Wellens' Syndrome with LBBB Presentation and Post Revascularization T-wave Normalization

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Citation

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Abstract

Wellens' syndrome is a pattern of electrocardiographic (ECG) T-wave changes associated with critical, proximal left anterior descending artery (LAD) lesions. The classic ECG presentation is in the painless pre-infarction stage of coronary artery disease, typically in the context of recurring episodes of unstable angina (UA) or an uncaptured transient episode of anterior ST-elevation myocardial infarction (MI). We present a case of Wellens' syndrome with initial ECG showing left bundle branch block (LBBB) followed by classic Wellens' T-wave inversions after treatment of pulmonary edema. We also documented the T-wave changes post-LAD revascularization, in which T-wave normalization occurred after 4 months.

CASE PRESENTATION

A 68-year-old male presented to the emergency room with sudden onset of 9/10 retrosternal chest pain with radiation to his back followed by shortness of breath that began 3 hours prior to arrival. The patient denied similar past episodes and stated his current episode was precipitated by taking a routine walk around the neighborhood while having had a cold with cough for 3 days.

The patient had a medical history of hypertension and diabetes type 2, but has had no medical follow up for 3 years and was not taking any medications at time of presentation. The patient had no other pulmonary or cardiac history. The patient had bilateral knee surgery and lumbar surgery after a work-related accident 6 years prior with a preoperative 2D echocardiogram showing an ejection fraction of 60%. The patient had a 40+ pack-year history of smoking.

The initial workup on admission was significant for HR of 140, RR of 34, BP of 182/92, WBC of 14.5, lactic acid of 4.0, and troponin 0.11. Chest X-ray was significant for pulmonary edema. Initial ECG performed upon arrival to the ER while patient was having chest pain revealed a new LBBB (Figure #1). Ultimately, the patient was admitted for pulmonary edema. He was not taken to cardiac catheterization laboratory urgently due to his normal EF, stable troponin, and resolved chest pain by the time of admission.

However, within the first 48 hours after admission, the patient's pulmonary edema was resolved with diuretics and repeat ECG revealed resolution of LBBB with new deeply inverted T-waves in leads V2-3-4 (Figure #2). The patient was planning to be discharged until diagnosis for Wellens' syndrome was made and emergent transfer to the cardiac catheterization laboratory was initiated.

The patient underwent coronary angiography and was found to have single vessel coronary artery disease, reported as 99% focal stenosis in the mid portion of the LAD (Figure #3). The patient underwent a successful transluminal coronary angioplasty with stent deployment. Follow up ECG was performed 2 weeks after with unchanged deeply and symmetrically inverted T-waves in leads V2-3-4 (Figure #4). Repeat ECG performed 4 months after revealed normalized findings (Figure #5).

Figure 1

ECG Initial Presentation

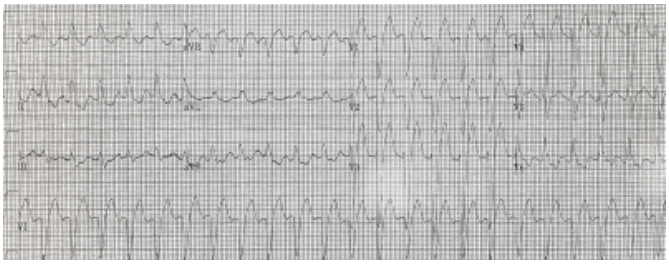


Figure 2

ECG 48 Hours After

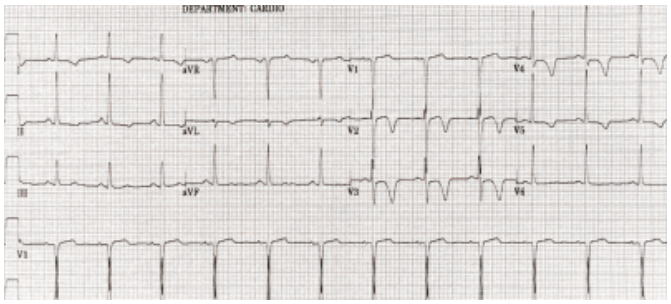


Figure 3a

Coronary Angiography with Stent Deployment

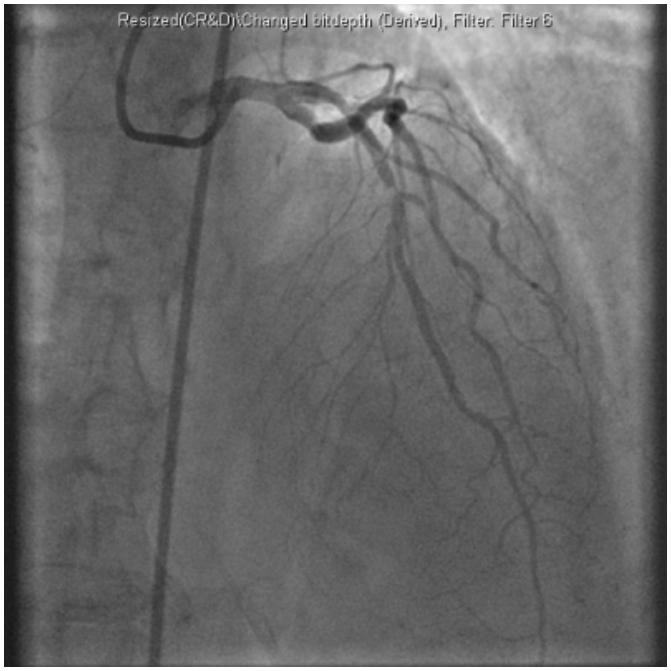
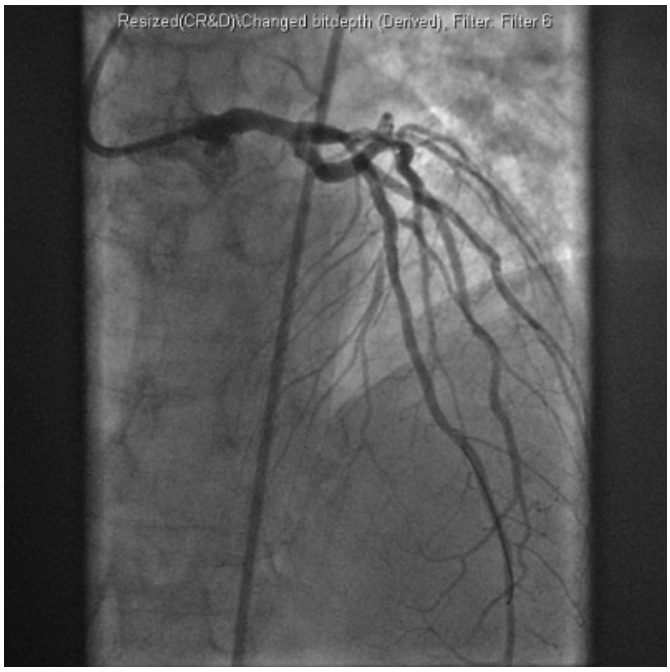


Figure 3b

Coronary Angiography with Stent Deployment



DISCUSSION

In 1982, Wellens published his observation that a pattern of inverted T-wave in the precordial leads was strongly associated with early large anterior MI and a poor prognosis in patients presenting with UA. He further demonstrated that most of these patients had significant disease of the proximal LAD, and that without coronary revascularization 75% went on to develop extensive anterior wall infarction within weeks, even with medical management.[1] Consequently, proper diagnosis and early cardiac catheterization with subsequent angioplasty or coronary bypass surgery is indicated for these patients.[2] Proper recognition of Wellens' syndrome is critical as exercise stress tests in these patients can lead to infarction at the time of increased cardiac demand and should be avoided.[3]

The key diagnostic features of Wellens' syndrome are the T-wave findings which can present in one of two patterns. Wellens' type A (also known as type II) has a biphasic pattern, as seen in lead V2 in Figure #2, and represents 25% of cases. Wellens' type B (also known as type I) is deeply inverted with symmetric contours, as seen in lead V3-4 in Figure #2, and is the more common variant representing 75% of cases seen. These ECG changes are usually seen in the midprecordial leads, but may appear in leads V1-V6. In addition to these ECG changes, other diagnostic criteria for Wellens' syndrome are as seen in Table #1.[3,4]

Table 1

Criteria for Wellens' Syndrome

Criteria for Wellens' syndrome
Wellens type A or type B wave in leads V2 and V3, occasionally in leads V1-V6
Recent history of angina
Pattern present during pain-free state
Isoelectric or minimally elevated (<1mm) ST segment
No precordial Q waves and preserved R wave progression
Normal or slightly elevated cardiac serum markers

In the described case, ECG performed during the chest pain phase showed new-onset LBBB. After treatment and symptomatic improvement of the patient's pulmonary edema, discharge planning was considered. Repeat ECG showed resolution of the LBBB, but also revealed new T-wave changes consistent with Wellens' syndrome. Coronary angiography was performed demonstrating a critical mid-LAD lesion and a stent was deployed. During outpatient follow-up, routine ECGs documented normalization of T-waves after a period of 4 months post-revascularization.

To our knowledge, there are limited reports of Wellens' syndrome presenting with new-onset LBBB. 5 In addition, there is no previously known duration of time until T-wave normalization after revascularization. We demonstrate here that T-wave abnormalities can persist for months even after revascularization. The mechanism for the delayed T-wave resolution is not exactly clear to us and may need further investigation. Overall, our case sheds light on a unique variant of Wellens' syndrome that could have been missed, leading to tragic outcomes.

CONCLUSION

The ECG pattern of Wellens' syndrome is well established in the medical community. Failure to recognize this syndrome when present can lead to deleterious outcomes –whether by inadequate therapy or inappropriate stress testing. Our case illustrates a unique variant of Wellens' syndrome with an initial ECG presentation of new-onset LBBB in the presence of pulmonary edema. If not diagnosed properly in this case, treatment would have been inadequate, and as symptoms resolved the patient may have been discharged only to have an adverse outcome at a later time. Additionally, we documented the persistence of T-wave abnormality for the duration of 4 months post-revascularization.

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