



An Interesting Case of Wellens Syndrome Dissimulating an Acute Coronary Sub-Occlusion

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ABSTRACT

Wellens syndrome is an electric pattern highly specific of critical stenosis of the proximal LAD. It's defined by abnormalities of T waves in V2-V3 : type 1 (biphasic T waves), type 2 (negative T waves). Wellens also does not show signs of infarction by definition.

Our work is about a young man of 33 yo who accused quickly resolving chest pain. His EKG showed biphasic T waves in V2 V3 V4 and V5 and negative T waves in DI aVL with 0,3mm ST suspension. His T waves kept changing: disappearing in V5 and becoming less negative in other derivations. The cardiac catheterization showed tight lesion of the ostium of proximal LAD artery with a high thrombotic load. Patient was put under glycoprotein IIb/IIIa inhibitors and taken back to the cath-lab 1 week after for PCI (percutaneous coronary intervention) which was successful.

First we want to discuss electrical findings. T waves inversion extended to V4 V5, which is uncommon. We have also to put forward the abnormalities we found in high lateral territory. Other particularity of our patient is the dynamic EKG showing he was threatening.

Furthermore, definitive treatment typically involves cardiac catheterization with PCI to relieve the occlusion and there's no place for stress tests.

Wellens syndrome is a real challenge, which is underdiagnosed and needs more attention from emergency physicians. The interest of thrombolysis in this pre-infarction condition still needs to be substantiated.

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INTRODUCTION

Wellens syndrome is an electric pattern highly specific of critical stenosis of the left anterior descending coronary artery in her ostio-proximal portion. It's defined [1] by abnormalities of T waves in V2-V3 derivations: type 1 (biphasic T waves with terminal inversion) and type 2 (deep, fine and symmetrical negative T waves), type 1 to 2 evolution, sometimes V1 V4 V5 or V6 derivations. The incidence [2] of Wellens syndrome is certainly under diagnosed (around 5,7% of acute coronary syndrome). Wellens presents little or no ST-segment elevation, normal evolution of precordial R waves and no necrosis Q waves. It represents a pre-infarction state that requires urgent assesment and should directly lead to cardiac catheterization.

CASE PRESENTATION

Our work is about a young man of 33 yo, chronic

active smoker, no other drug use and no particular medical history. He accused a first episode of chest pain 1 month before admission that he neglected, then, the day he came to our department, the patient accused one hour of more intense chest pain at rest, followed by angina that occurred for any effort. So he was admitted for unstable angina. The patient presented pain free at rest. His EKG showed biphasic T waves in V2 V3 V4 and V5 and negative T waves in DI aVL with 0,3mm ST suspension (figure 1). His T waves kept changing: disappearing in V5 and becoming less negative in other derivations (figure2). He went to the cath-lab one hour after admission.

The cardiac catheterization showed tight lesion of the ostium of the proximal left anterior descending (LAD) artery with a high thrombotic load (figure 3). Patient was put under glycoprotein IIb/IIIa inhibitors and taken back to the cath-lab 1 week after for PCI (percutaneous coronary intervention) which was successful (figure 4).

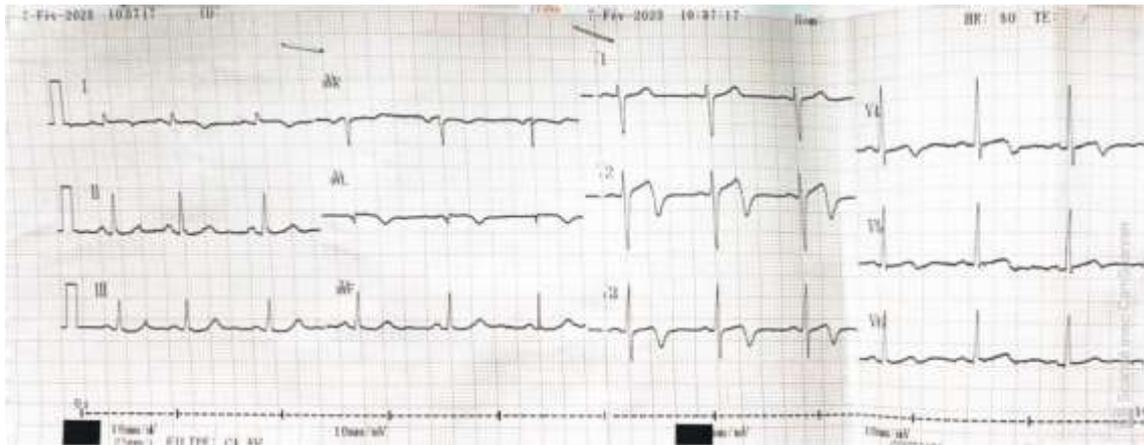


Figure 1: EKG 6 hours after beginning of pain

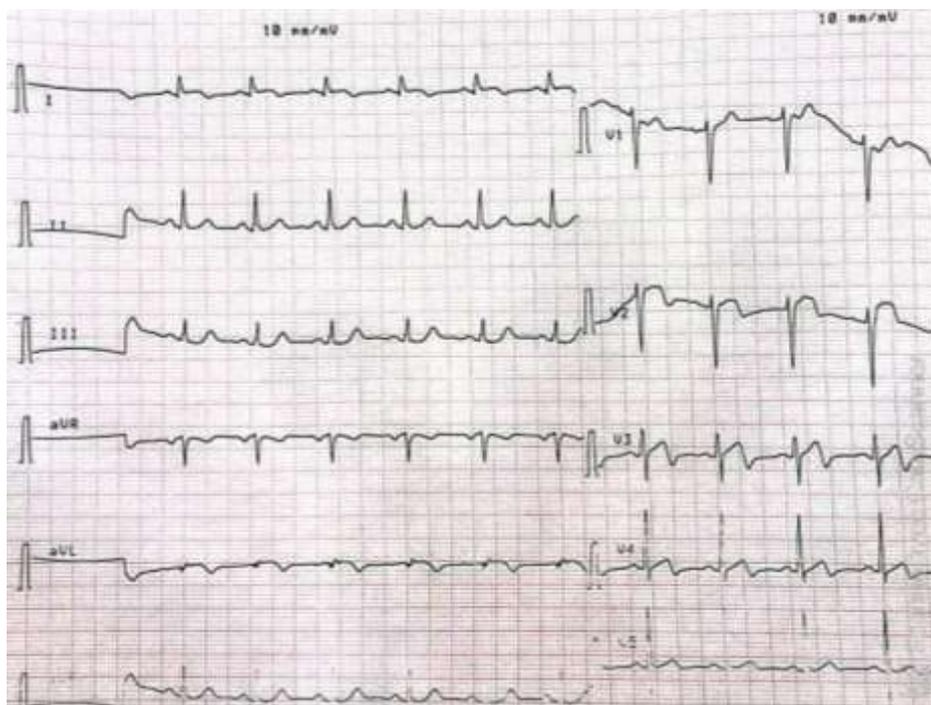
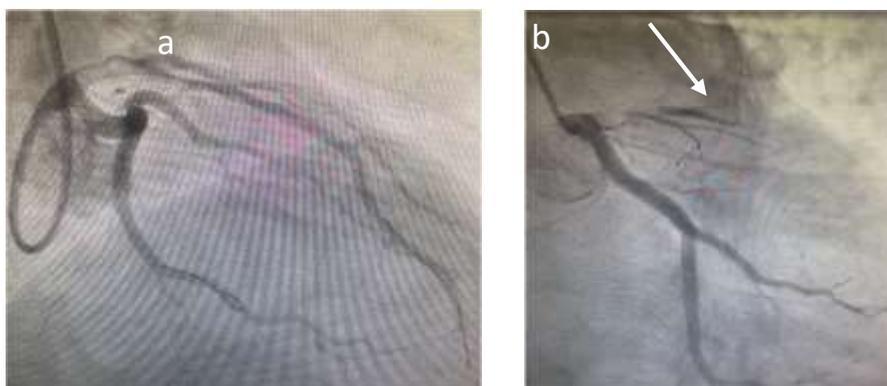
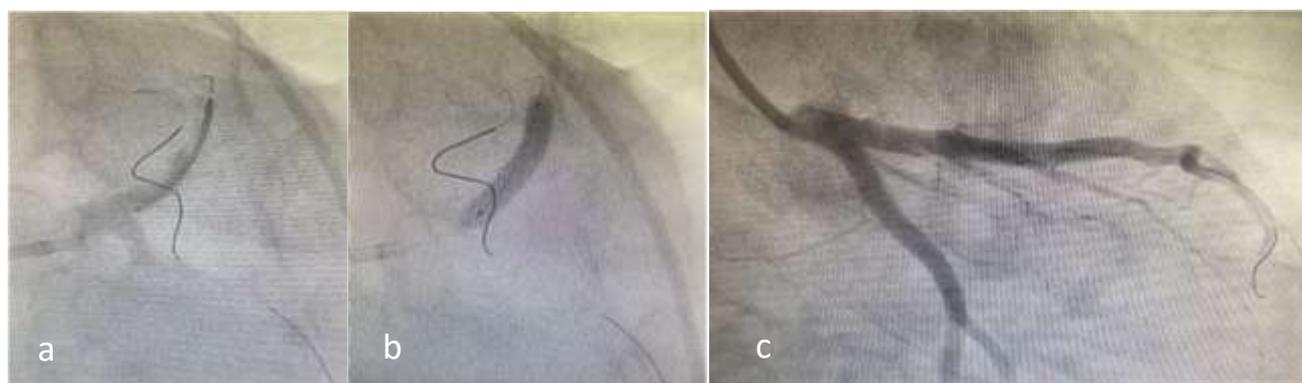


Figure 2: EKG 16 hours after beginning of pain



a: arrow showing thrombotic lesion on LAD b: arrow showing TIMI 0 flow grade

Figure 3: Angiographic images 17h after beginning of pain



a: stent placement

b : stent inflation

c : final result

Figure 4: Angiographic images one week after showing stenting of the lesion

DISCUSSION

First we want to discuss the electrical findings:

- T waves inversion are extended to V4 and V5, description of the pattern says it is common in V4 but uncommon in V5 [3].

- We have also to put forward the abnormalities we found in high lateral territory since we asked ourselves if it was a beginning infarction in this territory. Gerson in 1980 [1] defined similar findings in DI aVL and V4->V6 (describing negative U waves instead of biphasic T waves). There were 27 patients with this « negative U wave » and 89% of them had critical occlusion in LAD artery.

- Other particularity of our patient is the changes between EKGs showing he was threatening on the LAD.

Moreover, the angiographic aspect of the lesion makes us think deeply about the place of thrombolysis in wellens syndrome which constitutes an atypical form of STEMI even if it's not considered as a true STEMI equivalent. However, no study was found comparing PCI and thrombolysis in STEMI equivalents, except for ischemic left bundle branch bloc where thrombolysis has all its place.

Further more, should we really discuss place of stress tests in this type of patients? De Zwaan and colleagues in 1982 took 145 patients with unstable angina, 26 patients met the criteria of this syndrome. Among them, 16 were followed only with medication: 75% in this group presented extensive anterior myocardial infarction a few weeks after [4].

Therefore, management and definitive treatment typically involves cardiac catheterization with PCI (2) to relieve the occlusion and this is a rule we can't question. However, in the case of asymptomatic patients that come with same T waves abnormalities in their routine EKG but no history of pain; these patients don't join any standardized rules but advanced diagnosis methods can be proposed such as stress tests [3].

CONCLUSION

Wellens syndrome is a real challenge, which is underdiagnosed and needs more attention from emergency physicians. The interest of thrombolysis in this pre-infarction

condition still needs to be substantiated. Like we saw in our case, the thrombotic load can be very high and maybe thrombolysis would have some advantages, especially in conditions of no-availability of cath-lab immediately.

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