A 75-Year-Old Woman with COVID-19 Pneumonia and Wellens Syndrome Diagnosed by Electrocardiography

George S. Prousi, Jacob Giordano, Patrick J. McCann

Corresponding Author: George S. Prousi, e-mail: gprousi@gmail.com
Conflict of interest: None declared

Patient: Female, 75-year-old
Final Diagnosis: Wellens’ syndrome
Symptoms: None
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare disease

Background: There are increasing reports of cardiovascular complications associated with coronavirus disease 2019 (COVID-19) due to infection with severe acute respiratory syndrome coronavirus 2. Wellens syndrome, or left anterior descending T-wave syndrome, is diagnosed by a pattern of electrocardiographic (ECG) changes that include inverted or biphasic T waves in leads V2-V3.

Case Report: A 75-year-old woman presented to the emergency department with a 1-week history of fatigue and progressive shortness of breath who acutely decompensated, necessitating mechanical ventilator support. Initial lab workup revealed COVID-19 positivity, which was confirmed by repeat testing. A routine ECG obtained during her hospitalization and compared with her baseline revealed diffuse T-wave inversions of her precordial leads, which was highly suggestive of Wellens syndrome. Cardiac enzymes obtained were slightly elevated and an echocardiogram did not demonstrate wall motion abnormalities. The patient was initiated on non-ST segment elevation myocardial infarction protocol with heparin infusion for 48 hours and dual antiplatelet therapy, in addition to beta blockade. Repeat ECGs showed complete resolution of Wellens syndrome shortly after therapy.

Conclusions: Although rare, Wellens syndrome is a significant indicator of left anterior descending artery stenosis and is commonly associated with acute medical illness. COVID-19 pneumonia has been associated with many adverse cardiovascular outcomes, with ischemia and arrhythmia becoming increasingly more common. Diagnosis of Wellens often includes coronary angiography; however, during the current pandemic, many authorities have recommended medical management alone during the acute phase of care, depending on the severity of concomitant illness.

Keywords: Acute Coronary Syndrome • Coronary Angiography • Coronary Stenosis • COVID-19
Background

Wellens syndrome is a pattern of electrocardiogram (ECG) changes including deeply inverted or biphasic T waves and is highly suggestive of left anterior descending artery (LAD) stenosis [1]. Patients exhibiting these ECG changes present in many ways; they are often asymptomatic and with little evidence of ischemia as demonstrated by minimal cardiac enzyme elevation [2]. Because of the concern of LAD involvement and the possibility of an anterior wall myocardial infarction, invasive evaluation is recommended [3]. Despite this suggestion, current limitations exist during the coronavirus disease 2019 (COVID-19) pandemic, challenging providers with the decision to pursue conservative strategies in hemodynamically stable patients with COVID-19 [4].

Case Report

A 75-year-old woman presented to the emergency department with a 1-week history of fatigue, body aches, and intermittent diarrhea, with subsequent development of shortness of breath the day of hospital evaluation. On presentation, she was afebrile, hemodynamically stable, but hypoxic to 80% oxygen saturation on room air. Laboratory investigation was significant for an elevated procalcitonin of 0.43 ng/mL, creatinine of 1.3 (baseline 0.7-0.9), and a positive COVID-19 test (Diasonin Molecular LLC, Cypress, CA, USA) with positive repeat test for confirmation. Imaging including chest X-ray and chest computed tomography (CT) revealed streaky opacities and dense bilateral airspace consolidation, respectively. An ECG on presentation demonstrated sinus rhythm with nonspecific T-wave flattening most prevalent in the inferior and lateral precordial leads (Figure 1). Because of the progression of hypoxia and respiratory distress, the patient was intubated and admitted to the intensive care unit for further management of COVID-19. Treatment strategies included the emergency use of Remdesivir, convalescent plasma, and dexamethasone. On the fifth day of her hospitalization, 4 cardiologists were consulted because of concern for ECG changes. Routine ECG revealed diffuse symmetric T-wave inversions precordially with biphasic T waves noted in V1 and V2. There was associated minimal ST depression inferiorly in leads II, III, and aVF in addition to incomplete R wave progression consistent with combined types A and B Wellens pattern (Figure 2). Cardiac enzyme trends showed only a slight rise of troponin I to 0.16, 0.11, and 0.10 ng/mL. An echocardiogram was obtained to further evaluate left ventricular function and wall motion, which did not reveal any abnormality. A thorough review of the patient’s history was conducted and, although never formally evaluated via stress imaging or catheterization, the patient did not have a diagnosis of coronary artery disease. Given the patient’s current infected state and general cardiovascular stability, the decision was made to pursue medical management. She was initiated on a high-intensity statin, loaded with aspirin and P2Y12 inhibitors, and maintained on daily dose regimens. Additionally, therapeutic heparin was provided for 48 hours and beta blockade was recommended. At the time of cardiovascular evaluation, she remained hemodynamically stable and no inciting event aside from her current infected state was identified. Repeat ECGs after non-ST segment elevation myocardial infarction (NSTEMI) therapy demonstrated resolution of the deep T-wave inversions and ST depressions (Figure 3).

Discussion

The establishment of Wellens syndrome includes several criteria, namely deeply inverted T waves or biphasic T waves in

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Figure 1. Presenting electrocardiogram demonstrating sinus rhythm with nonspecific T-wave flattening most prevalent in the inferior and lateral precordial leads.
leads V2 and V3 in addition to absence of Q waves and only minimally elevated cardiac enzymes [1], which were demonstrated in our case. Historically speaking, the decision to proceed to coronary angiography for further evaluation of critical LAD [1] stenosis is case-dependent but oftentimes conducted.

For the establishment of the diagnosis of COVID-19, neither the absence nor the presence of clinical signs or symptoms is accurate enough to rule in or out the disease [5]. Therefore, laboratory testing should be utilized in the diagnosis of COVID-19. There are multiple methods for testing, but currently the preferred method of detecting severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection is with nucleic acid amplification testing (NAAT), usually involving using a reverse-transcriptase polymerase chain reaction (RT-PCR) on a sample acquired from the patient’s upper respiratory tract [6]. A positive NAAT typically confirms the diagnosis of COVID-19; no additional diagnostic testing is necessary. For patients with a negative test, false negatives are known to occur. In these circumstances, if a high-enough clinical suspicion exists, repeat NAAT testing may occur 24-48 hours after the initial test [6]. Given the current circumstances related to the COVID-19 pandemic, patients admitted with acute hypoxic respiratory failure have been shown to develop systemic manifestations such as deep vein thrombosis and pulmonary embolism in addition to myocarditis and even myocardial infarction [7]. Although guidelines continue to recommend optimal therapy for these occurrences, many societies have added the treatment strategy of conservative therapy, if appropriate, for stable NSTEMI [4].

A recent case report in the Egyptian Heart Journal noted a connection between a suspected case of COVID-19 and Wellens syndrome. After having come into contact with COVID-19-positive...
relatives, the patient sought medical treatment after experiencing typical squeezing chest pain for 4 hours before presentation, with fever, dyspnea, sore throat, and fatigue for 3 days before presentation. A chest X-ray showed preceding consolidation along with new consolidative changes in the left apical middle lower zone and the right lower peripheral region. A chest CT scan was subsequently performed and suggested diffuse pneumonia in both lungs with multifocal ground-glass opacities and crazy paving pattern, which can be a common finding in patients with COVID-19. However, the patient tested negative on a SARS-CoV-2 rapid immunochromatographic test (Wondfo Biotech, Guangzhou, China) and then subsequently tested negative again with repeat testing, this time using a RT-PCR assay (Abbott RealTime SARS-CoV-2 assay, Abbott Molecular, Inc., Chicago, IL, USA) from a nasopharyngeal swab. Their presumed diagnosis of COVID-19 was made on the basis of symptoms and radiographic evidence alone, without laboratory confirmation. This patient also did not undergo coronary angiography, as the presenting symptoms had resolved and the patient insisted on discharge with close outpatient follow-up for elective coronary angiography. In our patient, the diagnosis of COVID-19 was established via NAAT (Diasorin Molecular, LLC), and the concurrent diagnosis of Wellens syndrome was made given the characteristic ECG findings, the minimally elevated troponin, and the absence of chest pain. Coronary angiography was not attempted in this patient because of the increased risk for exposure of cardiac catheterization laboratory personnel, as well as the relative asymptomatic nature of the patient [8]. The patient was treated as an NSTEMI with heparin and was eventually discharged after resolution of the acute hypoxic respiratory failure secondary to COVID-19.

Wellens syndrome, although highly suggestive of underlying critical stenosis, remains a trivial pathology but can be reasonably approached from an NSTEMI perspective. In conclusion, COVID-19 manifests itself in myriad ways and at times is associated with myocardial ischemia. Wellens syndrome is highly suggestive of LAD stenosis and oftentimes prompts angiographic evaluation, but in certain circumstances it is reasonable to pursue medical management of ischemic heart disease.

Conclusions

Although rare, Wellens syndrome is a significant indicator of LAD stenosis and is commonly associated with acute illness. The current recommendation for evaluation commonly consists of coronary evaluation to determine the degree of LAD stenosis and the potential need for stenting or revascularization. During the current pandemic, however, authorities such as the European Society of Cardiology and the American College of Cardiology have recommended the addition of medical management during the acute phase of concomitant COVID-19 pneumonia. The workup at present includes structural evaluation with echocardiography in addition to the evaluation of potential coronary artery disease risk factors such as hyperlipidemia. Therapies, as for most NSTEMIs, consist of high-intensity statin, dual antiplatelet aspirin, and P2Y12 inhibitors, in addition to heparin infusion for 48 hours. If hemodynamically stable, the consideration of beta blockade is also suggested. This report describes a rare cardiovascular association with SARS-CoV-2 infection. At this time, the diagnosis of Wellens syndrome may be challenging because of the historical suggestion for the use of coronary angiography to support the ECG findings.

Conflicts of Interest

None.

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