Case Report

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Wellens' Syndrome: An Acute Coronary Syndrome, a Diagnosis often Misssed in Emergency Department

Tanmay Kumar Jha[1], Chintala Venkata Sai Chiranjeevi[2], Gebran Manzoor[3], Sarat Kumar Naidu[4], Dheeraj Bhaskaran Nair[5]

Author's Affiliation:
[1]DNB Resident [2]Senior Resident [3]MEM Resident [4]Attending Consultant [5]Head of Department, Department of Emergency Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh 201012, India.

Corresponding Author:
Tanmay Kumar Jha, DNB Resident, Department of Emergency Medicine, Max Super Speciality Hospital, Vaishali, Ghaziabad, Uttar Pradesh 201012, India.

E-mail: drtanmaykumarjha@gmail.com

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Abstract

Wellens' syndrome is a pre-infarction stage of critical proximal left anterior descending (LAD) coronary artery stenosis in patients with angina chest pain without typical ECG changes nor with elevated cardiac enzymes. Misdiagnosis or delayed diagnosis of this condition can lead to full-blown myocardial infarction and death if not intervened appropriately. We describe a case of 59 years old male who has had recurrent episodes of chest painwithout typical ECG changes and significant cardiac markers but was found to have critical LAD stenosis on angiography. We also describe the diagnosis criteria of Wellens' syndrome and its importance.

Keywords: Wellen's syndrome; Biphasic T wave; Inverted T-wave; Left Anterior Descending Artery (LAD); LAD Critical Stenosis; Coronary Angiography (CAG); Primary PTCA + Stent to LAD; Anterior Wall Myocardial Infarction (AWMI); Cardiac enzymes; Left Ventricular Dysfunction; Left Bundle Branch Block (LBBB).

Introduction

Wellens' syndrome is an ECG manifestation of proximal left anterior descending (LAD) coronary artery critical stenosis in patients with unstable angina [1]. It is actually apreinfarction stage of coronary artery disease. In 1982 Wellens et al. first published the ECG and clinical criteria of a subgroup of patients with myocardial ischaemia that later came to be known as Wellens' syndrome. This ECG pattern recognition allowed the identification of patients who had proximal left anterior descending (LAD) coronary artery critical stenosis and thus were at increased risk of developing extensive anterior wall myocardial infarction. In this syndrome the T-wave changes usually occur during a pain-free period. Although these patients might respond well to medical management initially, they ultimately have poor outcomes with a conservative therapy and require revascularisation therapy.

Wellens' syndrome is also referred to as LAD coronary T-wave syndrome. The criteria for this syndrome include the following.

1. History of anginal chest pain;
2. Precordial biphasic/inverted T-wave changes
3. Normal or mildly elevated cardiac enzyme levels;
4. ECG without significant ST-segment elevation, without Q-wavesand with normal R-wave progression in precordial leads.

The risk factors for the development of this condition are: smoking, hypertension, diabetes mellitus, advanced age, hypercholesterolaemia, hyperlipidaemia, metabolic syndrome and family history of premature heart disease [2].

There are 2 patterns of T-wave abnormality in Wellens' syndrome:

- *Type A* = Biphasic, with initial positivity & terminal negativity (seen in 25% of cases)
- *Type B* = Deep (>2mm) and symmetrically inverted (seen in 75% of cases)

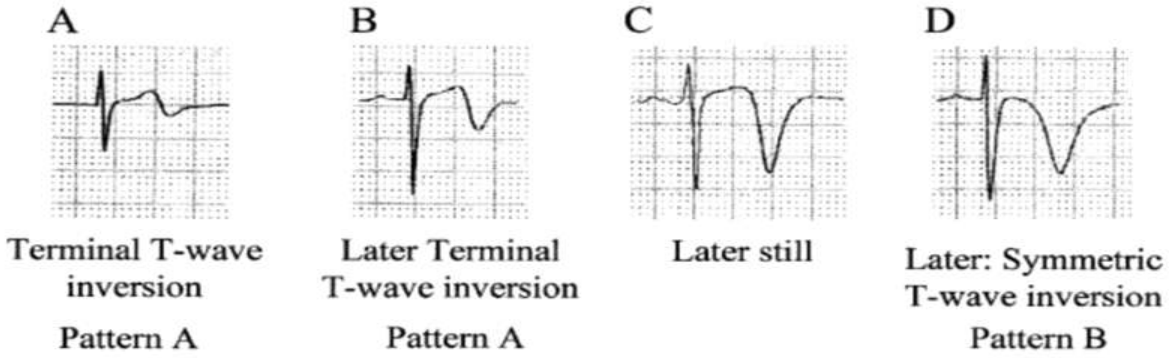

Fig. 1: T wave abnormalities in Wellens' syndrome

The T waves evolve over time from a Type A to a Type B pattern.

In patients admitted to the hospital because of unstable angina, a subgroup was recognized which was at high risk for the development of an extensive anterior wall myocardial infarction. These patients, who show characteristic ST-T segment changes in the precordial leads, have a critical stenosis in the proximal LAD. Urgent coronary angiography and, when possible, coronary revascularization therapy should be done in patients with unstable angina who show this ECG pattern [3].

In risk stratification of patients with unstable angina, the ECG is of great value for recognizing a subset of patients with a proximal LAD lesion having a poor prognosis because of a substantial area of preinfarction myocardium. Early cardiac revascuarization is indicated [4].

Normally the activation of septum occurs from left to right, producing small Q waves in the lateral leads. In LBBB, the normal direction of septal depolarisation becomes right to left, because the impulse spreads first to the right ventricle via the right bundle branch and then to the left ventricle via the septum.

This sequence of septum activation from right to left extends the QRS duration to more than 120 ms and eliminates the normal septal Q waves seen in the lateral leads.

This overall right to left depolarisationdirection produces tall R waves in the lateral leads (I, V5-6) and deep S waves in the right precordial leads (V1-3), and often leads to left axis deviation.

Because the ventricles are activated sequentially from right to left rather than occurring simultaneously, this produces a broad or notched ('M'-shaped) R wave in the lateral leads [5].

ECG diagnostic criteria of LBBB

1. QRS duration of > 120 ms with 'W' pattern in V1/V2 and 'M' pattern in V6/V5.
2. Dominant S wave in V1.
3. Broad monophasic R wave in lateral leads (I, aVL, V5-V6) with T inversion.
4. Absence of Q waves in lateral leads (I, V5-V6; small Q waves may be seen in aVL)
5. Prolonged R wave peak time > 60ms in left precordial leads (V5-6) [5].

New LBBB along with chest pain or even otherwise is traditionally considered part of the criteria for evaluation for ACS and thrombolysis or revascularization.

However, more recent data suggests that patients presenting with new onset LBBB have little increased risk of acute myocardial infarction at the time of initial presentation [5].

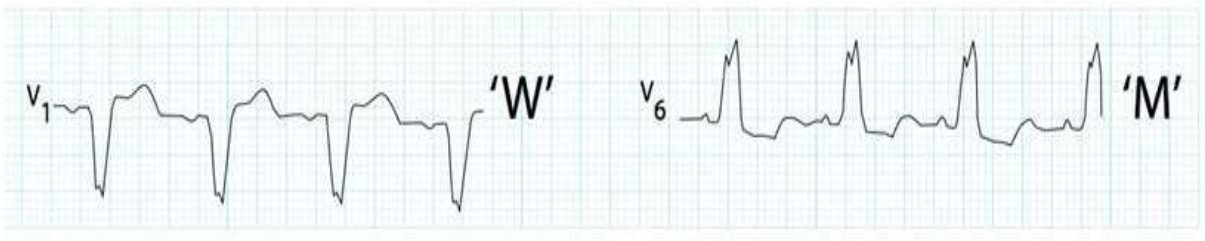

Fig. 2: ECG changes in LBBB

Case Study

A 59 years old male of Indian origin with past history of hypertension on Tab. Telmisartan 40 mg OD and Tab.Lasilactone (Furosemide 20mg + Spironolactone 50mg) OD presented to the Emergency Department (ED) of our hospital with the C/O left sided chest pain lasting for around 15 minutes, 1 hour back. At the time of presentation to ED he had no chest pain and was complaining of mild epigastric discomfort. He gave h/o similar episodes of chest painfor the past 1 month which subsided on its own.

He was taken to a monitored bed and initial evaluation was done. He was conscious and cooperative and oriented to time, place and person. His pulse rate was 52/min, blood pressure was 100/50 mmHg, respiratory rate was 18/min, temperature was 98.6^{0}F, random blood sugar was 92 mg/dl and SpO_2 was 98% on room air. He did not have any pallor, icterus, cyanosis, jugular venous distention nor any peripheral edema.

Cardiac monitor showed sinus rhythm and the 12-lead ECG showed deeply inverted T waves in leads V2,V3,V4 along with no precordial Q waves and preserved precordial R wave progression. Initial ECG of the patient is shown below (Figure 3).

A large i.v. cannula was inserted in left cubital vein and samples were taken for ABG, cardiac enzymes, D-dimers and BNP, CBC, LFT and KFT. Initial cardiac enzymes sent from the ED were later found to be within normal ranges.

Cardiology consultation was sought and a provisional diagnosis of Type B Wellen's syndrome was made. After stabilization in the ED the patient was immediately shifted along with ED personnel and cardiologist with defibrillator attached, pulse oximeter and a crash cart to the catheterization laboratory. On the way to the cathlab, he developed severe chest pain and then became mildly drowsy. He was shifted to CCU for evaluation before taking him to cathlab. He was responding to verbal commands but was drowsy. Pulse was 82/min, BP 90/40 mmHg, RBS 102mg/dl, SpO2 88% on O2.

A 2nd ECG was done which was suggestive of LBBB (Figure 4). This was a new onset LBBB which was highly indicative of ACS in conjunction with the symptoms and earlier ECG findings.

He was immediately given loading dose of ecosprin 325mg, clopidogrel 600mg, atorvastatin 80mg and then shifted to cathlab for CAG.

CAG was performed in this patient in view of recurrent H/O chest pain, Wellens' syndrome type 2 and new onset LBBB.

Coronary Angiography immediately which showed mid thrombotic lesion with 85% stenosis in LAD (Single Vessel Disease) and thus confirming the diagnosis.

Subsequently he underwent primary PTCA + Stent (DES) to LAD.

Post procedure 2D Echo was done which showed LVEF= 45%. Post procedure Gastroenterology consultation was sought in view of epigastric discomfort and advice incorporated. Ultrasonography Whole Abdomen revealed no significant abnormality.

He was kept in the CCU for 2 days for observation and started on post-stenting medication. He was discharged in a stable condition with the following medication advice Cap. Clopitorva (Atorvastatin 10 mg + Clopidogrel 75 mg) OD, Tab. Telmisartan 40 mg OD, Tab.Lasilactone (Furosemide 20mg + Spironolactone 50 mg) OD, Tab. Lesuride 25 mg TDS, Tab. Pantoprazole 40 mg OD.

The patient was followed up in Cardiology OPD after 5 days and was found to be stable and symptom-free.

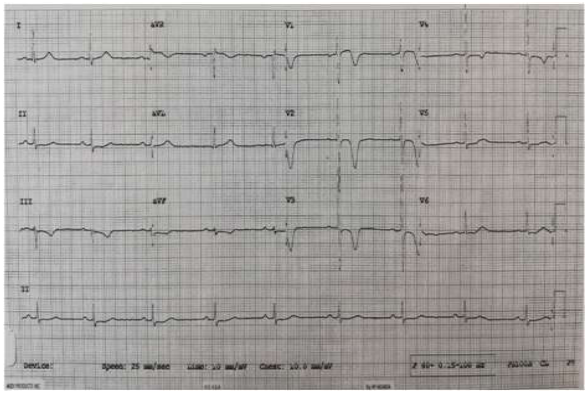

Fig. 3: Initial ECG of the patient showing Wellens type B

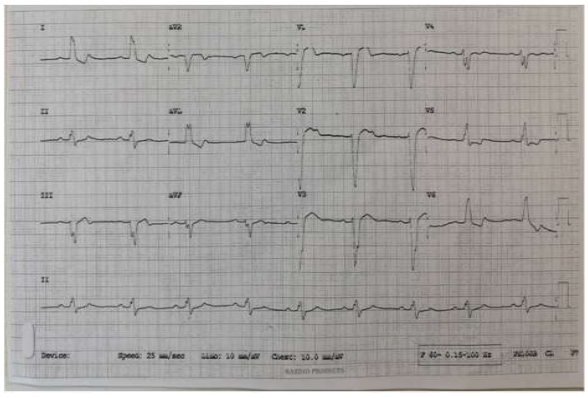

Fig. 4: 2nd ECG of the patient showing LBBB

Discussion and Therapeutic Considerations

The diagnostic criteria for Wellens' syndromeare:

- Deeply-inverted or biphasic T waves in V2-3 (which may extend to V1-6)
- Isoelectric or minimally-elevated ST segment (< 1mm)
- No Q waves in precordial leads
- Precordial R wave progression is preserved
- Recent history of angina
- Characteristic ECG pattern is present in pain-free interval
- Normal or minimally elevated serum cardiac ezymes [6]

The sequence of events believed to occur in patients with Wellens' syndrome is:

Sudden occlusion of the LAD, which causes a transient anterior STEMI. The patient has chest pain & diaphoresis. This stage may be missed on an ECG recording as the characteristic ECG changes may not be present.

Re-perfusion of the LAD occurs (for example, due to spontaneous clot lysis). The chest pain is relieved. ST elevation settles and the T waves become biphasic or inverted. This T wave morphology is similar to patients who reperfuse after a successful PCI.

If the artery does remain open, the T waves over time evolve from biphasic to deeply inverted.

However this coronary perfusion is unstable, and the LAD can occlude again at any time. When this happens, the first sign on the ECG is that T waves normalize, also called as "pseudo-normalisation". The T waves switch from biphasic or inverted to upright and prominent. This is suggestive of hyperacute STEMI and is usually accompanied by chest pain recurrence, although the ECG changes can occur before the symptoms.

If the LAD remains occluded, the patient develops an evolving anterior wall STEMI.

Sometimes, a "stuttering" pattern may develop, with intermittent reperfusion and occlusion. This would be seen as alternating ECGs demonstrating Wellens' and pseudo-normalisation/STEMI patterns.

This sequence of events is not only seenin the anterior leads. Similar changes may be seen in the inferior or lateral leads. Also, the initial event does not necessarily have to be thrombus formation. Wellens' syndrome may also occur in normal coronary arteries following a vasospasm episode. However, it is better to assume critical LAD stenosis and order and evaluate an urgent angiogram [7].

Some issues relate to the performance of PCI and these include the development of bare metal stents (BMS), which have surpassed balloon angioplasty in the management of coronary artery disease because of their ability to prevent restenosis by suppressing arterial recoil and contraction. However, 10% to 20% of patients may still experience restenosis because of excessive growth of a neointima. These have been reduced due to the development of drug eluting stents (DES), which have led to reduced revascularization rates by as much as 70%. For this reason, DES is preferred in the majority of PCI procedures [8].

In view of the potential extensive area of myocardium at risk, the importance of recognizing the ECG pattern of Wellen's syndrome could not be stressed more for the emergency department physicians [9].

The most common symptom of myocardial ischemia and myocardial infarction is retrosternal chest discomfort. The patient may perceive this discomfort more as a pressure or tightness than actual pain.

Symptoms suggestive of ACS may also include:

1. Uncomfortable pressure, fullness, squeezing or pain in the center of the chest lasting more than several minutes.
2. Chest discomfort spreading to the shoulders, neck, one or both arms, or jaw.
3. Chest discomfort spreading into the back or between the shoulder blades.
4. Chest discomfort occurring withdizziness, light-headedness, fainting, sweating, nausea or vomiting.
5. Sudden shortness of breath which is unexplained, which may occur with or without chest discomfort [10].

In this case unless an experienced ECG interpreter could diagnose Wellen's syndrome, it is difficult to make a diagnosis since the patient was symptom free at the time of presentation and the cardiac enzymes were also negative. With high degree of suspicion and timely interventions, our patient was treated and saved.

Conclusion

Why is it difficult to diagnose Wellens syndrome in the ED and why is it often missed during initial

asessment? The following discussion will answer the question.

Firstly the symptoms are not as typical as of a STEMI. Patient gives a vague h/o recurrent episodes of chest pain or discomfort. If ECG is done during the pain, it shows normal findings with upright T (may not be tall).

If the pain is relieved, the patient usually doesn't come to ED.

Very few patients with recurrent episodes may present to ED and if the ECG is taken during the pain-free period, it may show either of the type A or type B patterns which are not known to many physicians as Wellens. Moreover in the ECG, there is no Q waves nor poor progression of R waves which are usually present in an ACS.

The cardiac enzymes are mostly normal or may sometimes be mildly elevated and this can further mislead the physicians.

All these lead to discharging the patient after observation without doing a CAG and this can cause a full-blown AWMI within a few weeks of discharge.

All physicians in general and Emergency physicians in particular must be aware of this syndrome with these ECG changes and must inform the concerned cardiologist for further interventions to save the patient's heart before progressing to infarction.

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