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A Pathognomonic Electrocardiogram That Requires Urgent Percutaneous Intervention: A Case of Wellens Syndrome in a Previously Healthy 55-Year-Old Male

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Patient: Male, 55
Final Diagnosis: Wellens' syndrome
Symptoms: Chest pain
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare disease

Background: Acute chest pain is a common presentation in emergency departments worldwide. Ruling out acute coronary syndrome is essential in ensuring patient safety. Workup includes electrocardiogram (ECG) and cardiac biomarkers. Wellens syndrome is characterized by a history of chest pain, normal or minimally elevated biomarkers, no STEMI/Q-waves, and specific ECG changes. These changes consist of biphasic T waves in lead V2 and V3 or deep symmetrically inverted T waves in leads V1–V4.

Case Report: A 55-year-old male presented to the emergency department with acute chest pain in a background of active smoking, hypertension, and hyperlipidemia. His ECG was characteristic of Wellens syndrome type 1 and negative cardiac biomarkers. His TIMI (thrombolysis in myocardial infarction) score was 2, however, he failed conservative management necessitating urgent coronary angiogram. Critical stenosis of the proximal left anterior descending (LAD) coronary artery was found which required 2 drug eluting stents. He was discharged home asymptomatic on optimal medical therapy.

Conclusions: Conventional management of patients with NSTEMI (non-ST-elevation myocardial infarction) and unstable angina with risk stratification utilizing TIMI score may not be appropriate in patient with Wellens syndrome. This highlights the importance of ECG recognition and urgent percutaneous intervention in patients with Wellens syndrome. Failure to identify this clinical syndrome could result in significant morbidity and mortality because it relates to critical stenosis and imminent large myocardial infarction.

MeSH Keywords: Acute Coronary Syndrome • Electrocardiography • Percutaneous Coronary Intervention

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Background

Acute chest pain accounts for 10% of all emergency department visits. Acute chest pain raises the concern of possible acute coronary syndrome (ACS). After thorough workup only 10–15% of patients have the diagnosis of ACS. Diagnosis of ACS can be missed in 2% of patients [1]. It is important for all physicians to be aware of subtle presentation of ACS such as Wellens syndrome. When it was first described Wellens syndrome was thought to represent 14–18% of cases of unstable angina [2]. Wellens syndrome consists of 2 types of characteristic electrocardiographic (ECG) findings. Type 1 accounts for 24% and consists of biphasic T waves most commonly in lead V2 and V3, but can involve leads V1–V5/V6. Type 2 is more common and is identified by deep, symmetrically inverted T waves in leads V1–V4 [3,4]. This is accompanied by a history of anginal chest pain, normal or minimally elevated cardiac biomarkers, absence of Q-waves/STEMI (ST-elevation myocardial infarction) and normal R wave progression. Wellens syndrome is a pre-infarction stage of coronary artery disease because it is associated with critical proximal left anterior descending (LAD) coronary artery stenosis [5]. Early recognition is important because it can lead to a massive anterior wall myocardial infarction.

Case Report

A previously asymptomatic 55-year-old male presented with acute chest pain in a background significant for active smoking, hypertension, and hyperlipidemia. There was no known coronary artery disease or relevant surgical, family, or allergy history. His medications included atorvastatin 10 mg and lisinopril 10 mg daily.

He awoke with sudden onset of crushing central chest pain with 8 out of 10 severity. The pain initially lasted 10 minutes but then became intermittent in nature. He denied any aggravating or relieving factors but did endorse associated diaphoresis. He specifically denied any palpitations, shortness

of breath, nausea, or any other symptoms. After about half an hour he decided to present to the emergency department.

He arrived at the emergency department and an ECG was performed in triage while he was asymptomatic and was significant for biphasic T waves in V2–V4 (Figure 1). Out of concern for the possibility of Wellens syndrome versus typical unstable angina he was treated with loading doses of dual antiplatelets (DAP) aspirin 325 mg and clopidogrel 300 mg, heparin (LMWH) 1 mg/kg, sublingual nitroglycerin tablet 0.6 mg, and oral metoprolol 25 mg twice daily. We continued his lisinopril and increased the dose of atorvastatin (80 mg/day) as he had clinical atherosclerotic cardiovascular disease. Chest x-ray, complete blood count, d-dimer, and comprehensive metabolic panel were all unremarkable. Troponin I at 1, 4, and 7 hours after initial acute chest pain were all normal with no upward trend. Cholesterol (low-density lipoproteins. 96 mg/dL) and glycosylated hemoglobin (5.5%) were unremarkable.

Over the first day of admission, the cardiology service was consulted and recommended medical management because his thrombolysis in myocardial infarction (TIMI) score was 2. Echocardiogram revealed a mildly depressed ejection fraction at 45% with no regional wall abnormalities. He was, however, still experiencing chest pain that was responsive to administration of sublingual nitroglycerin tablet. The ECG remained unchanged and his cardiac biomarkers negative.

Over the next 2 days, in spite of optimizing medical therapy, the patient remained symptomatic with central chest pain at rest. On day 3, in light of ongoing symptoms, the decision was made to undergo coronary angiogram (CAG). A high grade 90% LAD artery lesion proximal to the first septal and 70% stenosis of the mid LAD artery were noted (Figures 2, 3). Percutaneous coronary intervention (PCI) was undertaken with 2 drug eluting stents (DES) placed across the proximal lesion and one DES to the mid LAD artery with good angiographic result (Figure 4). After intervention, there was symptomatic improvement with no further episodes of chest pain. He was discharged home to establish care with a cardiologist and to continue aspirin 75 mg

Figure 1. Electrocardiogram on presentation consistent with Wellens syndrome type 1.

Figure 2. Diagnostic coronary angiogram showing critical stenosis of the proximal left anterior descending artery (yellow arrow) and moderate stenosis of the mid left anterior descending artery (red arrow).

Figure 3. Second view diagnostic coronary angiogram showing critical stenosis of the proximal left anterior descending artery (yellow arrow) and moderate stenosis of the mid left anterior descending artery (red arrow).

daily, clopidogrel 75 mg daily, metoprolol 75 mg twice daily, atorvastatin 80 mg daily, and lisinopril 10 mg daily.

Figure 4. Angiogram post placement of 2 drug eluting stents.

Discussion

In evaluating chest pain, the history, physical examination, ECG, and cardiac biomarkers are essential to identify patients with ACS. Patients without typical chest pain, positive biomarkers, or ECG evidence of ischemia are considered low risk for ACS. These patients are referred for outpatient stress test within 72 hours of discharge. Identification of ECG changes in Wellens syndrome is important because patients may be asymptomatic, and the majority of these patients have negative cardiac biomarkers (88%) [3]. Cardiac stress testing is contraindicated as it may precipitate myocardial infarction.

The Wellens syndrome pattern ECG changes are thought to be highly characteristic for proximal LAD artery stenosis. Critical stenosis of the proximal LAD artery results in a cycle of occlusion and reperfusion. This is thought to be the etiology of biphasic T waves in the anterior leads [6]. It is important to identify this characteristic ECG in low-risk patients as this would re-stratify these individuals to a high-risk group and avoid unnecessary delays in CAG and percutaneous coronary intervention.

Re-stratifying low-risk patients in to high risk by identifying the Wellens syndrome pattern on ECG is presented in the literature. One such case involved a 24-year-old female who was deemed to be at low risk for ACS and the failure to recognize a Wellens syndrome pattern ECG resulted in her being discharged from the emergency department. She unfortunately

represented with NSTEMI (non-ST-elevation myocardial infarction) one week later [7].

Wellens syndrome requires urgent percutaneous coronary intervention rather than medical management alone. Patients with Wellens syndrome who do not undergo percutaneous coronary intervention can go on to develop extensive anterior wall myocardial infarction within 8.5 days [3]. Echocardiography should be obtained because it can provide risk stratification [8]. Failure to identify this clinical syndrome could result in significant morbidity and mortality because it relates to critical stenosis and imminent large myocardial infarction.

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Conclusions

Acute coronary syndrome must be ruled out in a patient presenting with chest pain. Cardiac biomarkers and TIMI score are helpful in risk stratifications. However, sometimes a patient may have negative biomarkers and low TIMI score and yet require urgent percutaneous coronary intervention. Therefore, recognition of Wellens syndrome is important because prompt intervention can prevent a massive acute myocardial infarction. We hope all physician are aware of these ECG findings, and hope to help advance clinical practice with this case report.

Conflicts of interest

None.