leads, V2-V3), found in about 24% of cases, or a type B WS pattern (negative, deep, and symmetrical T waves in V2-V3, and occasionally in V1, V4, V5 and V6, are most frequent).[1-3,5]

The differential diagnosis is varied. Other causes of altered T-wave morphology should be considered when approaching a patient with suspected WS, including brain "T's" due to central nervous system injury, left ventricular hypertrophy, right bundle-branch block, hypertrophic cardiomyopathy, pulmonary embolism, coronary vasospasm, and myocardial bridge.[5]

Although its etiology is established, its pathophysiological mechanism, which involves electrocardiographic changes, is still unknown, but hypotheses such as coronary spasm, myocardial stunning, and repetitive transmural ischemia-reperfusion causing myocardial edema may be the basis of this phenomenon, which represents a pre-AMI status.[5-7]

The electrocardiographic changes suggestive of WS are poorly known and generally underestimated, mainly because most patients are pain free on admission to the emergency department, impacting the late clinical-electrocardiographic correlation, early detection, and immediate specific treatment, increasing the chance of unfavorable outcomes.[7]

In case 1, the delay in performing a PCI involved several factors, such as nonspecific changes in the electrocardiogram, absence of pain, and slightly altered troponin level. After evaluation by the Interventional Cardiologist hours later, a PCI was successfully performed on the culprit vessel. The early invasive strategy by PCI or bypass grafting has satisfactory prognoses, and is superior to maintaining clinical treatment, which is associated with high short-term mortality rates.[3-6]

The patients described here had different treatment strategies. In case 1, the reperfusion strategy was via PCI, which is the most commonly reported one in several studies.[3,6] In case 2, in which there was evidence of severe proximal lesion of the anterior descending artery involving the left main artery, a bypass grafting was performed. The choice of reperfusion method selected depends on factors such as comorbidities, multivessel disease and complexity of the lesions.[5] The patient in question underwent a successful left internal mammary artery-anterior descending artery bypass and is followed up on a regular basis by cardiologists.

Wellens syndrome represents an impending AMI status. Its electrocardiographic pattern should not be underestimated even if the patient presents with no pain or negative troponin. Its early diagnosis and immediate specific treatment with myocardial reperfusion are associated with a better prognosis.

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None.

DECLARATION OF CONFLICTS OF INTEREST

The authors declare there are no conflicts of interest.

CONTRIBUTION OF AUTHORS

Conception and design of the study: MBAV; data collection: LMLD; data interpretation: LMLD, ELB and TCMP; text writing: LMLD and MBAV; approval of the final version to be published: MBAV, LMLD, AVAK, ELB and TCMP.

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