Clinical Image

An incidental case of Wellens' syndrome in a community emergency department

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INTRODUCTION

Wellens' syndrome, also called left anterior descending (LAD) coronary T-wave syndrome, is a characteristic electrocardiographic (ECG) pattern suggestive of coronary artery disease in the preinfarction stage.^[1–3] ECG changes in Wellens' syndrome may signify critical stenosis in the proximal LAD coronary artery.^[1–5] Furthermore, ECG changes may occur during a pain-free period with no other evidence of ischemia or unstable angina and none to minimal elevation of cardiac enzyme levels.^[3,5] Hence, recognition of the aforementioned pattern is important in preventing the development of a devastating anterior wall myocardial infarction.^[2–5]

There are two patterns of ECG changes related to Wellens' syndrome. The first involves a minimally elevated or isoelectric ST segment with inverted T waves in leads V2 and V3, often in leads V1 and V4, and occasionally in leads V5 and V6.^[1–3,5] This pattern accounts for approximately 75% of Wellens' syndrome cases.^[3,5] The second pattern involves biphasic T waves in leads V2 and V3^[1–3,5] and accounts for roughly 25% of Wellens' syndrome cases.^[3,5]

CASE

The patient, a 59-year-old female, was accompanying her husband on his visit to the emergency department. She had complained to the staff nurse taking care of her husband about her left arm discomfort that developed overnight and resolved spontaneously by the following morning. With the assistance from the staff nurse, she registered at the department's triage station and underwent further assessment. On further questioning, she stated her discomfort did not radiate to the chest

and she also experienced right jaw pain secondary to a "wisdom tooth extraction one week ago". She also complained of shortness of breath (SOB) on moderate exertion but indicated that the severity of her respiratory symptoms had not gotten worse recently.

She had familial hypercholesterolemia, hypertension (HTN), bilateral knee osteoarthritis, gastroesophageal reflux disease, migraine, anxiety and depression. Her father died at age of 48 from a myocardial infarction (MI), and her mother died at age of 86 and did not have any cardiovascular risk factors. She was treated with a series of antihypertensives but did not use statin for years because she had myalgia in the past.

On physical examination, her vital signs were stable. No adventitious respiratory sounds were heard. She had normal S1 and S2 heart sounds, but neither S3 nor S4 was present. No elevation in jugular venous pressure (JVP) was noted, and she had bilateral pitting edema to mid shin.

Investigations and laboratory results

Complete blood count, electrolytes, urea, creatinine and random glucose were all within normal limits. On admission to the emergency department, creatinine kinase was 29 units/L (normal=30 to 200) and high sensitivity troponin T was 15 ng/L (normal≤14). Three hours after admission, creatinine kinase level was 22 U/L, and troponin T level was 16 ng/L. Approximately 6 hours after admission, creatinine kinase level was 19 U/L, and troponin T level was 15 ng/L. In short, the patient had slightly elevated serial troponin T levels.

Posterior-anterior and lateral chest radiographs were not marked as there was no evidence of pleural effusion, pulmonary edema or acute intrathoracic pathology.

The nursing staff at triage conducted an ECG and notified the emergency physician of new ECG changes compared to one done approximately 18 months ago. T wave inversion in anterolateral leads was noted on two ECGs that were conducted approximately 90 minutes apart (Figures 1 and 2).

Subsequently, the patient was admitted to the department of internal medicine. After the pain was relieved overnight, an exercise stress test was performed at the department of internal medicine one day after she was admitted to the emergency department. She was able to attain 7.4 METs and the test was limited by left sided chest pain and left arm pain with less than 2 mm ST depression.

Treatment and follow-up

The patient was given acetylsalicylic acid (ASA), clopidogrel, fondaparinux and statin therapy. She also underwent cardiac catheterization (Figure 3) four days after admission, which demonstrated 90% proximal

stenosis of the LAD branch of the left coronary artery and 80% attenuation in the left circumflex branch. Both critical LAD and circumflex lesions were intervened using drug eluting stents and no residual narrowing of stent was noted.

Before being discharged, echocardiogram of the patient showed normal left ventricular systolic function. Her low density lipoprotein level was 5.49 and high density lipoprotein level was 1.0. Her lipid level was determined with titration of statin. She was continuously subjected to ASA, clopidogrel, angiotensin converting enzyme inhibitor and statin therapy in addition to regular anti-HTN medication.

On follow-up, about one month after admission to the emergency department, the patient was asymptomatic and denied chest pain or worsening of SOB. Vital signs were within normal limits and JVP was not elevated. ECG after a 6-month follow-up showed resolution of her initial ECG changes (Figure 4).

Figure 1. Initial ECG with inverted T waves in anterolateral leads especially precordial leads V2 and V3.

Figure 2. Second ECG 90 minutes later with inverted T waves in anterolateral leads especially precordial leads V2 and V3.

DISCUSSION

Since the patient was asymptomatic on admission and had almost normal biomarkers, Wellens' syndrome could be diagnosed on the basis of ECG changes. ECG changes with inverted T waves can occur in a variety of contexts other than Wellens' syndrome including previous myocardial infarction, persistent juvenile T wave pattern, acute coronary ischemia (non-Wellenoid ischemia and non-ST segment elevation acute myocardial infarction), bundle branch block, left ventricular hypertrophy, acute myocarditis, pre-excitation syndromes, acute pulmonary embolism, later stages of pericarditis, cerebrovascular accident, and digitalis effect.^[3] Consequently, the criteria listed below can be used to differentiate Wellens' syndrome from other causes of inverted T wave changes on ECG:^[3,5] prior history of chest pain; normal or slightly elevated cardiac enzymes; absence of precordial Q waves; patterns in a pain free state; isoelectric or minimally elevated ST segment; biphasic T waves in leads V2 and V3 or deeply

inverted T waves in leads V2 and V3.

Although the presented patient did not experience typical chest pain, she did complain of left arm discomfort suggestive of an atypical cardiac manifestation.^[6-7] Moreover, her serial troponin T levels were slightly elevated and ECG changes were noted while she was pain free. ECG changes included inverted T waves in V2 and V3. No precordial Q waves were noted and ST segment was isoelectric. In turn, the patient met the discussed criteria.

Current guidelines lack specific recommendations for the management of Wellens' syndrome. In our patient, the consulting service employed the conservative strategy for revascularization. Conservatively, patients with unstable angina or non-ST segment elevation MI (NSTEMI) should receive pharmacological therapy and then undergo revascularization if they have recurrent symptoms of inducible ischemia upon non-invasive testing.^[8] Alternatively, a routine invasive strategy is suitable for all patients with unstable angina or NSTEMI who undergo coronary angiography followed by revascularization based on angiography results.^[8] Benefits of the routine invasive strategy include a significant reduction in mortality and MI from hospital discharge to the end of follow-up.^[8] However, the routine invasive strategy is associated with a higher early mortality.^[8]

Our patient underwent an exercise stress test, such a procedure should not be performed in patients with Wellens' syndrome as increased cardiac demand along with a critically stenosed LAD branch can trigger a MI.^[5,9] Thus, we recommend the routine invasive strategy for patients with Wellens' syndrome. It is very important for health care providers to identify patients with ECG findings that are characteristic of Wellens'

Figure 3. Coronary angiogram showing lesions (arrows) in the LAD and left circumflex branches of the left coronary artery.

Figure 4. ECG at a 6-month follow-up showing resolution of initial ECG changes.

syndrome. Furthermore, as in our case, ECG changes can normalize completely in 90% of cases after appropriate intervention.^[1]

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