Acute Pulmonary Embolism Masquerading as Wellens' Syndrome

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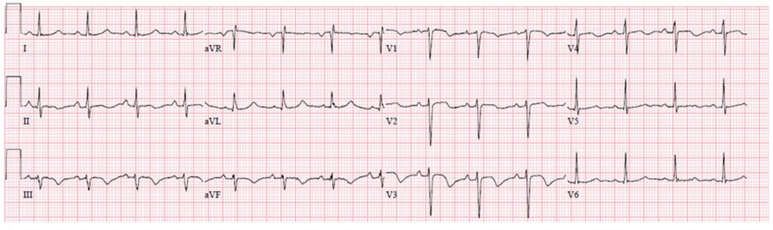
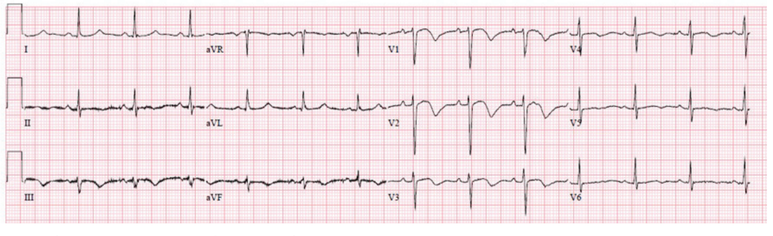
INTRODUCTION

- Severe acute pulmonary embolism (APE) often presents with T-wave inversions (TWI) in anterior precordial leads. However, this is also found in patients with acute coronary syndromes (ACS) and often reflects critical LAD stenosis.
- Symptoms of APE such as dyspnea and chest pain can be difficult to differentiate from ACS, and troponin levels are frequently elevated in patients with severe APE. As each condition has distinct diagnostic and therapeutic strategies, prompt differentiation is crucial.
- TWI in precordial leads are frequently considered a sign of myocardial ischemia, particularly Wellens' pattern which is thought to primarily reflect critical LAD stenosis. However, TWI in leads V1-V4 have been reported in up to 40% of patients with massive APE (1).
- We present a case of saddle APE with history and ECG findings suggestive of Wellens' Syndrome.

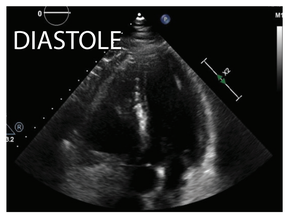
CASE REPORT

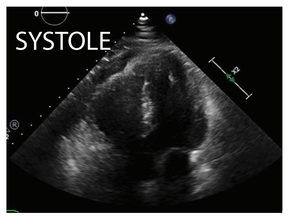
- 54-year-old man with a history of tobacco use presented with 15 days of intermittent anginal chest pain without dyspnea or pleuritic pain.
- T 36.5 HR: 97 BP: 100/78 SpO2: 96% on room air.
- Wells Score 0. PERC rule 1 (age > 50).
- ECG was notable for new TWI in anterior precordial and inferior leads, with the 2nd ECG showing biphasic T waves in anterior precordial leads consistent with Wellens' Type A pattern.

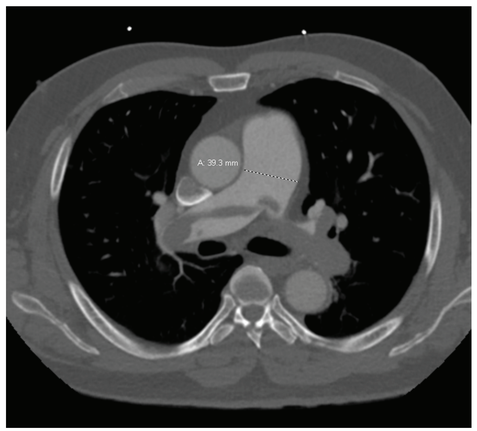




- Serial troponins were subsequently negative.
- The patient was admitted at that time for further evaluation of ischemic heart disease in the setting of TWIs raising concern for Wellens' Type A syndrome
- Subsequent echocardiography then demonstrated LVEF 55% with a dilated, hypokinetic RV and hyper-dynamic RV apex (McConnell's sign) and an estimated RVSP of 40-50 mmHg.







- Echocardiographic findings raised suspicion for APE.
- CT pulmonary angiography was subsequently obtained and revealed large saddle PE with extension into all 5 lobar pulmonary arteries.
- DVT studies of the extremities were negative.
- Heparin drip was initiated cautiously in setting of unexplained thrombocytopenia.
- CT abdomen/pelvis showed no evidence of malignancy.
- The patient remained stable throughout the admission and was discharged on warfarin once therapeutic level was achieved.

DISCUSSION

First described by Zwann et al in 1982 and further characterized by Rhinehardt et al in 2002 (2, 3), Wellens' pattern, in the context of anginal symptoms, is typically associated with critical proximal LAD occlusion and is an indication for early revascularization. In Type A, the anterior precordial leads (particularly V2-V4) demonstrate a biphasic pattern with an initial positive deflection followed by a terminal negative deflection. In Type B, these same leads show a deep, symmetrically inverted T wave (Figure 1). Some experts suggest that the T waves evolve from Type A to Type B. The pattern often occurs during a chest-pain free interval following an episode of typical chest pain, suggesting that Wellens' syndrome represents the spontaneous reperfusion of a complete occlusion in the proximal LAD (4) (Figure 2).

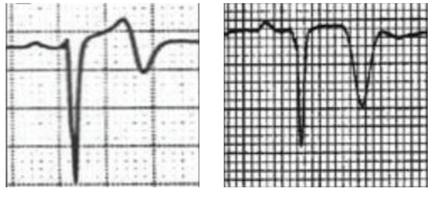


Figure 1. Wellens' Type A (left) and Wellens' Type B (right). Tracings from: Rhinehardt et al (2002).

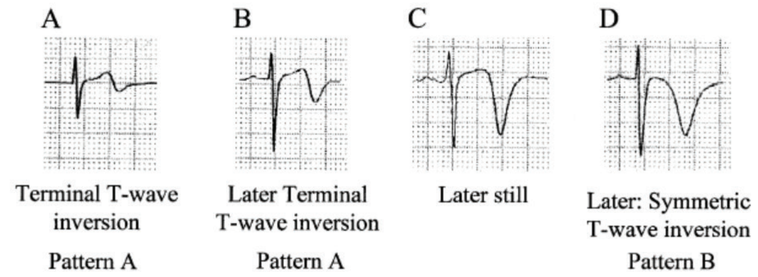


Figure 2. Evolution of T-wave inversion (A-D) after coronary reperfusion in STEMI reperfusion and in Wellens' syndrome (NSTEMI). Reprinted from: Smith et al (2002).

While the ECG on presentation was consistent with Wellens' Type A, there were subtle features suggestive of APE. Specifically, TWI in both III and V1 is frequently found in APE in conjunction with precordial TWI. Notably, peak negative T-wave was in V3 and not in V1-V2; had this been the case, diagnosis of APE would have been highly favored as the combination of these findings has near 100% specificity for APE rather than ACS in this scenario (5). The 2nd ECG did demonstrate both these findings, but also revealed a convincing Wellens' Type A pattern indicative of critical LAD stenosis. However, these ECGs did not follow the aforementioned evolution of Wellens' Syndrome which in retrospect likewise argues against ACS.

FUTURE DIRECTIONS

A 2011 multicenter study revealed 3% of patients with APE were initially admitted with diagnosis of ACS (1). ECG findings such as simultaneous TWI in anterior and inferior leads, while somewhat unusual (4-11% of APE) can aid the clinician at the point of care. Furthermore, given that ST-segment and T-wave changes often mark more threatening APE, more prospective studies on using this inexpensive and universally available modality to differentiate between acute life-threatening presentations will assist in hastening appropriate diagnosis and treatment. Future directions also include correlating ECG findings with findings on bedside echocardiography, which is often a more reliable differentiator of ACS and APE and can be obtained simultaneously to electrocardiographic data. Correlating simultaneous bedside ECG to bedside echocardiogram may reveal additional insight into differentiating ECG criteria for these emergent conditions.

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