WELLENS' SYNDROME, A PRESENTING SIGN OF LAD OCCLUSION : A CASE REPORT

Muhammad Hafiizh Alfarrisi[1], Michael[2], Emir Yonas[3], Raymond Pranata[4]
Department of Cardiology and Vascular Medicine, Faculty of Medicine Universitas Indonesia, National Cardiovascular Center Harapan Kita, Jakarta, Indonesia;
Faculty of Medicine Universitas Indonesia
Faculty of Medicine, Universitas Kristen Krida Wacana
Faculty of Medicine, Universitas YARSI, Jakarta, Indonesia
Faculty of Medicine, Universitas Pelita Harapan, Tangerang, Indonesia

ABSTRAK

Latar belakang: Di era pentingnya tindakan reperfusi, ada beberapa pola EKG atipikal yang dapat mengancam jiwa sindrom koroner akut risiko tinggi yang perlu diperhatikan. Salah satunya adalah sindrom Wellens dengan karakteristik adanya abnormalitas gelombang T (gelombang T biphasic atau T dalam terbalik) pada hasil elektrokardiogram (EKG) pasien dalam episode tanpa nyeri dada. Hal ini menunjukkan stenosis pada arteri koroner proksimal anterior descending kiri (LAD) derajat tinggi yang dapat mengakibatkan infark akut pada dinding anterior miokard (AMI) jika tidak dilakukan pengobatan maupun reperfusi.

Tujuan: Untuk menyajikan kasus Wellens sindrom yang mengancam jiwa, sindrom koroner akut risiko tinggi.

Ilustrasi kasus: Seorang pria berusia 48 tahun, perokok berat, datang ke ruang gawat darurat rumah sakit National Cardiac Center Harapan Kita (NCCHK) dengan nyeri dada berulang dalam waktu 18 jam sebelum datang ke rumah sakit. Pemeriksaan EKG menunjukkan irama sinus dengan T negatif yang dalam di V2-V4, tanpa gelombang Q patologis. Pasien diduga sebagai Wellens Sindrom, sindrom koroner akut dengan risiko tinggi. Pasien kemudian dilakukan intervensi koroner perkutan dini (PCI). Ditemukan 90% sumbatan pada LAD proksimal dan berhasil di lakukan pemasangan satu stent.

Kesimpulan: Semua pasien dengan/ tanpa riwayat angina dengan EKG yang dicurigai sebagai sindrom Wellens harus menjalani terapi invasive reperfusi sesegera mungkin. Setiap pasien dengan temuan EKG khas Sindrom Wellens tidak boleh menjalani segala bentuk tes jantung lainnya untuk menegakan diagnostik lebih lanjut karena risiko terjadinya kematian jantung mendadak.

Kata kunci: Sindrom koroner akut risiko tinggi, sindrom Wellens, obstruksi arteri *descending* kiri anterior; revaskularisasi, perubahan Elektrokardiografi

ABSTRACT

Background: In the era of the early reperfusion importance, there are some life-threatening atypical ECG patterns of high risk acute coronary syndrome need to be concerned. One of them is Wellens' syndrome characterized by T-wave abnormality (biphasic T wave or deep T inverted) on a patient's electrocardiogram (ECG) result during free chest pain. This finding suggests high-degree stenosis of proximal left anterior descending (LAD) coronary artery that will soon result in an acute anterior wall myocardial infarction (MI) if not reperfused and treated.

Objective: To present a case of life-threatening Wellens' syndrome, a high risk of acute coronary syndrome.

Case illustration: A 48-year-old male, heavy smoker, came to the emergency room of NCCHK hospital with recurrent chest pain within 18 hours before admission to hospital. ECG showed sinus rhythm with deep T inverted

Address for corespondance :
ina.j.chest@gmail.com

How to cite this article :
WELLENS' SYNDROME, A PRESENTING SIGN OF LAD OCCLUSION : A CASE REPORT

in V2-V4, without pathological Q waves. The patient was assessed as Wellens' Syndrome, a high risk of acute coronary syndrome. The patient was then performed early percutaneous coronary intervention (PCI). There was a 90% proximal LAD occlusion and being successfully implanted with one drug-eluting stent.

Conclusion: All patients with/without a history of angina and ECG suggesting a Wellens' syndrome should undergo an urgent invasive reperfusion therapy. Any patient with typical ECG findings of Wellens' syndrome must not undergo any form of cardiac stress test for further diagnostic tests due to the danger of sudden cardiac death.

Keyword: High risk acute coronary syndrome, Wellens' Syndrome, left anterior descending artery obstruction, revascularisation, electrocardiographic changes

INTRODUCTION

Acute myocardial infarction continues to be the most common cause of death globally. Acute ST-Elevation pattern in ECG is already known to be associated with acute total occlusion of coronary artery and demand revascularization as early as possible (primary PCI).[1] However, there are atypical life-threatening patterns of ECG that equivalent to high risk acute coronary syndrome (ACS) that also demand early invasive strategy. Electrocardiography patterns that considered with high risk critical coronary artery stenosis are Wellens' Syndrome, isolated posterior STEMI, hyperacute T wave, Sgarbossa criteria of LBBB (criterion 1, 2, & 3), "shark fin", De Winter Syndrome, and acute ischemia in LVH.[2] Accurate and early diagnosis would prevent unwanted morbidity and mortality.

CASE ILLUSTRATION

A 48-year-old male came to the emergency room of NCCHK hospital with recurrent chest pain since 18 hours before admission. Chest pain was on retrosternal, sharp sensation, radiated to his left arm with ± 30 minutes duration while he was sitting, with diaphoresis and vomiting. It was accompanied by diaphoresis and vomit. At the time of presentation, the chest pain did not abate with nitrate. And the patient decided to go to NCCHK. No dyspnea and no history of syncope. The VAS Score was 4/10. No history of ripped-like chest pain, chest trauma, prolonged cough, and prolonged immobilization. Previously, the patient had experienced multiple episodes of chest pain that improved with the use of nitrates.

He was an active smoker, 2 packs of cigarettes/day for the last 30 years (60 packs years). He had history of hypertension without medication but had no history of diabetes mellitus, dyslipidemia or history of cardiovascular disease in his first-degree relatives. He had no previously known disease and routine medicines. He also never had regular exercise.

The patient was fully alert and aware. His vital sign showed the blood pressure 166/100mmHg, heart rate 69 bpm, respiratory rate 18 x/m, and peripheral oxygen saturation 99%. Cardiac physical examination showed normal heart sound, no murmur or gallop.

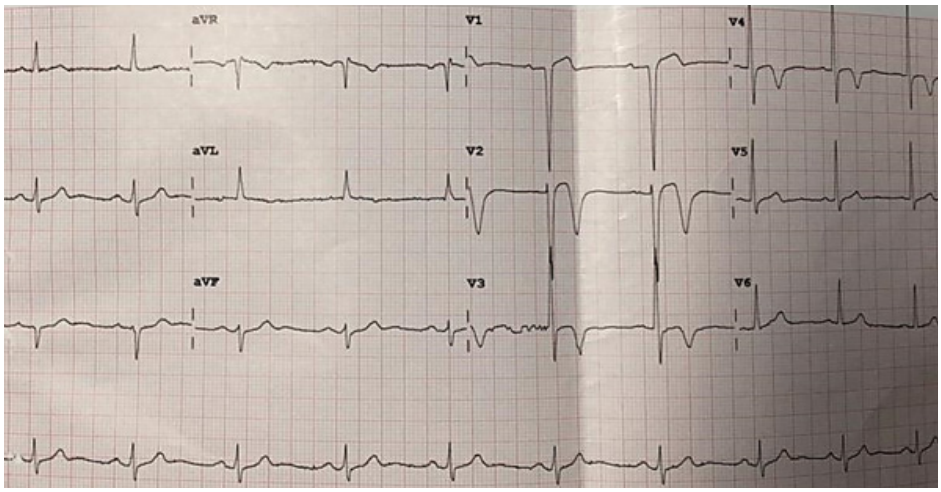

Figure 1. ECG from the patient in NCCHK since 18 hours onset of first chest pain.

Another physical examination was unremarkable with no signs of congestion and hypoperfusion. Electrocardiography showed sinus rhythm, 64 bpm, normoaxis, normal P wave with 0,18s of Interval PR, 0,09s of QRS duration, Deep T inverted V2-V4 and no ST-segment deviations.

The patient was diagnosed as Non-ST Elevation Acute Coronary Syndrome with TIMI (Thrombolysis in Myocardial Infarction) 4/7 GRACE (Global Registry of Acute Coronary Events) 122 CRUSADE (Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes With Early Impelementation of the ACC/AHA guidelines) 22, grade II hypertension (No prior medication). Routine blood test and cardiac enzyme examination were done and showed 62 ng/ml of HsTroponin T, Hb 18 g/dL, Ht 54,2%, leukocyte 19.750/uL, platelet 766.000/uL, ureum 17,40mg/dL, creatinine 0,81 mg/dL, eGFR 105 ml/mnt/1.73m2, GDS 91 mg/dL, Na 140 mmol/L, K 3,7 mmol/L, Cl 103 mmol/L, Ca total 2,16 mmol/L, Mg 2,1 mg/dL. The chest X-Ray showed CTR (Cardio-Thoracic Ratio) 49%, normal aorta and pulmonal segment, apex downward, normal cardiac waist, without any sign of congestion and infiltrate. Bedside echocardiography showed 57% of Ejection Fraction, TAPSE 2.6 cm, with global normokinetic, and valves within normal limit.

Loading dose of double antiplatelet (clopidogrel 600 mg and aspilet 320 mg) were given, and also NTG, Atorvastatin 20 mg, Captopril 3x6,25 mg, and Lovenox 0,6 cc. Upon observation in the ER, the patient still experienced chest pain. Thus, an early revascularization strategy was performed. Coronary angiography showed one vessel disease with 90% stenosis at proximal LAD, with no thrombus. One drug-s stent was implanted with a good result, a TIMI 3 Flow and no residual thrombus.

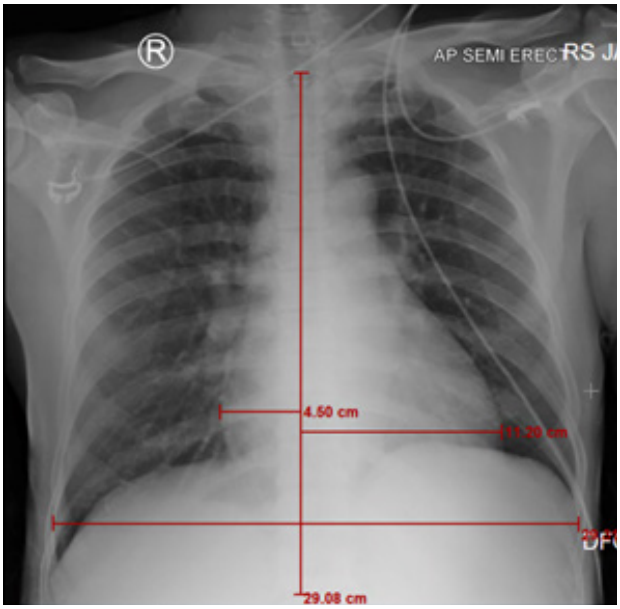

Figure 2. Chest X-ray of patient in NCCHK

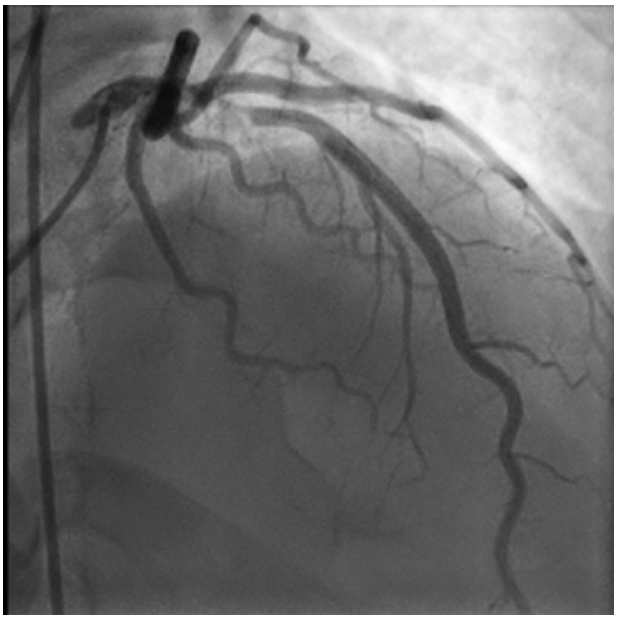

Figure 3. Angiography of the patient

BACKGROUND

Wellens' syndrome is a pattern of T-wave changes seen in the **anterior (V2-V3) leads** on electrocardiography that is highly specific for **critical stenosis of the left anterior descending (LAD)** coronary artery. It is also called as a LAD coronary T-wave syndrome, and was first described in 1982 by a group of cardiologists in the Netherlands (de Zwaan, Bär, Wellens) who were investigating the management of patients admitted to the hospital for unstable angina.[3] Wellens' syndrome is classified into type A or type B, and definitive treatment typically involves cardiac catheterization with percutaneous coronary intervention (PCI) to relieve the occlusion.[4–6]

There are two variations of Wellens' syndrome T-wave. Type A (Type 1) is characterized by biphasic T-waves in V2 and V3. Type B (Type 2) is the more common abnormality is characterized by deeply inverted

T-waves in V2 and V3. The diagnostic leads for T-waves of Wellens' syndrome are V2 and V3, corresponding with a lesion between the first and second septal branches of the LAD. However, if the lesion is more proximal in the LAD, the T-wave changes will be more widely spread along the precordial leads.[4–6]

Wellens' syndrome is signified by deep T wave inversion ≥ 5 mm in lead V2 and V3 (may also be seen in leads V1, V4, V5, and V6), or biphasic T waves (with initial positivity and terminal negativity) in V2 and V3, in the face of Isoelectric or minimally elevated ST segment, less than 1 mm.[7]

Wellens' syndrome results from a temporary obstruction of the proximal LAD coronary artery. Usually, it is caused by the rupture of an atherosclerotic plaque leading to LAD occlusion, with subsequent clot lysis or other disruption of the occlusion before complete myocardial infarction has taken place.[4–6]

As soon as the diagnosis of Wellens' syndrome is suspected, the definitive treatment is urgent cardiac catheterization with PCI, treat Wellens' syndrome similarly to an acute myocardial infarction. All symptomatic patients need intensive care until (ICU) admission and immediate consultation with an interventional cardiologist for cardiac catheterization on a more emergent basis.[8]

DISCUSSION

For this case presentation, we have a patient with intermittent episodes of chest pain. He got a sublingual nitrate every time he felt chest pain before admission to NCCHK hospital. The first and second chest pain always relieved after nitrate was given, but for the third time he felt the next chest pain, he didn't feel right and going to NCCHK hospital. In NCCHK's ER, he had a 4/10 VAS score, or a free chest pain, but we got a typical feature of type B Wellens' syndrome there, which is a deep T inverted in V2-V4. We checked his blood exam and got 52,56 ng/ml of HsTroponin T that indicated a slight increase than normal limit, with normal echocardiography results. Then the CTX shows that in normal limits that contradict with hypertension within the patient. Then he underwent an urgent coroangiography for diagnostic and the therapeutic approach. The result showed a 90% stenosis of Proximal LAD, so he got a drug-eluting stent as the definitive treatment. The coronary circulation there had been restored, a TIMI Flow 3. He was escorted to ICVCU for the next treatment and observation.

Wellens' Syndrome has a similar etiology and risk factors to STEMI, such as hypertension, diabetes mellitus, dyslipidemia, smoking, etc. But in Wellens' Syndrome, it is suspected that a repetitive ischemia-reperfusion theory has a role to its typical T waves ECG pattern, with a auto-clot lysis or disruption of plaque which leads to its pathophysiology.

In the Non-ST Elevation Acute Coronary Syndrome (NSTE-ACS) setting, T-wave inversion without concomitant ST depression is a sign of reperfusion of prior active ischemia, not of ongoing active ischemia itself. This is to be distinguished from the isolated chronic localized T waves that may persist after resolution of an acute event. The extent and magnitude of T-wave inversion may assist in the estimation of the extent and severity of the preceding acute ischemic insult. An example of such a pattern is the Wellens' sign. Deep T-wave inversion in the precordial leads with insignificant ST deviation after the resolution of symptoms is indicative of a tight proximal LAD lesion.

Without aggressive treatment, re-occlusion may occur with transformation to acute STEMI. Thus, this pattern should be considered as a marker of high risk, though the patient does not have active ongoing ischemia. In other cases, T-wave inversion may be less prominent or biphasic T waves with inversion of the terminal portion of the T waves can be seen as a sign of reperfusion. Although there is no indication for emergent reperfusion therapy to prevent ongoing necrosis, this ECG evidence of reperfusion points toward the presence of a culprit coronary lesion that may progress and lead to reischemia/ reinfarction. It is plausible that the degree and depth of T-wave inversion reflect the severity and extent of the previous ischemia; thus, deep negative T waves could be a sign of more extensive preceding transmural ischemia. However, this issue has not been adequately studied.[9]

CONCLUSION

Wellens' Syndrome is a useful typical ECG marker of critical proximal LAD stenosis, it usually could evolve into an extensive anterior infarction if it left unrecognized and/or untreated. Any patient with Wellens' Syndrome mandates urgent angiography and intervention. Any patient with typical ECG findings of Wellens' Syndrome must not undergo any form of cardiac stress test for the further diagnostic test due to the danger of sudden cardiac death.

REFERENCES

1. Balasubramanian K. A dangerous twist of the 'T' wave: A case of Wellens' Syndrome. *Australas Med J*. 2013;6(3):122-125.
2. Asatryan B, Vaisnora L, Manavifar N. Electrocardiographic Diagnosis of Life-Threatening STEMI Equivalents. *JACC Case Reports*. 2019;1(4):666-668.
3. Al-assaf O, Abdulghani M, Musa A, AlJallaf M. Wellen's Syndrome. *Circulation*. 2019;140(22):1851-1852.
4. Singh D, Suliman I, Chyshkevych I, Dabage N. A Pathognomonic Electrocardiogram That Requires Urgent Percutaneous Intervention: A Case of Wellens Syndrome in a Previously Healthy 55-Year-Old Male. *Am J Case Rep*. 2019;20:117-120.
5. Ramanathan S, Soaly E, Cherian A, Heidous MA. 'T' twist: Wellens syndrome. *QJM An Int J Med*. 2019;112(5):373-374.
6. Wang X, Sun J, Feng Z, Gao Y, Sun C, Li G. Two case reports of Wellens' syndrome. *J Int Med Res*. 2018;46(11):4845-4851.
7. Udechukwu N, Shrestha P, Khan MZ, Donato AA. Wellens' syndrome: a close call. *BMJ Case Rep*. June 2018:bcr-2018-225376.
8. Rhinehardt J, Brady WJ, Perron AD, Mattu A. Electrocardiographic manifestations of Wellens' syndrome. *Am J Emerg Med*. 2002;20(7):638-643.
9. de Winter RJ, Verouden NJW, Wellens HJJ, Wilde AAM. A New ECG Sign of Proximal LAD Occlusion. *N Engl J Med*. 2008;359(19):2071-2073.