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Wellen's Syndrome: An Infamous Example of Occlusive Myocardial Infarction (OMI)

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Wellen's Syndrome: an infamous example of occlusive myocardial infarction (OMI)

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Introduction:

Wellen's syndrome is a condition with characteristic ECG changes that can be highly specific for acute myocardial infarction involving the left anterior descending (LAD) artery. Acute myocardial infarctions are one of the leading causes of death in the developed world. Its estimated annual prevalence is nearly 3 million worldwide, with more than 1 million in the United States [1]. The importance of timely diagnosis cannot be understated. Here we describe a case of myocardial infarction secondary to LAD occlusion which presented with the atypical ECG pattern known as Wellen's syndrome.

Case Presentation:

A 58-year-old male presented to the Emergency Department with complaint of diffuse anterior chest pain and upper back pain described as burning in quality, pleuritic and worse with exertion over the past 2-3 weeks. The patient initially suspected the pain was due to gastric reflux, but was unimproved with antacids. He denied cough, lightheadedness, syncope, leg swelling, diaphoresis, abdominal pain, fever or chills.

On presentation, his vital signs were blood pressure 156/106 mmHg, heart rate of 91 beats per minute, temperature of 97.7 degrees Fahrenheit orally, with respiratory rate of 22 breaths per minute and pulse oximetry of 99% on room air. His body mass index (BMI) was 30.65 kg/m^2.

Physical examination revealed a normal-appearing male in no acute distress with no diaphoresis. Mucous membranes were moist with clear oropharynx. Extraocular movements were intact and pupils 3 mm and equal, round and reactive to light. On auscultation heart was regular rate and rhythm without murmur. Lungs were clear to auscultation bilaterally with normal respiratory effort. Abdomen was soft and non-tender, without rebound rigidity or guarding. Musculoskeletal exam revealed diffuse upper thoracic paraspinal muscular tenderness. There was no crepitus, swelling, ecchymosis or deformities noted, no midline tenderness and no step-off. There was no anterior chest wall tenderness to palpation. Neurologic exam revealed no focal neurologic deficits.

An initial electrocardiogram (ECG) revealed normal sinus rhythm at 89 beats per minute with normal axis, LVH, and 1 mm ST depressions in inferolateral leads. Lab work completed revealed an initial high-sensitivity troponin level of 789 ng/L. The brain natriuretic peptide (BNP) was 16 pg/mL. A complete blood count was performed without evidence of leukocytosis or anemia. The basic metabolic panel revealed a creatinine level of 1.29 mg/dL, glucose of 137 mg/dL, and no significant electrolyte derangements. After noting the initial elevated troponin result, a second ECG was ordered revealing similar findings as the first: normal sinus rhythm at 87 beats per minute, normal axis, with LVH and ST depressions in the inferolateral leads. A chest x-ray was performed which revealed no cardiomegaly, pneumothorax pleural effusions or infiltrate. A repeat high-sensitivity troponin level was ordered 2 hours after the first and came back at 8,109 ng/L. The patient was treated with a full-dose (325 mg) Aspirin and heparin bolus and infusion was initiated for presumptive non-ST elevation myocardial infarction (NSTEMI). Patient was also treated with Pepcid 20 mg PO, Metoprolol tartrate 25 mg PO, Hydrocodone-acetaminophen 5-325 mg PO, morphine 4 mg IV, and Zofran 4 mg IV. Despite initial analgesia, the patient complained of persistent pain. He was given nitroglycerin 0.4 mg sublingual with symptomatic relief. Blood pressure had improved to 134/82 mmHg.

Arrangements were made initially for admission and patient was accepted to the hospitalist service. Cardiology was consulted and the patient was arranged to undergo a full cardiac workup as an inpatient. The cardiologist evaluated the patient at bedside and a third ECG was ordered while still in the Emergency Department. This third ECG revealed sinus rhythm with 1st degree AV block at 66 beats per minute, normal axis, persistent inferolateral ST depressions, and now with biphasic T-wave inversions in leads V1 through V3. This appeared highly suspicious for Wellen's syndrome, suggestive of acute anterior wall myocardial infarction. The third troponin level drawn 2 hours after the second was noted to be 23,000 ng/dL. Patient was then emergently transferred to the catheterization lab where he was noted with 100% LAD occlusion. Patient underwent PCI and one stent was placed. The patient was discharged on DAPT, statin and beta blocker the next day.

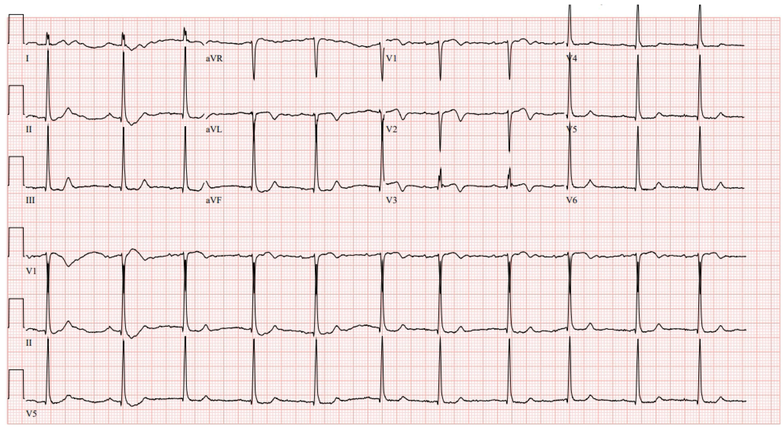

Figure 1: An ECG tracing demonstrating the pattern of Type-B Wellen's Syndrome, with biphasic T-waves in leads V2 & V3.

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Discussion:

Wellen's syndrome describes an abnormal electrocardiogram (ECG) pattern that is highly specific for proximal stenosis of the left anterior descending (LAD) artery [2]. There are two variations that are seen on ECG. Type A is the more common abnormality, characterized by deep T-wave inversions in leads V2 and V3. Type B is less common and is characterized by biphasic T-waves in V2 and V3 [3]. For the diagnosis of Wellen's Syndrome to be made, in addition to these ECG changes, the patient must be pain-free at the time of ECG tracing. Cardiac enzymes may also be normal or slightly elevated [4]. It has been estimated that approximately 75% of patients diagnosed with Wellen's Syndrome that are untreated will go on to develop extensive anterior wall myocardial infarction in the days to weeks following admission [5].

Acute myocardial infarction is one of the leading causes of death worldwide. Recent studies have suggested the prevalence of Wellen's syndrome among patients with acute coronary syndrome to be approximately 5.7% [6]. The mainstay of treatment for these conditions include aspirin and antiplatelet therapy, as well as emergent catheterization and percutaneous intervention (PCI). In some cases when PCI is not available in less than 120 minutes, thrombolytics are recommended [7]. Due to the high morbidity and mortality of acute myocardial infarction, timely diagnosis and intervention is paramount.

When discussing myocardial infarctions, one often mentally sorts the pathology into 2 categories: STEMI or NSTEMI. Despite the infamous ECG presentation of ST-segment elevation, there are other ECG patterns that can represent similar pathology that are just as dangerous, and just as crucial to diagnose. Wellen's Syndrome is one of those patterns. The de Winter pattern is another finding that does not present with an obvious ST-segment elevation, yet does represent an acute myocardial infarction [8]. These two patterns have been at the forefront of a proposed paradigm-shift in the diagnosis of acute myocardial infarctions. It has been suggested that instead of the STEMI/NSTEMI nomenclature, we use the titles acute coronary occlusion (ACO) or occlusion myocardial infarction (OMI)/non-OMI (NOMI) instead [9]. This would encompass the pathology in all of its various presentations - STEMI, Wellen's Syndrome, and de Winter patterns, together under one category. This would lead to increased notoriety and recognition of atypical ECG patterns that are specific for the same potentially life-threatening pathology.

Conclusions:

We reported a case of myocardial infarction due to LAD occlusion in a patient who presented with dynamic ECG changes. The ST-segment depressions seen initially were followed by a byphasic T-wave inversion pattern that is formally known as Wellen's syndrome. Wellen's syndrome can be an example of occlusive myocardial infarction which does not present like the typical ST-segment elevation. Myocardial infarction can present in different forms, and it is crucial to monitor and trend electrocardiogram changes which can reflect a dynamic disease process.