Wellens Syndrome: A Rare ECG Finding in Subendocardial Myocardial Infarction

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Case Report

ABSTRACT

Wellen's Syndrome describes a pattern of electrocardiographic (ECG) changes, particularly deeply inverted or biphasic T waves in leads V2-V3, which is specific for proximal stenosis of the left anterior descending (LAD) coronary artery. It is also known as 'Anterior Descending T-Wave Syndrome'[1]. These patients usually do not report any chest pain and their cardiac enzymes are normal or only mildly elevated. Nevertheless, it is crucial to recognize ECG patterns as these patients are at high risk for imminent large 'Anterior Wall Acute Myocardial Infarction'. We illustrate here in an asymptomatic patient reporting in the emergency department with history of chest pain. He was identified as a case of 'Wellen's Syndrome' based on typical ECG findings.

KEY WORDS: electrocardiographic (ECG), left anterior descending (LAD), syndrome

INTRODUCTION:

Wellens' Syndrome is recognized by T-wave changes in pre-cordial leads of an electrocardiogram (ECG) with proximal stenosis of the left anterior descending (LAD) artery, and is frequently associated with acute myocardial infarction and sudden cardiac death[2]. However, this syndrome is not always an acute process. T-wave alterations in Wellens' syndrome are typically seen in asymptomatic patients and even though these patients may at first respond well to medical management, they eventually fare inadequately with conservative therapy and require revascularization[3]. It is vital to spot the electrocardiogram characteristics of Wellens' syndrome in order to provide a suitable intervention, or to be able to intervene before myocardial infarction develops[4]. The ability of ECG`s to assist decision-making in severe myocardial infarction is straightforward, particularly in patients with characteristic ECGs. However, the Wellen's syndrome has broad spectrum of clinical signs and the ECG patterns may manifest itself persistently over weeks. Therefore, ECG parameters should be combined with coronary angiography to verify the presence of lesions.

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We present a case report of a middle aged man identified from history and ECG findings indicative of Wellen's Syndrome.

CASE REPORT:

A 50 year old man reported to the medicine OPD (out patient department)for routine investigation of chest pain since past 10 days. At presentation, the patient was asymptomatic and was sitting comfortably. He started having chest pain 10 days back, which was crushing in nature, retrosternal, increased on exertion, relieved at rest and was radiating to the jaw and both arms. He was a government employee, working 10-12 hours a day and was chronic smoker for about 30 years, smoking 10-12 cigarettes per day. He was also a tobacco chewer and consumed around 180 ml of hard liquor every day. On examination, Pulse was 90/minute, regular. BP was 160/100 mm of Hg in right arm supine position and random blood sugar was 109 mg/dl. General and Systemic examination were normal. ECG (Figure 1) showed typical pattern consistent with Wellens Syndrome with Biphasic T waves (with initial positivity and terminal negativity) in V2 and V3, T wave inversion in I and avL and II, III and avF and Deeply inverted T waves in leads V4, V5, V6. Troponin I was negative and CPK-MB was mildly elevated 31 IU/L (Normal <24 IU/L). Hemoglobin of 17.1 gm% reflected his chronic smoking. Total Leukocyte Count and Platelets were normal. 2D echocardiography showed CAD(Coronary Artery Disease) with RWMA (Regional Wall Motion

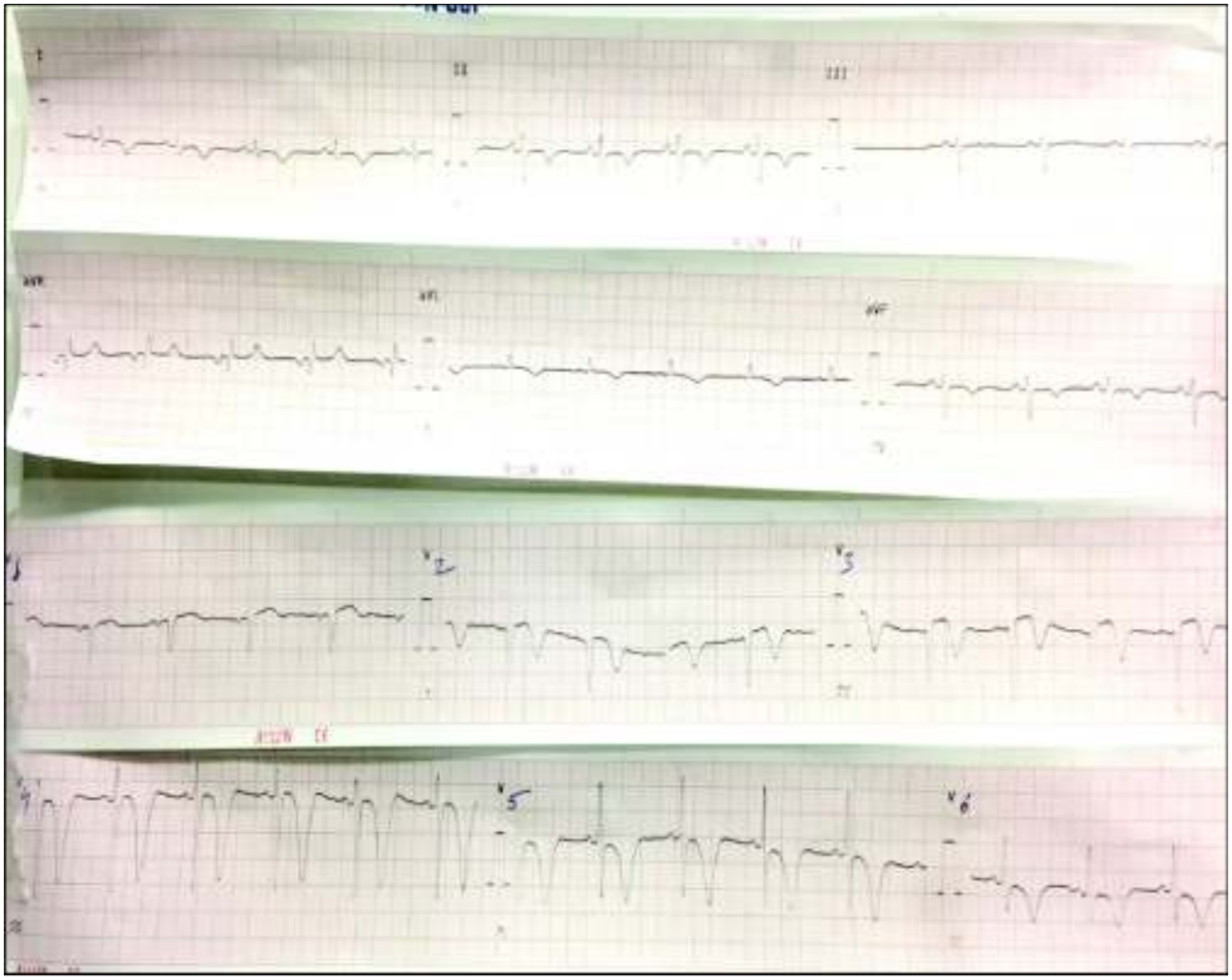

Figure 1: ECG in the reported case of Wellen's Syndrome.

Abnormality) involving apical septal wall of Left Ventricle(LV), EF (Ejection Fraction)=55% and normal LV systolic function.

DISCUSSION:

Wellens' Syndrome results from transient obstruction of the LAD coronary artery. Generally, this is caused by the rupture of an atherosclerotic plaque leading to LAD occlusion, with consequent clot lysis or other disruption of the occlusion before complete myocardial infarction takes place. Wellens' Syndrome denotes a pre-infarction state. These patients usually show symptoms of acute coronary syndrome. The characteristic complaints include chest pain, explained as tightness or pressure, often provoked by physical activity and relieved by rest. The pain may radiate to the neck, jaw, or shoulder. Upon presentation to the emergency room, patients usually do not present with pain. However, the typical ECG pattern may persist. Patients appear comfortable on physical examination though they may exhibit mild distress with diaphoresis, comparable to those patients having an acute myocardial infarction.

Risk factors of Wellen's Syndrome include diabetes mellitus, family history of premature heart disease, hypertension, increased age, hypercholesterolemia, hyperlipidemia, metabolic syndrome, occupational stress and smoking. Diagnostic criteria for Wellens syndrome include: deeply inverted T waves in leads V2 and V3 (may also be seen in leads V1, V4, V5, and V6; or biphasic T waves (with initial positivity and terminal negativity) in V2 and V3[5][6] PLUS a) isoelectric or minimally elevated ST segment, less than 1 mm (in other words, no signs of an acute anterior wall myocardial infarction); (b) preservation of precordial R-wave progression and no precordial Q waves (in other words, no signs of old

anterior wall infarct); (c) recent history of angina; (d) ECG pattern present in a pain-free state; and, (e) normal or slightly elevated cardiac markers.

Two patterns of T waves can be seen in Wellens syndrome. Type-A T waves are biphasic, with initial positivity and terminal negativity. These T waves are present in approximately 25% of cases. Type-B T waves are deeply and symmetrically inverted. These are present in approximately 75% of cases. The 2 types of T waves found in Wellens syndrome exist on a spectrum of disease with type-A T waves developing into type-B T waves.The T-wave abnormalities may be constant for hours to weeks, even when the patient is pain-free.

Wellens syndrome is not always an acute process. It can develop over days to weeks. The ECG pattern frequently develops when the patient is not feeling chest pain as evident in this patient. Cardiac biomarkers including troponin may be incorrectly reassuring in patients with Wellens syndrome as they frequently result within normal limits.

CONCLUSION:

Wellens syndrome is a result of critical stenosis of the left anterior descending coronary artery apparent in characteristic ECG changes. It is a pre-infarction state. However, if not recognized early and properly treated, the disease tends to progress to a large acute anterior wall myocardial infarction, which can lead to considerable morbidity and mortality. Therefore it is important for clinicians to be familiar with its patterns at the earliest opportunity. It has wide spectrum of clinical expressions, and although characteristic ECG identification of Wellens' syndrome can offer diagnostic confirmation, additional CAG is needed to make a definite diagnosis.The risk factors, characteristic ECG, 2D ECHO findings and biochemical markers point strongly towards Wellens Syndrome. Early diagnosis shall enable averting further cardiac complications including Myocardial Infarction.

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Cite this article as: Puraskar P & Bhatnagar A: Wellens Syndrome: A Rare ECG Finding in Subendocardial Myocardial Infarction. . PJSR ;2019:12(2):45-47.
Source of Support : Nil, Conflict of Interest: None declared.