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Case Report: Wellens Syndrome in 28-year-old with Pleuritic Chest Pain

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Abstract:

We report a case of a 28-year-old male with months of diffuse, pleuritic, intermittent, non-exertional chest pain. He had an elevated troponin and ECG showing Wellens' syndrome and was found to have 80% LAD coronary artery occlusion and 100% apical occlusion. Identification and proper management of Wellens' syndrome relies on being familiar with its ECG patterns. Other information such as age, cardiac risk factors, chest pain with exertion and at rest, and elevated troponin, are all helpful supplemental information, but as demonstrated in the following case report, presentations may vary or even cause as distractors to the diagnosis.

Case Presentation:

A 28-year-old male presented to the emergency department (ED) with a complaint of four to five months of intermittent, diffuse chest pain that lasted for 2 to 5 minutes intervals, was worse with deep breaths, non-exertional, and which improved when stretching out his chest wall and especially with drinking water. His last episode occurred while in the shower and was 3.5 hours prior to presentation to the ED. He had no past medical history. He had not been lifting anything heavy recently and denied any recent trauma to his chest wall. He claimed that the pain occurred up to several times per day and seemingly randomly such as while in the shower, while at rest, and while driving his car. He denied any recent illnesses, recent long-distance travel, recent procedures, history of blood clots, or hormone therapy. He denied any history of sudden cardiac death in his family and denied any drug use besides smoking marijuana weekly.

The patient's vital signs were as follows: blood pressure 105/55, heart rate 66, temperature 98.3°F, respiratory rate 20, and resting comfortably at time of presentation. Physical exam showed a healthy appearing male without chest wall tenderness, without rubs, gallops, or murmurs, and clear to auscultation of the lungs bilaterally. The ECG was normal sinus rhythm, had a prolonged PR interval indicative of 1st degree atrioventricular block, and showed biphasic T-wave in V3 and V4 characteristic of type A Wellens' syndrome along with T wave inversions in V3-V6, II, III, and aVF [Figure 1]. His CBC and his BMP were within normal limits except for an elevated platelet count of 526 B/L. His drug abuse panel had no positive values. His sedimentation rate and hepatic function panel were within normal limits. Abnormal values included elevated C-Reactive Protein of 0.70 mg/dl, high sensitivity troponin of 220 ng/L with a repeat high sensitivity troponin of 182 ng/L taken two hours later. The chest x-ray was unremarkable. Cardiology was consulted over the phone and was given oral Colchicine, chewable aspirin 162mg, and told to not eat until he was seen by cardiology in the morning. The patient was then admitted to the hospital.

A computerized tomography angiogram (CTA) scan of the chest was completed with contrast for concern of pericarditis and pulmonary embolism. The results of this study were unremarkable. Transthoracic Echocardiogram was also unremarkable and without pericardial effusion. The patient had a left heart catheter (LHC) placed and computerized tomography coronary angiogram (CTCA) which showed 80% proximal left anterior descending (LAD) coronary artery and 100% apical occlusions [Figure 2]. The patient was then started on heparin drip and transferred to another hospital for percutaneous intervention the next day. Following the intervention, the patient was started on baby aspirin, clopidogrel and atorvastatin.

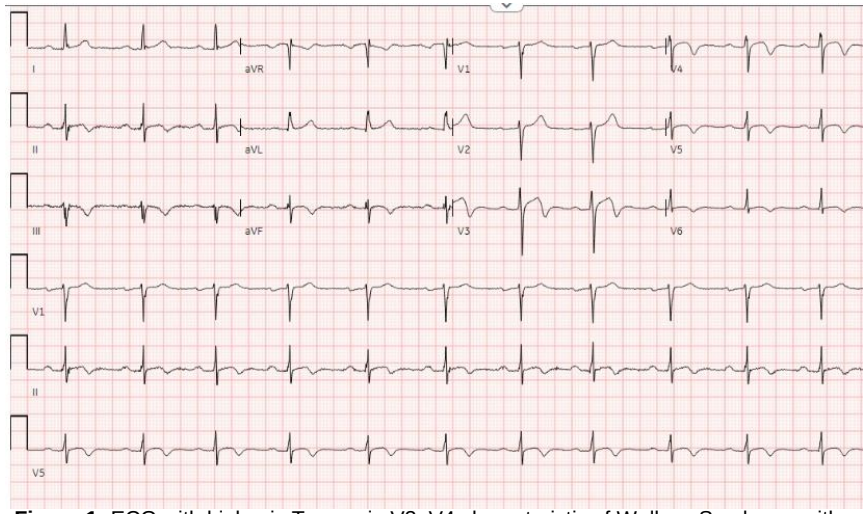

Figure 1: ECG with biphasic T-wave in V3, V4 characteristic of Wellens Syndrome with T wave inversions in V3-V6, II, III, & aVF

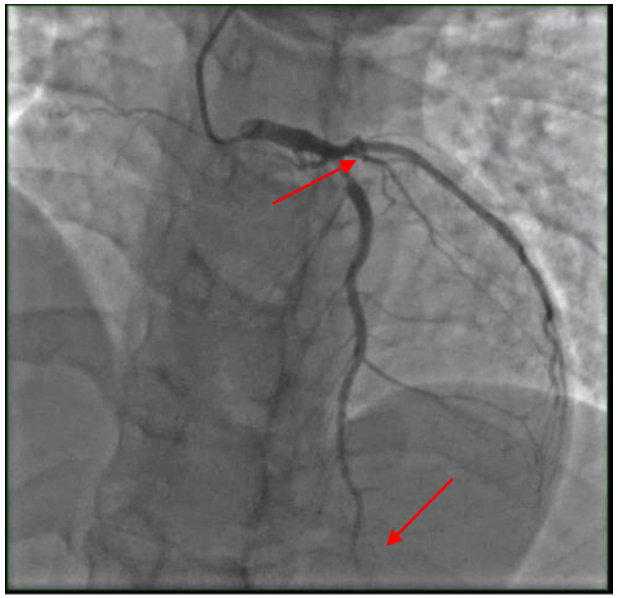

Figure 2: CTCA: Top arrow pointing to 80% proximal LAD occlusion and bottom arrow to 100% apical occlusion

Discussion:

Pathophysiology:

It was first described in 1982 by H. J. J. Wellens et al as one of two characteristic ECG patterns of T-waves in the precordial leads that have an association with critical stenosis of the proximal left anterior descending coronary artery. [1] The exact mechanism of what causes these specific ECG changes is not certain, but because the ECG changes occur when the patient is pain free they are thought to be due to reperfusion of the ischemic myocardium due to alleviation of spasm of the proximal LAD. [2, 3, 4] Others have considered the syndrome may be due to myocardial stunning or myocardial hibernation.

Incidence and presentation of Wellens Syndrome:

In the largest study available on Wellens' syndrome to date, in 3528 patients who had acute coronary syndrome, the incidence of Wellens' syndrome was 5.7% (200 of 3528). In this same group of patients, those with Wellens' syndrome were much less likely to have pre-existing coronary heart disease (39.6% vs 23%) and previous PCI (19.5% vs 9%), which is consistent with our patient who had no previous medical history.

Types of Wellens' Syndrome

Wellens' syndrome may present on ECG in one of two categories, type A or type B. Type A has biphasic T-waves in leads V2 and V3 that are initially positive and terminally negative, while type B has deeply symmetrical T-wave inversions in leads V2 and V3, often including the other precordial leads as well [5]. Approximately 24% of Wellens are type A, and this finding is more specific for Wellens' syndrome. The remaining 76% of cases are type B which are less specific, thus our patient presented with the rarer form of Wellens' syndrome; type A. [6]

Variations in presentation

Uncharacteristically, our patient was 28 years old compared to the average age of those with Wellens' syndrome being 63 years old + or – 10.5 years for one standard deviation according to Zhou et al. [7]. Our patient also presented with pleuritic chest pain and improvement with stretching of the chest wall and with drinking water, all of which are not patterns evident in any of the current data or case reports available. Each of these are possible detractors from the actual diagnosis and could lead us to believe that this pain was esophageal or pleuritic in nature rather than a myocardial infarction.

Laboratory studies, imaging, and management:

Due to the suspected pathophysiology of Wellens' syndrome many patients who present do not have elevated cardiac troponin levels. Those patients that do present immediately after an episode of chest pain are more likely to have elevated levels such as our patient. Current reports indicate that 69% of Wellens' syndrome present as NSTEMI while 31% present as unstable angina pectoris (no elevation of troponin) [7]. In Wellens' syndrome, early consultation of cardiology for LHC with CTCA is essential to properly identify areas of occlusion and for greatest survival outcomes. Early PCI is the preferred treatment. [7, 8]

Conclusions:

Wellens' syndrome can present similarly to NSTEMI or unstable angina, but there are differences between them as well as variations within presentations of Wellens' syndrome. Wellens' may present with findings such as normal troponin level, nonstandard presentations of chest pain, variations in age, and lack of cardiac risk factors. This was exemplified by our case of a 28-year-old male with no past medical history who presented with pleuritic chest pain and was found to have PDA occlusion requiring percutaneous intervention (PCI).

References:

Available on request