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A CASE REPORT OF AN ATYPICAL DYNAMIC WELLENS PATTERNS

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ABSTRACT

Wellens' syndrome is characterized by T-wave changes in electrocardiogram (EKG) during pain-free period in a patient with intermittent angina chest pain. It carries significant diagnostic and prognostic value because this syndrome represents a pre-infarction stage of coronary artery disease involving proximal left anterior descending (LAD) artery, which can subsequently lead to extensive anterior myocardial infarctions (MIs) and even death without coronary angioplasty. Therefore, it is crucial for every physician to recognize EKG features of Wellens' syndrome in order to take appropriate immediate intervention to reduce mortality and morbidity for MI. Here, we report a case of a man with a hyperlipidemia cardiac risk factor, diabetes mellitus, presented to the Hospital with intermittent pressing chest pain who was found to have type 1 Wellens' sign, which was biphasic T-waves in precordial leads V2 and V3 during pain-free period with minimally cardiac enzymes elevation. He was given therapeutic lovenox and subsequently underwent coronary angioplasty and had 95-99% occlusion in proximal LAD artery. The unique feature of our case was that Wellens' type 1 EKG changes were seen before reduction of stenosis with LAD artery stent, which was likely explained by the reperfusion of the ischemic myocardium. The EKG was normalized after angioplasty. Therefore, it is important for physicians to recognize EKG features of Wellens' syndrome in order to take appropriate therapy to reducing mortality and morbidity from impending MI.

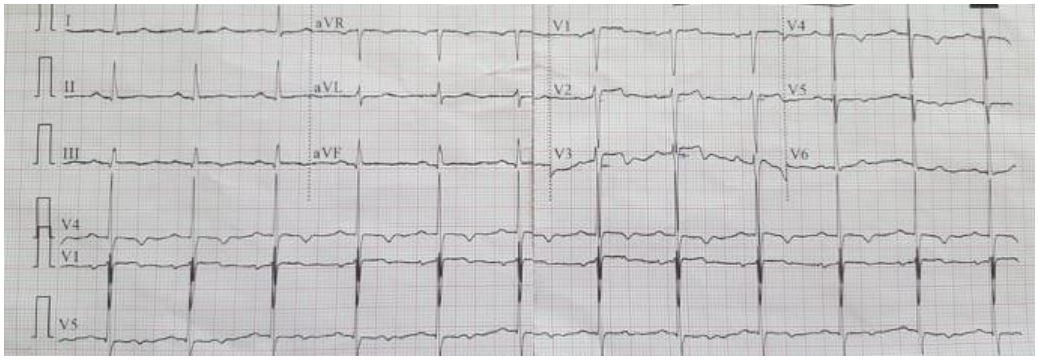
Key words: Wellens' syndrome, EKG, left anterior descending, dynamic wellens patterns.

I. INTRODUCTION

First described in 1982 by Hein J.J. Wellen and colleagues, Wellens pattern represents a temporal relationship between an initial ischemic event in the context of coronary artery disease and the point at which the ECG is captured [1]. Wellens pattern is not a rare phenomenon, occurring in 14%-18% of patients admitted to hospital for unstable angina [2]. The classical ECG finding (type 2 Wellens pattern) accounting for 75% cases is a minimally elevated or isoelectric ST segment with deeply inverted T waves in leads V2 and V3[3]. By contrast, a type 1 pattern is observed in around 25% of cases and is characterized by biphasic T-waves (with initial positivity and terminal negativity) in leads V2 and V3[3]. Despite symptomatic relief from medical management, the unresolved high-grade stenotic lesion often degenerates rapidly into an acute anterior wall myocardial infarction (MI), left ventricular dysfunction, arrhythmias, or death [4-6]. Therefore, the definitive treatment for patients presenting with Wellens pattern is cardiac catheterization.

II. CASE REPORT

A 68-year-old male with hyperlipidemia, impaired fasting glucose, with no known cardiac disease, presented with a one week history of intermittent exertional chest discomfort without radiation. Symptoms included dyspnea. The pain settled soon after his admission. The patient was diagnosed with a high risk NSTEMI. On admission, high-sensitive Troponin I (hsTnI) level was 3.9 ng/L (normal value < 0.1 ng/L). The initial electrocardiogram (ECG) captured after resolution of chest discomfort revealed sinus rhythm with biphasic T-waves in V1-V3 and T-wave inversion in V4(Figure 1A), consistent with a type A Wellens pattern. 24 hours after admission, hsTnI increased to 44 ng/L and a repeated ECG demonstrated biphasic T-wave in V1 and new onset T-wave inversions in V3-V5 (Figure 1B) a third ECG showed deep T-wave inversions inV2 –V5 (Figure 2) consistent with a type B Wellens pattern. The patient denied any chest pain or discomfort during the course of admission. His initial treatment included aspirin (300 mg), clopidogrel (300 mg) and heparine . Cardiac catheterization demonstrated critical stenosis of left anterior descending artery (ostial and proximal stenosis). Three drug-eluding stents were deployed and TIMI grade 3 flow was achieved. A final ECG demonstrated normalization of the ECG findings (Figure 3).



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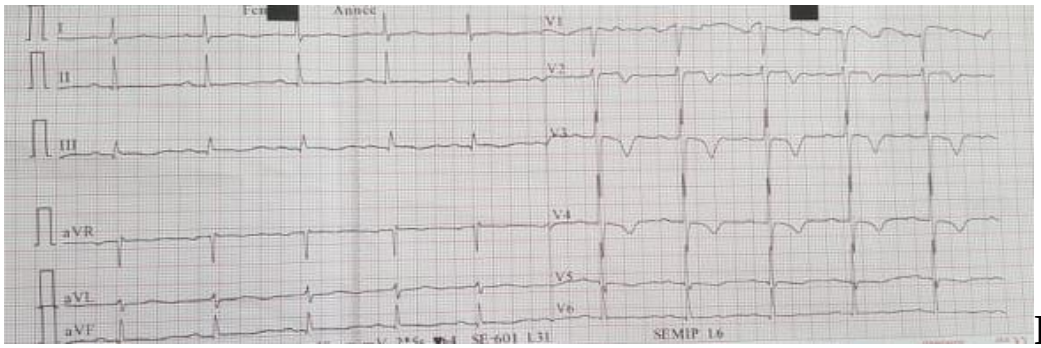

B

Figure 1. Initial electrocardiogram. (A) : Captured during a period of no chest discomfort, revealed sinus rhythm with biphasic T-waves in V1-V3, ST elevation in V1-V2-V3 (1mm), T-wave inversion in V4. Consistent with Wellens pattern Type 2. (B): biphasic T-wave in V2 and new T-wave inversions in V 3-V4.

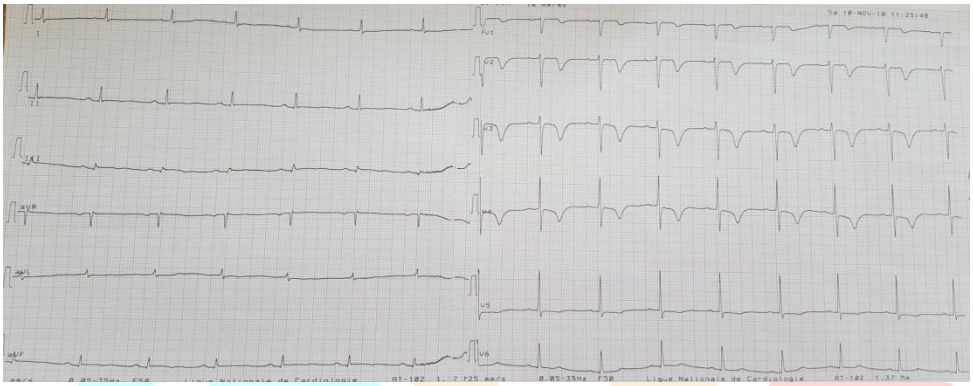

Figure 2. T-wave inversions in V2, V3,V4 andV5; biphasic T-wave V1. Consistent with Wellens pattern type 1.

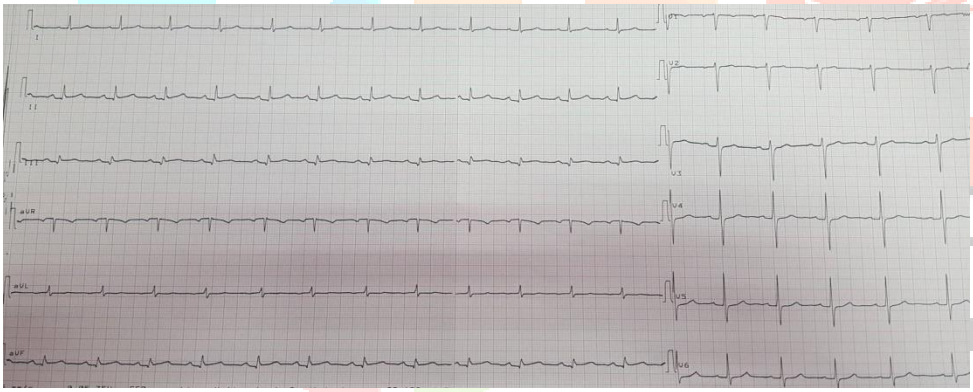

Figure 3. Electrocardiogram after cardiac catheterization and the placement of three drug-eluding stents: normal sinus rhythm without any T-wave inversions or biphasic T-waves.

III. DISCUSSION

Table 1. Clinical and electrocardiographic criteria for Wellens pattern. [7,1,8]

Biphasic or deeply inverted T-waves in leads V_2–V_3 but sometimes found in leads V_1 and V_4–V_6
Minimal or no ST-segment elevation (< 1 mm)
No loss of precordial R-wave progression
Absence of precordial Q waves
Minimal or no elevation of cardiac enzymes
History of angina
Observation of findings on the painless period

The mechanism of these dynamic ECG changes remains obscure. Some studies have shown that these types of ECG abnormalities may be caused by stunning and reperfusion of the myocardium [9,10]. Severe LAD obstruction causes myocardial ischaemia and angina [11]. When coronary flow is compensated by the collateral circulation, repolarization abnormalities caused by reperfusion injury result in T-wave symmetrical inversion or biphasic T-waves in the precordial leads [9]. A study conducted by Migliore et al [21] showed that myocardial oedema rather than stunning and reperfusion of the myocardium underlies the ECG changes of Wellens' syndrome. However, more studies are needed to explain this phenomenon.

In summary, ECG changes other than the classic ST elevations and depressions can indicate coronary artery disease, and patients presenting with atypical ECG patterns may also benefit from prompt catheterization.

This case illustrates the importance of recognizing nonconventional ECG presentations of severe coronary artery disease; the dynamic nature of such ECG manifestations that may reflect a spectrum of the ischemic processes; and the value of awareness among clinicians to help make correct and timely decisions to maximize myocardial salvage. Early identification of atypical ECG changes is important because Wellens pattern often occurs in asymptomatic patients with potentially normal cardiac biomarker levels.

Nevertheless, these are high-risk patients who require urgent angiography and revascularization to avoid evolution to acute anterior MI and possible sudden death.

IV. CONCLUSION

In conclusion, Wellens' syndrome always involves severe occlusion of the proximal LAD artery, but has a wide spectrum of clinical manifestations. Therefore, ECG parameters should be combined with cardiac catheterization to confirm the lesions; improve survival and to prevent complications.

Competing interests

The authors declare no competing interest.

REFERENCES

1. de Zwaan C, Bar FW, Wellens HJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. *Am Heart J* 1982; 103: 730–736.
2. de Zwaan C, Bar FW, Janssen JH, et al. Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery. *Am Heart J* 1989; 117: 657–665.
3. Rhinehardt J, Brady WJ, Perron AD, Mattu A. Electrocardiographic manifestations of Wellens' syndrome. *Am J Emerg Med* 2002; 20: 638–643.
4. Hollar L, Hartness O, Doering T. Recognizing Wellens' syndrome, a warning sign of critical proximal LAD artery stenosis and impending anterior myocardial infarction. *J Community Hosp Intern Med Perspect* 2015; 5: 29384.
5. Chi WK, Gong M, Bazoukis G, et al. Impact of coronary artery chronic total occlusion on arrhythmic and mortality outcomes: a systematic review and meta-analysis. *JACC Clin Electrophysiol* 2018; 4: 1214–1223.
6. Chi WK, Liu T, Nombela-Franco L, Tse G. The impact of chronic total occlusion in non-infarct-related coronary arteries. EuroIntervention. 2018.
7. Rhinehardt J, Brady WJ, Perron AD, Mattu A. Electrocardiographic manifestations of Wellens' syndrome. Am J Emerg Med 2002; 20(7): 638_43.
8. Balasubramanian K, Balasubramanian R, Subramanian A. A dangerous twist of the 'T' wave: A case ofWellens' Syndrome. Australas Med J 2013; 6(3): 122_5.
9. Abulaiti A, Aini R, Xu H, et al. A special case of Wellens' syndrome. J Cardiovasc Dis Res 2013; 4: 51–54.
10. Dhawan SS. Pseudo-Wellens' syndrome after crack cocaine use. Can J Cardiol 2008; 24: 404.
11. Eerdekens R, Chavez JF, Fox JM, et al. Predicting the infarct-related artery in STEMI from the surface ECG: independent validation of proposed criteria. EuroIntervention 2017; 13: 953–961.
12. Migliore F, Zorzi A, Marra MP, et al. Myocardial edema underlies dynamic T-wave inversion (Wellens' ECG pattern) in patients with reversible left ventricular dysfunction. Heart Rhythm 2011;8: 1629–1634.