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**Case Report**

# Wellens Syndrome: A Case like No Other!

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## Abstract

***Introduction:*** Wellens syndrome or anterior interventricular artery (AIV) disease is an electrocardiographic abnormality defined by the presence, apart from painful episodes, of biphasic T waves in the anterior leads (type 1), or deeply inverted T waves (type 2) in association with critical stenosis of the proximal AIV. It warrants aggressive management given the rather high incidence of death and re-infarction. ***Objectif:*** To report an atypical form of this syndrome through this observation and to provide an update on this rare entity through a literature review. ***Case report:*** A 48-year-old patient, chronic smoker with 40 BPs still active, admitted following an episode of infarct chest pain. At admission, the patient was asymptomatic. The clinical examination was unremarkable. The ECG showed biphasic T waves, negative in its terminal part in V2-V3-V4, without planed R waves, nor Q waves of necrosis or significant ST abnormality evoking a WELLENS type 1 syndrome. Ultrasensitive troponin was measured at 260 ng/L. The evolution was marked by the appearance of a fleeting negativation of T waves, without chest pain. Chest echocardiography showed a non-dilated left ventricle with preserved systolic function and no detectable segmental or global kinetic disorders, associated with concentric left ventricular hypertrophy. The coronary angiography performed in emergency shows tritroncular lesions, a very tight stenosis of the middle interventricular and the first diagonal, a tight stenosis of the middle circumflex and the middle right coronary, revealing a coronary artery bypass graft. ***Discussion and conclusion:*** Wellens syndrome is characterized by T-wave abnormalities that often fluctuate (from type 1 to type 2 or vice versa) during pain-free intervals, reflecting spontaneous reperfusion and the unstable nature of the coronary lesion found, which is represented mainly by involvement of the proximal AVI. Involvement of the middle IVA associated with other lesions is rarely observed. It is associated with a greater risk of anterior infarction or sudden death if not recognized and treated rapidly, so it seems essential to recognize the electrocardiographic criteria of this syndrome in order to propose coronary angiography for early revascularization.

**Keywords:** Wellens syndrome, transient T wave abnormality, reperfusion, anterior interventricular artery, revascularization.



## INTRODUCTION

Wellens syndrome, also known as VIA syndrome, is an electrocardiographic abnormality defined by the presence, apart from painful episodes, of diphasic T waves with terminal inversion in V2-V3 (type 1) or deeply inverted T waves in V2-V3, rather thin and symmetrical (type 2), without associated necrosis Q wave, in association with a critical stenosis of the proximal VIA [1]. It justifies an aggressive management considering the incidence of death and re-infarction which is rather important.

Recognition of this pattern, and appropriate intervention, prevents a potentially devastating anterior wall myocardial infarction. Importantly for the emergency physician, Wellens' T-wave changes usually occur during a pain-free interval when other evidence of ischemia or unstable angina may be absent. Although these patients may initially respond well to medical management, they ultimately fare poorly with conservative therapy and require aggressive treatment.

Our objectif is to report an atypical form of this syndrome through this observation and to review the literature on this rare entity.

## CASE REPORT

The patient was 48 years old, with a chronic smoking habit at 40 BP, still active, and dyslipidemia under treatment. He had no particular personal or family pathological history, and was admitted to our clinic following an episode of chest pain, constrictive, retro-sternal, radiating to the left upper limb and jaw, not rythmed by exertion, prolonged, 5 days before his admission, without any other associated signs, in particular dyspnea or palpitations.

On admission, the patient was asymptomatic. The clinical examination was unremarkable. Hemodynamics were stable (blood pressure 143/67 mmHg, heart rate 65 bpm, trans-oxygen saturation 97% on room air).

The ECG showed negative bifid T waves in its terminal part in V2-V3-V4 (Figure 1) evoking a Wellens type 1 syndrome. Ultrasensitive troponin was measured at 260 ng/L.

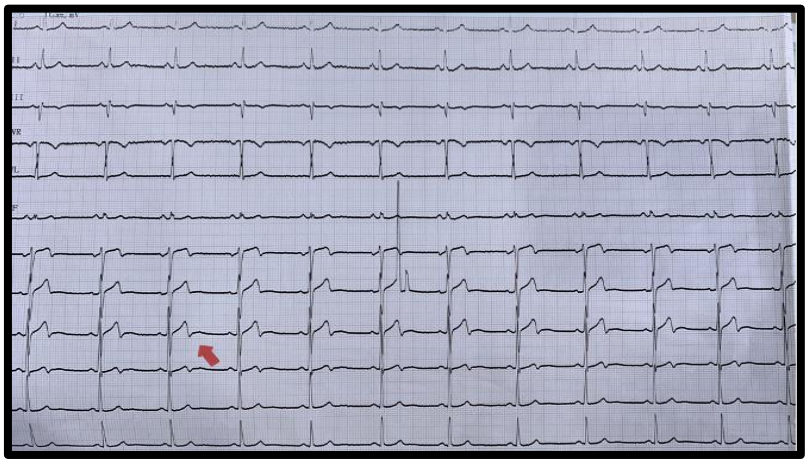

**Fig-1: ECG showed a negative bifid T wave in its terminal part in V2-V3-V4.**

The evolution is generally done with the appearance of a fleeting negativation of the T waves (type B) ( figure 2).

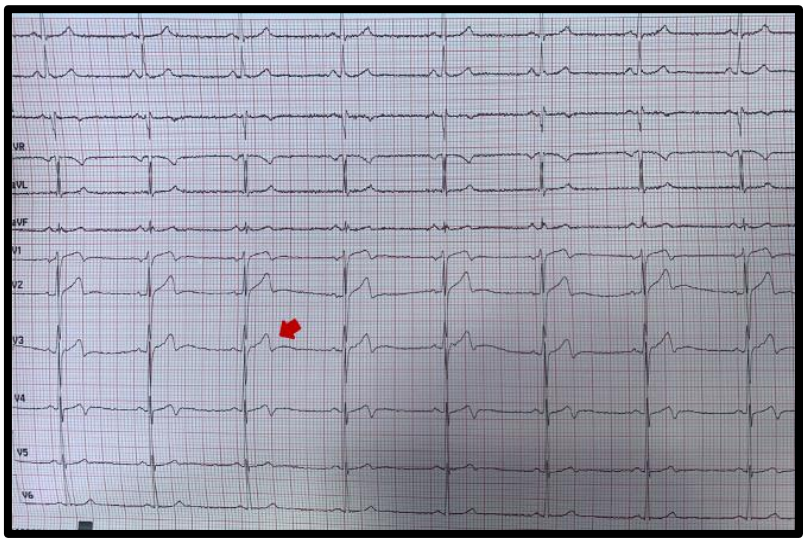

**Fig-2: ECG showed T-wave negativation in its terminal part in the same leads**

Thoracic echocardiography shows a non-dilated LV with preserved systolic function (LVEF= 67%), concentric left ventricular hypertrophy, with no detectable segmental or global kinetic disorders, and minimal mitral insufficiency.

Emergency coronary angiography showed tritruncal lesions, a very tight 90% stenosis of the middle interventricular, a 95% long ostial stenosis of the 1st diagonal, a 75% tight stenosis of the middle circumflex opposite the start of the second marginal, large caliber, and a 70% long and stepped tight stenosis of the middle right coronary (Figure 3), revealing coronary artery bypass surgery.

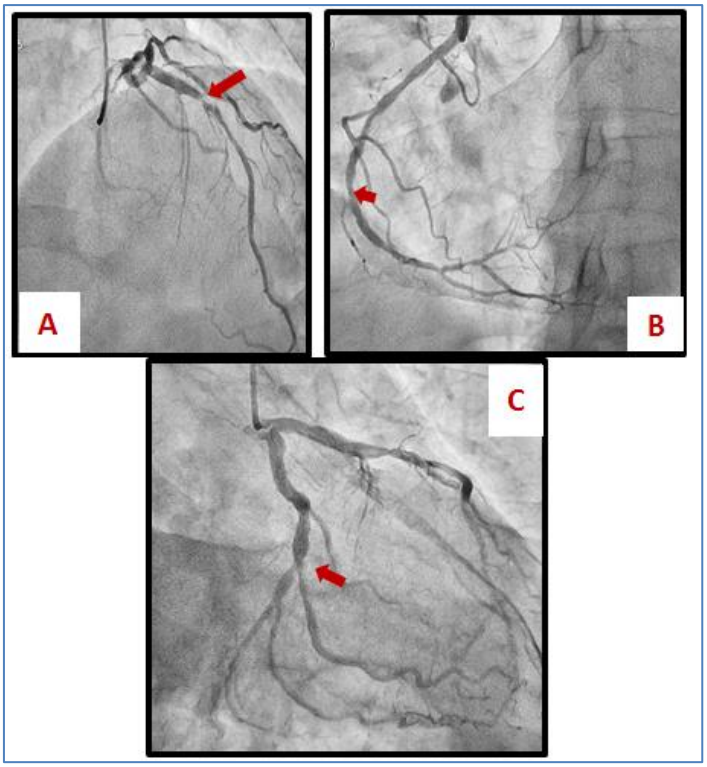


**Fig-3: Coronary angiography showing very tight stenosis of the middle VIA (A), tight stenosis of the CX2 (B) and CD2 (C).**

## DISCUSSION

Wellens syndrome described by HJ Wellens in 1982, defined by "cases of unstable angina associated with an inverted T wave in the anterior leads." This clinical and electrical presentation is associated with a greater risk of anterior infarction and/or sudden death in relation to critical anterior interventricular stenosis [2] ("VIA syndrome").

Wellens syndrome is characterized by T wave abnormalities that often fluctuate (from type 1 to type 2 or vice versa) during pain-free intervals, reflecting spontaneous reperfusion and the unstable nature of the coronary lesion found, which is represented mainly by involvement of the proximal AVI [3]. Involvement of the middle IVA associated with other lesions is rarely observed.

Simplified criteria for Wellens syndrome are as follows: history of chest pain, little or no elevation of cardiac enzymes, no pathological precordial Q waves, little or no ST-segment elevation, with no loss of precordial R waves, biphasic T waves in leads V2 and V3 or symmetrical, often deeply inverted T waves in leads V2 and V3[4].

**More precisely, WELLENS syndrome can be classified into 2 types**

- Type A: diphasic T waves with terminal inversion of T in V2 and V3, and constitutes 24% of cases
- Type B: deeply inverted T waves in V2 and V3, rather fine and symmetrical, the most frequent 76% of cases

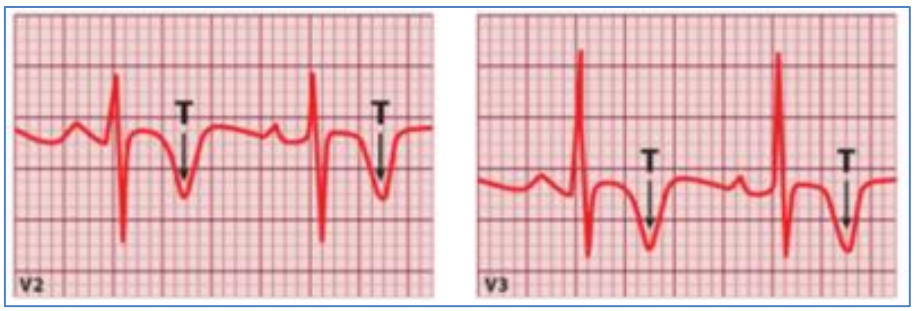


**Fig-4: Wellens syndrome type A**

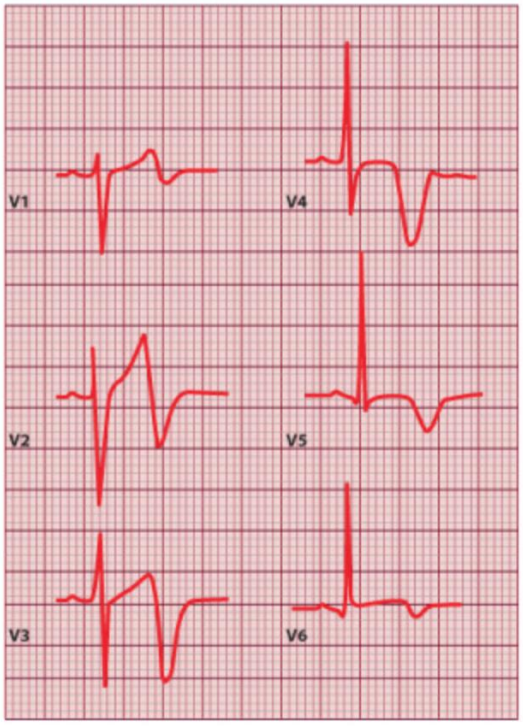


**Fig-5: Wellens syndrome type B**

Most patients remain asymptomatic at the time of consultation, but given the vascular involvement, there is a high risk of developing myocardial infarction and sudden death.

For this reason, patients require urgent angiography, given the potential lethality of coronary lesions. 75% of patients without catheterization develop an anterior infarction extended days after hospital admission [5].

The pathophysiology of the electrocardiogram changes associated with this syndrome has not been clearly defined, although related, on the one hand, to the possibility of myocardial stunning due to reperfusion from proximal occlusion of the descending pathway; but also with coronary spasm, right ventricular function overload or the presence of nonspecific repolarization disorders [6].

Therefore, as soon as Wellens syndrome is suspected, emergency coronary angiography is recommended rather than stress testing. It seems essential to recognize the electrocardiographic criteria of Wellens syndrome in order to propose coronary angiography for early revascularization, given the high morbidity and mortality in patients receiving drug therapy alone [3]. In addition, functional ischemic evaluations (stress test, stress myocardial CT, cardiac NMR, or stress echocardiography) should be avoided in these patients, as they may be dangerous in the presence of an unstable lesion on the proximal LAI [7].

## CONCLUSION

Wellens syndrome is associated with an increased risk of anterior infarction or sudden death if not recognized and treated promptly, so it seems essential to recognize the electrocardiographic criteria of this syndrome in order to immediately propose coronary angiography for early revascularization. Patients with Wellens syndrome should not be subjected to stress tests.

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