Early Diagnosis and Prompt Treatment of Severe Stenosis in The Proximal Left Anterior Descending Coronary Artery (Wellens' Syndrome)

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ABSTRACT

The electrocardiography misinterpretation occurs in 20-40% of misdiagnosed myocardial infarction cases. Wellens' syndrome is a typical T-wave finding that shows severe stenosis in the proximal of left anterior descending (LAD) artery, leading to extensive anterior myocardial infarction within days. Cardiac stress tests should not be performed in this condition. Prognosis is fair when identified early and proper coronary intervention was immediately performed.

Keywords: LAD artery, T wave, Wellens' syndrome

ABSTRAK

Kesalahan interpretasi elektrokardiografi terjadi pada 20-40% kasus infark miokard yang salah terdiagnosis. Sindrom Wellens merupakan temuan gelombang-T khas yang menunjukkan stenosis berat di arteri turun anterior kiri (LAD) proksimal, dapat menyebabkan infark miokard anterior luas dalam beberapa hari. Tes stres jantung tidak boleh dilakukan dalam kondisi ini. Kebanyakan pasien dapat membaik jika teridentifikasi dini dan intervensi koroner yang tepat segera dilakukan. **Har Rawishwar Singh Dhilion, Haikal, Vireza Pratama, Agus Harsoyo, Bambang Pamungkas. Diagnosis dan Tatalaksana Dini Stenosis Berat Arteri Koronaria Desendens Proksimal Anterior Kiri (*Sindrom Wellens*)**

Kata kunci: Arteri LAD, gelombang T, sindrom Wellens

INTRODUCTION

Ischemic heart disease is the significant cause of mortality with approximately 7 million deaths and 129 million disability adjusted life years (DALYs) worldwide.[1] The disease is usually due to coronary artery obstruction by thrombus formation as a result from ruptured atherosclerotic plaque. Clinical manifestations and laboratory examinations can be atypical, so it is often unrecognized.[2,3] Electrocardiography (ECG) is a simple and non-invasive examination for coronary artery disease (CAD detection, but misinterpretation occurs in 20-40% misdiagnosed myocardial infarction.[3,4] Isolated ECG findings such as biphasic T wave or T wave inversions in precordial leads may be the only markers of ischemia. These findings were first reported in 1982 by De Zwaan and Wellens. This ECG characteristics indicate severe stenosis (>90%) in the proximal LAD artery that can progress into extensive anterior myocardial infarction within days to weeks if not detected and treated promptly. Early diagnosis of this syndrome is very crucial.[3,5,6]

We present the case of a patient with previous chest pain, the ECG showed Wellens' syndrome that was initially overlooked and consequently progressed to acute coronary syndromes.

CASE

A 58-year-old man presents to emergency department complaining crushing chest pain lasting for about six hours accompanied with diaphoresis. Patients denied the presence of nausea, vomiting, shortness of breath, palpitation, and syncope. Similar pain episodes occurred at rest over the prior month, each episode lasted for about 30 minutes. He was usually given injections for stomach problems at every visit. No history of diabetes, hypertension or cardiac problem. No family history of coronary artery disease. He is obese (body mass index 25.7 kg/m^2) and current cigarette smoker. On physical examination, the patient appeared in moderate pain (VAS 4/10). He was alert, oriented, and coherent. Vital signs were temperature 36.5^{o}C, pulse 65 times per minute, blood pressure 110/60 mmHg, and respiration rate 20 times per minute with 98% oxygen saturation in room air. No pale conjunctivae, icteric sclerae nor cyanotic lip. Neck examination showed no increased jugular venous pressure. On auscultation, no ronchi or wheezing found. Cardiac examination shows no cardiomegaly, normal cardiac sound without extra heart sounds or murmurs. No abdominal pain, no palpable liver and spleen. The extremities were warm, capillary refill time is 2 seconds, no edema nor bluish colorization in both

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legs. The electrocardiography shows sinus rhythm with QRS rate 62 times per minute and normal axis. There is T-wave inversion in I, aVL and V2 leads. There is also deep T-wave inversion in V3-V6 leads. Chest X ray showed no radiological abnormality in the heart and lungs.

Laboratory examination showed hemoglobin level 12.2 g/dL, leukocyte count 6.310/mL, platelet count 252.000/mL. BUN 46 mg/dL, creatinine 1.1 mg/dL, GOT 13 U/l, GPT 19 U/l, sodium 137 mEq/L, potassium 3.6 mEq/L, chloride 107.0 mEq/L and random glucose 89 mg/dL. Total cholesterol 197 mg/dL, LDL 126 mg/dL, HDL 53 mg/dL, and triglyceride 164 mg/dL. Cardiac enzyme CK was 54 IU/l, CK-MB 15 IU/l, and troponin I < 0.01 ng/mL. Based on the symptoms and electrocardiogram, the diagnosis was unstable angina pectoris TIMI score 2, with dyslipidemia and Wellens' syndrome. We consider patient's ECG showed a Wellens' syndrome that was initially overlooked and consequently progressed to acute coronary syndromes.

Patient was advised to bed rest with semi Fowler position. Nasal oxygen cannula 3 litre/minute was given. Treatments were acetylsalicylic acid 80 mg od., clopidogrel 75 mg od., bisoprolol 5 mg od., nitroglycerin 2.5 mcg/minute, and atorvastatin 20 mg od. Patients were also given an 1800 calorie/24 hours heart diet type II and total fluid 1800 mL/24 hours. Patient was transferred to intensive care unit (ICU) for heparinisation. In the second day of hospitalization the patient had refractory chest pain, early percutaneous coronary angiography was performed. The result was 99% subtotal occlusion in proximal-mid left anterior descending artery with TIMI I-II flow. Percutaneus transluminal coronary angioplasty (PTCA) was then performed with drug-eluting stent (DES) implantation. After revascularization, patient's hemodynamics are stable and his chest pain is reduced.

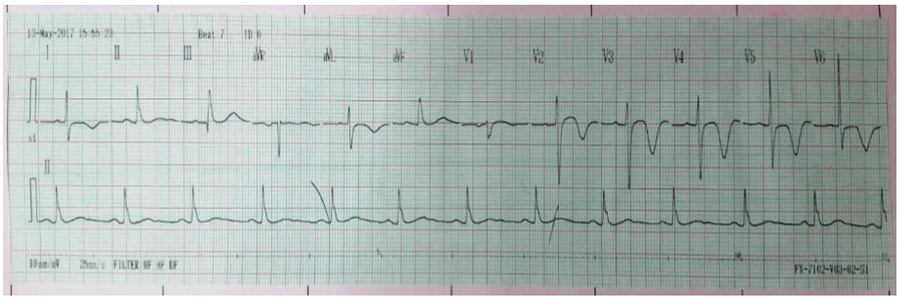
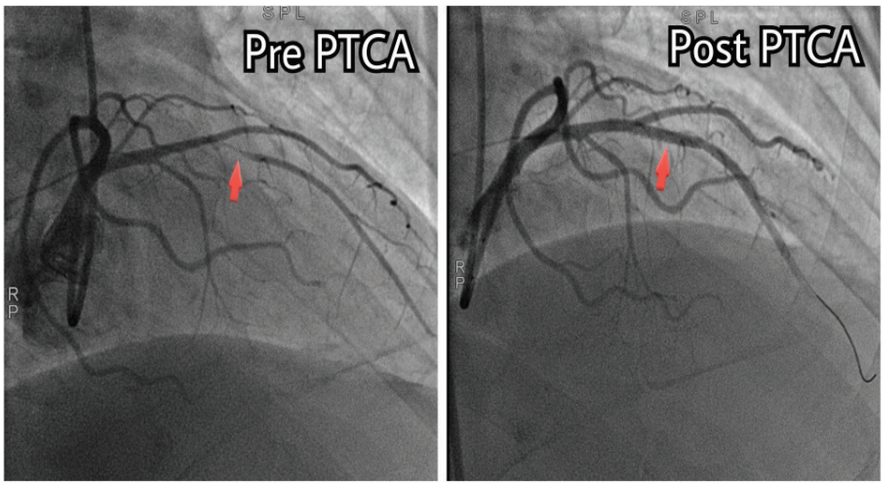


Figure 1. The first electrocardiogram on admission in emergency room: T-wave inversion in I, aVL and V2 lead and also deep T-wave inversion in V3-V6 leads.



A B

Figure 2. Coronary angiography showed 99% stenosis in proximal left anterior descending artery (A); percutaneus transluminal coronary angioplasty (PTCA) was performed with a drug-eluting stent implantation (B)

On the third day of hospitalization, chest pain was absent but the ECG still showed T wave inversion in I,aVL, V2-V6 leads. Laboratory examination showed normal cardiac enzyme and normal lipid profile. Patient was discharged 2 days later and was prescribed acetylsalicylic acid 80 mg od and clopidogrel 75 mg od for 12 months, bisoprolol 1.25 mg od. with atorvastatin 20 mg od. He was also counseled against tobacco use and to achieve normal body weight through proper exercise and healthy diet. He was still asymptomatic at 2-week follow-up.

DISCUSSION

The incidence of Wellens' syndrome is about 10-15% in any age group.[2,4-6,10] No stress test can be performed because it can increase the oxygen demand in the myocardium causing myocardial infarction and death.[2,5,6,10]

Wellens' syndrome consists of type A (25%) with typical biphasic T waves on leads V2 and V3, and type B (75%) with negative T waves in leads V2 and V3.[2,5,6,10] T wave deviations may appear in other precordial leads depending on the proximity of the lesion. It usually occurs during pain-free intervals in intermittent angina pectoris.[2,5,6,10] The T wave inversion has a depth of 1-5 mm, whereas deep T wave inversion has a depth of 5-10 mm.7 T-wave inversion on V1-V3 precordial leads is common in children and adolescents rather than adults, whose prevalence is only 0.5% in the 30-59-year-old population. Especially in trained athletes, inverted T waves in right precordial leads is considered as a benign ECG phenomenon. Negative T waves caused by ischemia are symmetrical and deep.[7,8] Other causes of T wave inversion include myocardial infarction, left ventricular hypertrophy, acute central nervous system disorders, and digitalis effect.[5,9] Biphasic T wave is a wave with two components starting with a positive wave then pass below the isoelectric line.[5,9] Cardiac enzymes are usually slightly elevated or within normal limits.[2,5,6,10]

Figure 3. Types of Wellens' syndrome[11]

Table. Diagnostic criteria for Wellens' syndrome[2,5,6,10]

Symmetrical deeply inverted or biphasic T-waves in V2 and V3, possibly V1,V4,V5 and/or V6 when pain free
Isoelectric ST segment or mild elevation (1 mm)
No pathological Q-waves in precordial leads or loss of R-waves progression
History of angina
Normal or mildly elevated cardiac enzymes

The pathophysiology of Wellens syndrome has been suggested from abrupt reperfusion after the occlusion of LAD artery; the reperfusion may be unstable and this vessel may re-occlude, causing further angina and ischemia. Ischemia in the epicardium may lead to delayed ventricular repolarization. When the endocardium first repolarizes rather than the epicardium, the vector of the electric direction is reversed and causes a negative T wave. This cycle of occlusion and reperfusion will continue until the coronary blood supply can no longer be reestablished and an acute myocardial infarction occurs.[6,10,13] All patients with this syndrome should receive standard ACS therapy which consists of antiischemic therapy to balance myocard oxygen demand and supply, as well as antithrombotic therapy to prevent further growth of coronary thrombus. Left anterior descending coronary artery supplies the left ventricular mass by as much as 70%, thus contributing to the poor prognosis of individuals who was managed late. Most patients, when identified early and taken for proper cardiac catheterization, T-waves was resolved after appropriate revascularization.[2,5,6,10]

CONCLUSION

Wellens' syndrome is an ischemic ECG findings which consist of biphasic T wave or deep T wave inversions in precordial leads. Cardiac stress tests should not be performed in this condition. Early identification and immediate proper coronary intervention can greatly improved prognosis. Awareness of Wellens' syndrome is essential to prevent life-threatening acute coronary syndrome.

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