

Wellens syndrome associated with prominent anterior QRS forces: an expression of left septal fascicular block?

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Abstract

Wellens syndrome is a clinical-electrocardiographic entity also referred to as left anterior descending (LAD) coronary T-wave syndrome or acute coronary T-wave syndrome. It is a complex of symptoms and signals indicating the existence of an undesirable condition secondary to critical high-grade proximal stenosis of the LAD coronary artery characterized by the association of prior history of acute coronary syndrome with little or no elevation of markers of myocardial damage (unstable angina) and characteristic electrocardiographic changes consistent with subepicardial anterior ischemic pattern (persistently symmetrical, deep negative and broad-based T waves) or plus-minus T waves with inversion of the terminal portion in the LAD coronary artery territory (V1 through V5 or V6). We present a case of a variant of Wellens syndrome that reveals association and, transitorily, the criteria described in literature for left septal fascicular block.

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Keywords:

Wellens syndrome variant; Left septal fascicular block; Prominent anterior QRS forces

Introduction

Wellens syndrome is a clinical-electrocardiographic entity also referred to as left anterior descending (LAD) coronary T-wave syndrome or acute coronary T-wave syndrome. It is a complex of symptoms and signals indicating the existence of an undesirable condition secondary to critical high-grade proximal stenosis of the LAD coronary artery characterized by the association of prior history of acute coronary syndrome with little or no elevation of markers of myocardial damage (unstable angina) and characteristic electrocardiographic changes consistent with subepicardial anterior ischemic pattern (persistently symmetrical, deep negative, and broad-based T waves) or plus-minus T waves with inversion of the

terminal portion in the LAD coronary artery territory (V1 through V5 or V6).

We present a case of a variant of Wellens syndrome that reveals association and, transitorily, the criteria described in literature for left septal fascicular block.

Case report

We describe the case of a 76-year-old white woman who presented at the emergency department with typical coronary chest pain with accelerating or “in crescendo” pattern over the last week. Her pain was retrosternal in location, radiating to the internal portion of the left arm until the elbow and mandible, not precipitated by exercise, oppressive in nature and intermittent, and lasting 15 minutes.

Sublingual nitroglycerin relieved the pain.

Her medical history includes controlled high blood pressure and type 2 diabetes mellitus associated since a long time ago (both more than 20 years).

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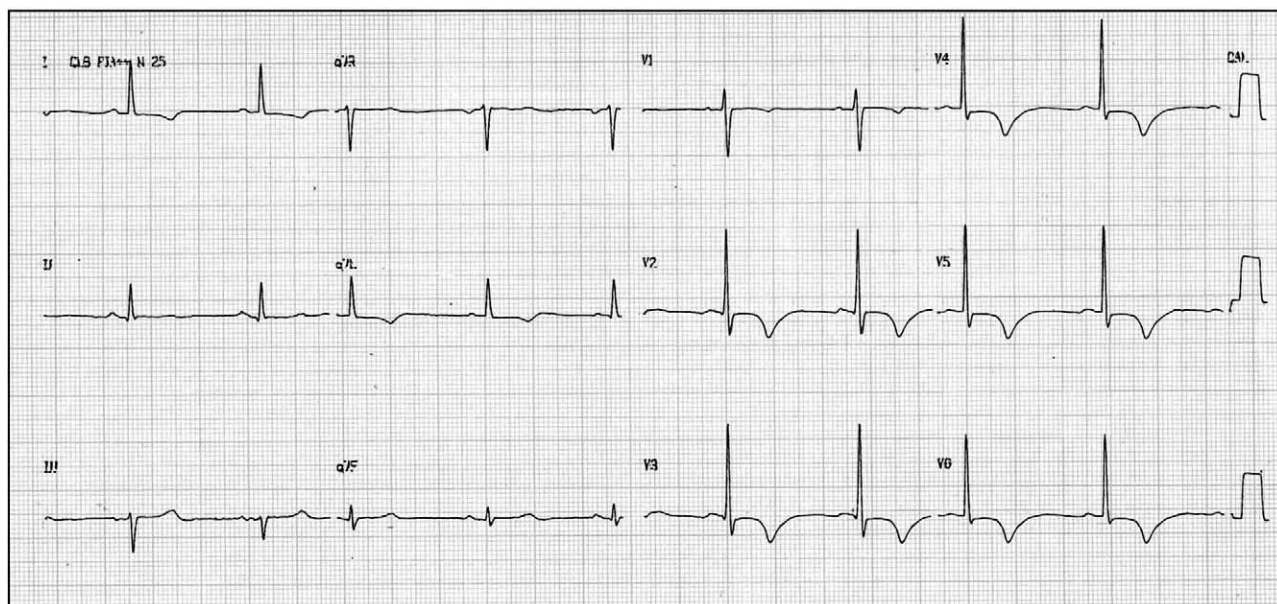


Fig. 1. Electrocardiogram performed upon arrival to the emergency department (April 29, 2008) and while having chest pain. Deep negative and broad-based T-wave inversions in precordial leads from V2 through V6, with high voltage R wave in V2 ($R = 18$ mm). Initial small q waves were observed in V2-V3. Left septal initial q waves in left leads are absent. R/S ratio in V2 > 2 . S wave depth in V2 < 5 mm. Conclusion: Type 2 Wellens pattern associated with prominent anterior forces; several left septal fascicular block criteria are present.

Her mother died of diabetes, and her father had sudden death at age of 70 years.

On admission, presence of a fourth heart sound (S_4) was heard between the left sternum border and the apex. Blood pressure was 140/85 mm Hg.

Electrocardiogram (ECG) performed upon arrival to the emergency department, and while having chest pain, revealed deep negative and broad-based T-wave inversions in precordial leads from V2 through V6, with high-voltage R wave in V2 ($R = 18$ mm). Initial small q waves were observed in V2-V3. Left septal initial q waves in left leads are absent (Fig. 1).

Serum markers of cardiac damage were within normal limits.

These ECG findings were consistent with Wellens syndrome; thus, the patient was urgently transferred to the cardiac catheterization laboratory, where she underwent coronary angiography. This showed 2-vessel coronary artery disease affecting the proximal portion of the LAD coronary artery with a 98% stenosis lesion and right coronary artery with 40% stenosis in the middle portion. In addition, the anterior wall of the left ventricle had minimally anterior hypokinetic motion.

The patient underwent a successful transluminal coronary angioplasty on the LAD with stent deployment.

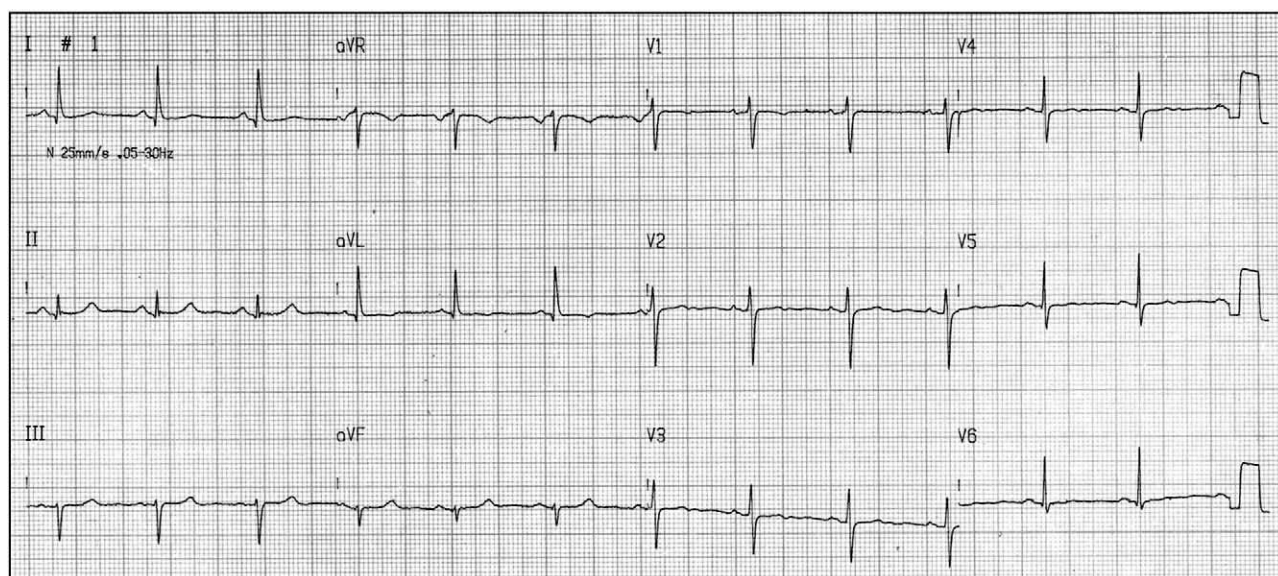


Fig. 2. Electrocardiograms performed 1 year before clinical manifestation (July 4, 2007). There are not subepicardial T-wave ischemic pattern and QRS complexes of the rS type in V2. Initial q wave are observed in left leads I, L, V5, and V6.

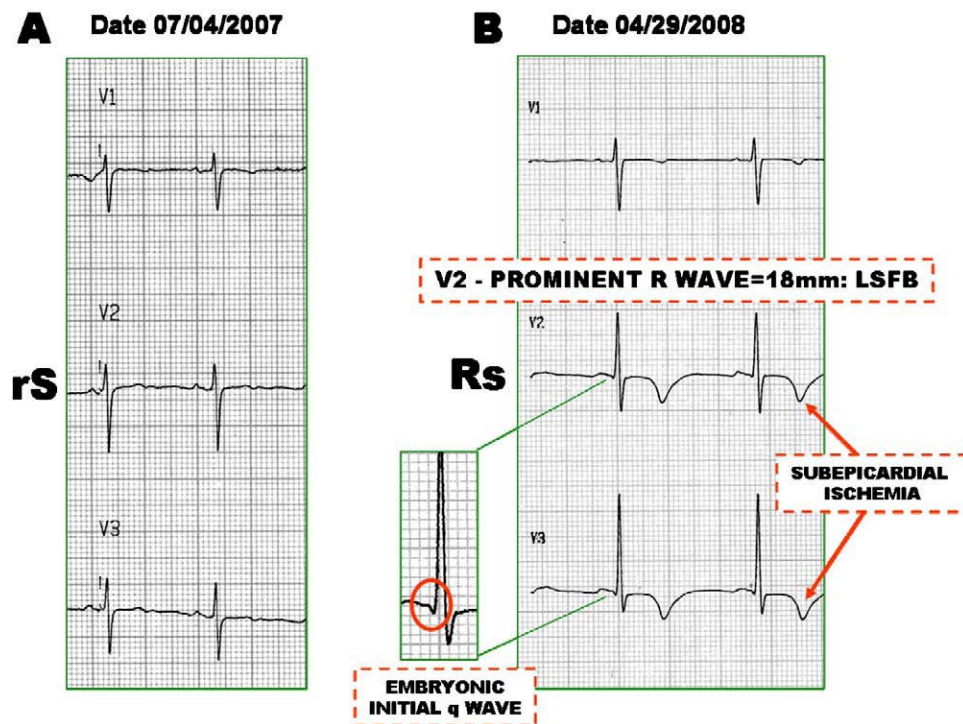


Fig. 3. A, Basal anteroseptal precordial leads performed approximately 1 year before onset of clinical picture. B, The same leads performed during the clinical manifestation (description in Fig. 1).

Three days after the angioplasty procedure, her daughter showed several ECGs from the previous year, without ischemic pattern and QRS complexes of the rS type in V2 (Fig. 2 shows the ECGs, and Fig. 3A and B the comparative tracings.)

Ten days after the successful placement of the stents, the ECG showed the ischemic pattern had disappeared, the lead V2 returned to rS, the initial q wave in V3 disappeared, and small q waves appeared in the left leads I, aVL V5, and V6 (Fig. 4).

Discussion

Wellens syndrome is observed in the context of acute coronary syndrome, and it manifests by plus-minus T waves with inversion of the terminal portion in leads V2–V6 (type 1). The anterior terminal inversion of T waves is called Wellens Warning. A second electrocardiographic presentation is symmetrical, deep T-wave inversion, both progressive and persistent (may remain in place for hours to weeks¹ especially in V₂ and V₃ [type 2]).²

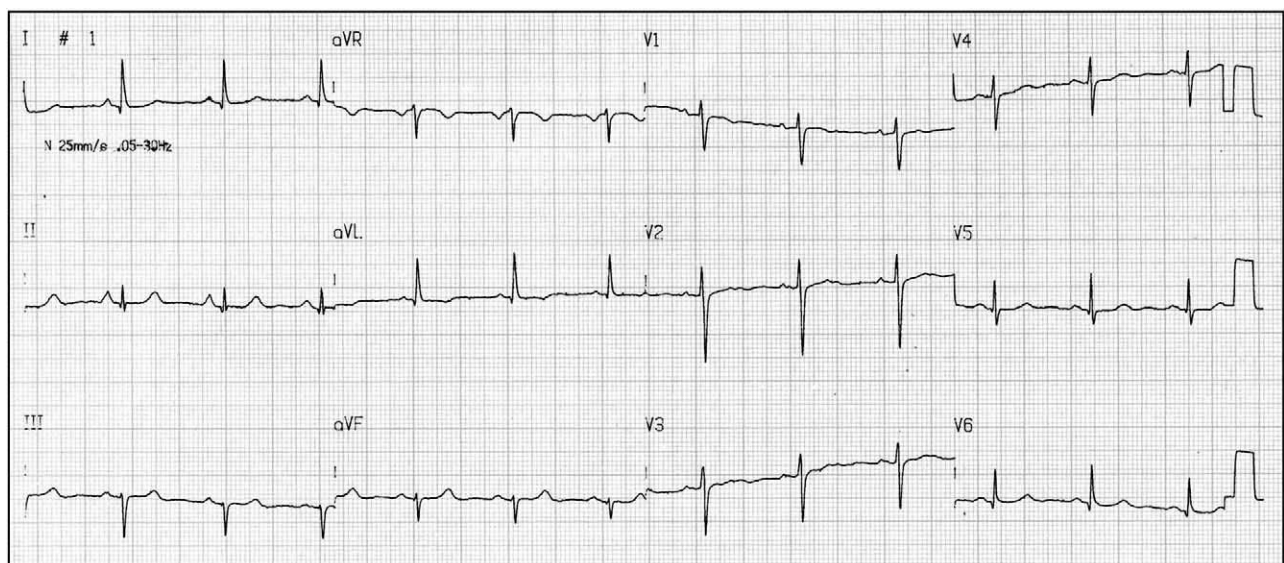


Fig. 4. Electrocardiogram performed ten days after (May 9, 2008) the successful placement of the stents in LAD. The ischemic pattern had disappeared, the lead V2 returned to rS, the initial q wave in V3 disappeared, and small septal q waves appeared in the left leads I, L, V5, and V6.

Both patterns are near-pathognomonic of that syndrome. Rarely, a change from type 2 to type 1 during observation is possible, and the coronary lesion located in the middle rather than the proximal part of the LAD artery.³ Note these ECG changes usually occur during a pain-free interval, when other evidence of ischemic or unstable angina may be absent. In view of the large area of myocardium at risk, the importance of recognizing the significance of this ECG pattern is of critical importance for the emergency physician, especially those involved in the evaluation of patients at emergency department chest pain centers.⁴ Thus, it is vital that this finding gets recognized promptly.

Others important features are critical myocardial ischemia with minor electrocardiograph changes, absence of pathological initial Q waves,⁵ little or no significant ST-segment elevation (≤ 1 mm) concave to the top or straight, normal precordial R-wave progression (no loss of precordial R waves), and little or no elevation of serum markers of cardiac damage.

The ECG changes usually occur during a pain-free interval when other evidence of ischemic or unstable angina may be absent. In addition, with definitive management of the stenosis, the changes resolve with normalization of the ECG.

The natural history of Wellens syndrome is anterior wall acute myocardial infarction. These patients should not have stress tests but rather emergent cardiac catheterizations.

The remarkable aspect of the present case is constituted by R wave voltage of V2 increasing, thus configuring an electrocardiographic pattern of anterior shift of electromotive forces of ventricular activation: prominent anterior forces. In addition, we observed the appearance of initial small q wave in V2-V3 and disappearance of initial septal q wave in left leads (I, VL, V5, and V6). These ECGs aspects that were absent in the ECG from the previous year make up the typical electrocardiographic pattern of left septal fascicular block (LSFB).

The left septal fascicle is irrigated exclusively by the septal perforating branches of the LAD. Critical lesions of the LAD before the first perforating septal constitute the main cause of LSFB in the developed countries.⁶

In most humans ($\approx 85\%$ of cases), the left bundle branch splits into tree fascicles. Electrocardiographic changes resulting from conduction abnormalities of the left anterior and left posterior fascicles manifest mainly in the frontal plane.

Conduction defects of the left septal fascicle manifest in the horizontal plane (precordial leads) by prominent anterior QRS forces (R greater than or equal to S in V1 and/or V2 leads), in the absence of lateral myocardial infarction according to the new terminology of the cardiac walls (denominate in near past dorsal infarction⁷), right ventricular enlargement, or type A Wolf-Parkinson-White syndrome, are related to intraventricular conduction disturbance, at times rate dependent.⁸

The main criteria for LSFB mentioned in literature are the following^{9,10}:

1. Increase of R wave voltage in V2: V2 R wave > 15 mm
2. R wave voltage of V1 ≥ 5 mm

3. Possible small initial q wave in V2 or V1, V2, or V3
4. Absence of septal initial q wave in left precordial leads I, V5, and V6 (secondary to absence of first Septal vector). The absence of septal Q waves is defined as the simultaneous absence of Q waves from all of leads I, V5, and V6. Absence of septal Q waves in otherwise normal ECGs may be a variant from normal.
5. Increased intrinsicoid deflection of V1 and V2
6. Normal QRS duration or with a discrete increase (up to 110 milliseconds)
7. Frontal plane leads with no modifications: normal QRS
8. R/S ratio in V2 > 2
9. S wave depth in V2 < 5 mm
10. RS or Rs pattern in V2 and V3 (frequent rS in V1) with R wave “in crescendo” from V1 to V3 and decreasing from V5 to V6.

Conclusion

The present case constitutes probably a new variant of Wellens syndrome that includes transient left septal fascicular block, manifested in ECG by increase of R voltage in V2, small q in V3, and absence of absent septal q wave in left leads in baseline ECG before the event. The case reinforces the need to consider the left branch of the His bundle functionally as trifascicular, with an electrocardiographic expression of its own.

References

1. Rhinehardt J, Brady WJ, Perron AD, et al. Electrocardiographic manifestations of Wellens' syndrome. *Am J Emerg Med* 2002; 20:638.
2. Wellens HJ. Isolated electrical alternant of the T wave. *Chest* 1972;62: 319.
3. Kardesoglu E, Celik T, Cebeci BS, et al. Wellens' syndrome: a case report. *J Int Med Res* 2003;31:585.
4. Tandy TK, Bottomy DP, Lewis JG. Wellens' syndrome. *Ann Emerg Med* 1999;33:347.
5. Kahn EC, Keller KB. Wellens' syndrome in the emergency department. *J Emerg Nurs* 1991;17:80.
6. Moffa PJ, Ferreira BM, Sanches PC, et al. Intermittent antero-medial divisional block in patients with coronary disease. *Arq Bras Cardiol* 1997;68:293.
7. Bayés de Luna A, Zareba W. New terminology of the cardiac walls and new classification of Q-wave M infarction based on cardiac magnetic resonance correlations. *Ann Noninvasive Electrocardiol* 2007;12:1.
8. Hoffman I, Mehta J, Hilsenrath J, et al. Anterior conduction delay: a possible cause for prominent anterior QRS forces. *J Electrocardiol* 1976;9:15.
9. MacAlpin RN. In search of left septal fascicular block. *Am Heart J* 2002;144:948.
10. Mori H, Kobayashi S, Mohri S. Electrocardiographic criteria for the diagnosis of the left septalfascicular block and its frequency among primarily elderly hospitalized patients]. *Nippon Ronen Igakkai Zasshi* 1992;29:293.