J Clin Exp Cardiolog 2021, Volume 12

LAD not PUD, a case of missed Wellen's syndrome

Joseph Abraham, Ebenezer Fozao, Roman Zeltser and **Amgad Makaryus**
Nassau University Medical Cener
Nassau University Medical Center

Wellens' Syndrome is a pattern of electrocardiographic T-wave changes associated with critical, left anterior descending (LAD) artery stenosis. Diagnostic criteria of Wellens' Syndrome are a history of chest pain, little or no cardiac enzyme elevation, little or no ST-segment elevation, no loss of precordial R waves, no pathologic precordial Q waves, and typical T-wave changes. Urgent cardiac catheterization is vital to prevent myocardial necrosis.

It is important for the emergency physicians and the whole emergency team to recognize the typical ECG findings of Wellens' Syndrome because these characteristic ECG findings are considered as a marker for critical LAD occlusions.

Case report: 61 years old Hispanic female with past medical history for gastritis and peptic ulcer disease (2004) presented to the Emergency Department on May 21st complaining of substernal chest pain since May 4th, 2019 almost for 3 weeks. She was seen at a local hospital in Pennsylvania and was diagnosed with gastritis and sent home on Omeprazole 40mg and antacids which were of little help in relifing her pain. she described the pain as substernal pressure like 9/10 in intensity radiating to the left arm associate with shortness of breath. Also, pain is intermittent and exacerbated by exertion and relieved by rest. She doesn't eat spicy food, Alcohol, Caffeine. She had a positive family history of heart attacks. Denied nausea, vomiting, palpitations, dizziness, fever, chills, headaches. Initial EKG was normal sinus rhythm with biphasic T wave in the anterior chest leads with poor R wave progression V1-3

The patient was admitted to the telemetry and a full workup was done for her including Troponins and Echocardiogram.

Physical exam: BP: 159/89, temp: 36.4. HR:70 bbm

Troponin 1.250 ng/ml.

Echo: EF = 40-45% visual estimation, moderate decrease global LV systolic function., Multiple LV regional wall motion abnormalities exist in the LAD territory.

The patient was diagnosed with NSTEMI given proper anticoagulation and transferred to NS-LIJ for cardiac catheterization, a long 99% stenotic tubular Mid- LAD lesion was found TEMI flow grade II, Balloon dilation followed by stenting placement was done for the culprit lesion, TEMI flow grade I regained.

patient-reported improvement of her breathing and no more chest pain after the procedure.

Conclusion: The diagnosis and management of Wellens syndrome are done by a multidisciplinary team. As soon as the diagnosis of Wellens syndrome is made or suspected, the cardiologist must be consulted. Until this occurs, treat Wellens syndrome similarly to an acute myocardial infarction including antiplatelet therapy with aspirin, anticoagulation with heparin, and nitrates and beta-blockers if the patient is not hypotensive. However, it is important to note that Wellens' patients do poorly with medical management alone, definitive treatment is procedural. These patients must always be admitted and monitored. An immediate cardiac catheterization is necessary. Because Wellens syndrome patients have critical narrowing of the LAD coronary artery, a stress test should be avoided as this can precipitate an acute myocardial infarction and sudden death. Upon coronary angiography, the cardiologist can plan for revascularization of the LAD coronary artery. The prognosis for patients who are managed with surgery or PCI is good, but if treatment is delayed or medical therapy is undertaken, the outcomes are poor. It's better to be early recognized by the ER team before losing any part of the myocardium. Raising awareness of wellen's syndrome among ER physicians and nurses can Save many patients.