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Case Report

Wellens Syndrome in a Female Patient: When NSTEMI Is Actually a STEMI Equivalent

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ABSTRACT

Background: Specific patterns in the precordial leads of an ECG are hallmarks of Wellens syndrome. Despite its association with imminent anterior myocardial infarction, the syndrome often presents with transient ECG changes, leading to under-recognition and delayed treatment. Female patients are underrepresented in reported cases, reflecting persistent sex-based variations in acute coronary syndrome diagnosis and management.

Case Presentation: A 52-year-old woman was admitted with escalating left-sided chest pain lasting over 20 minutes, following a week of intermittent exertional symptoms. Pretreatment ECG demonstrated biphasic T waves in leads V_1–V_4, consistent with Wellens Type A pattern. Five hours later, a repeat ECG revealed progression to deep, symmetric T-wave inversions in leads V_1–V_6, I, and aVL, consistent with Wellens Type B pattern and demonstrating dynamic ischemic evolution. Transthoracic echocardiography showed anterior wall hypokinesis with preserved left ventricular ejection fraction (51%). Coronary computed tomography angiography identified critical proximal left anterior descending artery (LAD) stenosis (99%), which was confirmed on invasive coronary angiography. Following the identification of Wellens syndrome, the patient received definitive treatment through successful percutaneous revascularization with overlapping drug-eluting stents, achieving TIMI grade III flow and complete revascularization.

Conclusions: This case highlights Wellens syndrome as a dynamic and unstable manifestation of critical LAD disease that should be recognized as a true STEMI equivalent. Serial ECG monitoring is essential for detecting rapid pattern evolution, and early invasive management is crucial to prevent extensive anterior myocardial infarction. Heightened clinical vigilance is particularly warranted in female patients to mitigate persistent sex-based disparities and improve outcomes. ***(Iranian Heart Journal 2026; 27(3): 81-89)***

KEYWORDS: Wellens syndrome; occlusion myocardial infarction (OMI); dynamic ECG evolution; female cardiovascular health; left anterior descending artery; reperfusion injury

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Acute ST-elevation myocardial infarction (STEMI) equivalents comprise a group of high-risk acute coronary syndromes caused by acute or critical coronary artery occlusion in the absence of classic ST-segment elevation on electrocardiography (ECG).[1] Despite not fulfilling traditional STEMI criteria, these entities carry comparable or higher risks of morbidity and mortality when recognition and reperfusion are delayed.[2, 3] Wellens syndrome is a classic STEMI equivalent characterized by a pathognomonic ECG pattern indicating critical stenosis of the proximal left anterior descending (LAD) coronary artery, which supplies a large myocardial territory and is associated with a high risk of extensive anterior MI.[4] The 2023 European Society of Cardiology (ESC) guidelines classify Wellens syndrome as a STEMI equivalent requiring immediate invasive management.[2]

Wellens syndrome presents a unique diagnostic challenge because its characteristic ECG abnormalities typically appear during pain-free intervals rather than during active ischemic symptoms.[5] This phenomenon reflects transient or intermittent LAD occlusion with spontaneous reperfusion, during which ischemic ECG changes become most pronounced. Two ECG patterns are recognized: type A, with biphasic T waves, and type B, with profound symmetric T-wave inversions in the anterior precordial leads. These patterns represent a continuum of evolving ischemia rather than distinct entities, and both confer equivalent prognostic significance. Without urgent revascularization, most patients progress to extensive anterior MI.[6]

From an epidemiologic perspective, Wellens syndrome is underrecognized and frequently underreported, particularly in the absence of persistent chest pain or substantial biomarker elevation at presentation.[1, 5] Published case series demonstrate a predominance of male patients, raising concern for underdiagnosis in women and other populations in whom atypical presentations may lower clinical suspicion. Misinterpretation of Wellens patterns as nonspecific T-wave abnormalities or "repolarization changes" can result in inappropriate conservative management or stress testing, potentially precipitating catastrophic outcomes.[4, 5]

We report a case of Wellens syndrome in a patient demonstrating dynamic ECG evolution from type A to type B within a short time interval, highlighting the progressive nature of ischemia in critical LAD disease. This case emphasizes the necessity of immediate recognition of Wellens syndrome, the value of serial ECG monitoring, and the importance of prompt invasive management to prevent extensive anterior MI. By illustrating the real-time evolution of ECG findings, this report reinforces awareness of Wellens syndrome as a true STEMI equivalent requiring immediate action.

CASE PRESENTATION

Clinical Presentation and Initial Assessment

A 52-year-old woman presented to the emergency department with classic anginal symptoms. She reported a 1-week history of intermittent chest pain, particularly during strenuous physical activity. The pain was described as a pressure-like sensation with radiation to the posterior chest wall. Episodes lasted approximately 2 to 3 minutes and resolved with rest. She denied diaphoresis but noted occasional nausea without vomiting. Her medical history was unremarkable for hypertension, diabetes mellitus, stroke, chronic kidney disease, or previously diagnosed cardiac disease,

although she had not undergone routine cardiovascular screening.

On physical examination, the patient appeared anxious and in moderate distress. Vital signs were blood pressure of 147/89 mm Hg, respiratory rate of 22 breaths/min, heart rate of 88 beats/min, and oxygen saturation of 97% on a nonrebreather mask at 10 L/min. She was fully alert and oriented, with a Glasgow Coma Scale score of 15 (E4V5M6). Pain intensity was rated as 9 of 10 on the visual analog scale, indicating severe pain. Findings from the general physical examination were normal.

ECG Findings and Dynamic Evolution

Initial ECG revealed biphasic T-wave morphology in leads V_2 and V_3, suggestive of a type A Wellens pattern. A follow-up 12-lead ECG performed 5 hours later during a pain-free interval showed progression to deeply inverted T waves in leads V_1–V_6 with extension to lateral leads I and aVL, representing a classic type B Wellens pattern.

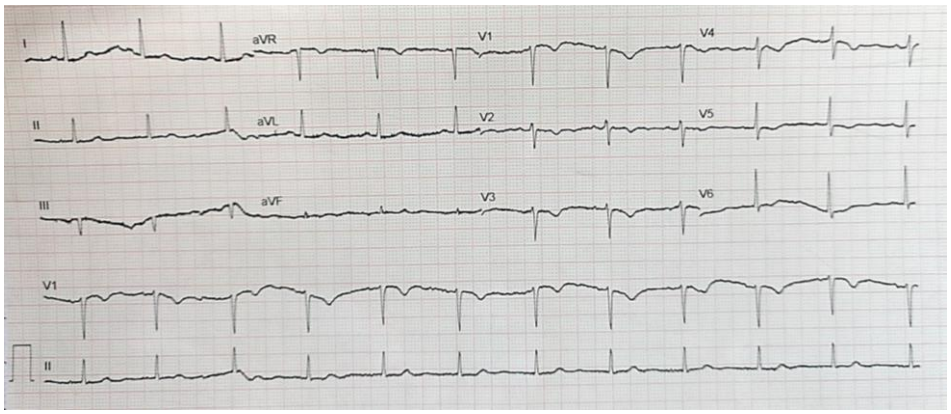

Figure 1. Twelve-lead ECG obtained at emergency department admission demonstrating biphasic T waves in precordial leads V_1–V_4, consistent with Wellens type A pattern

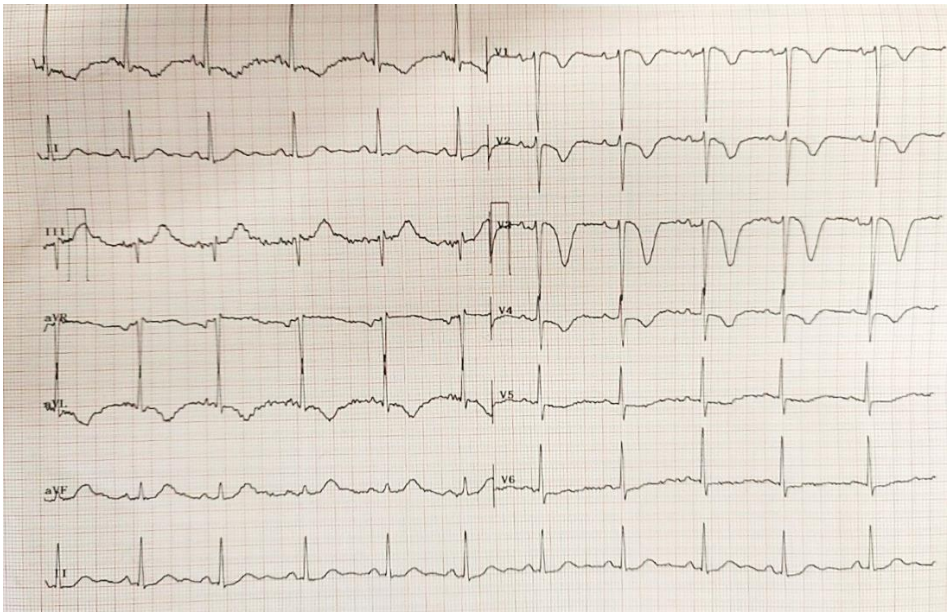

Figure 2. Twelve-lead ECG performed 5 hours later showing marked progression to deep T-wave inversions in leads V_1–V_6 and lateral leads, representing Wellens type B pattern

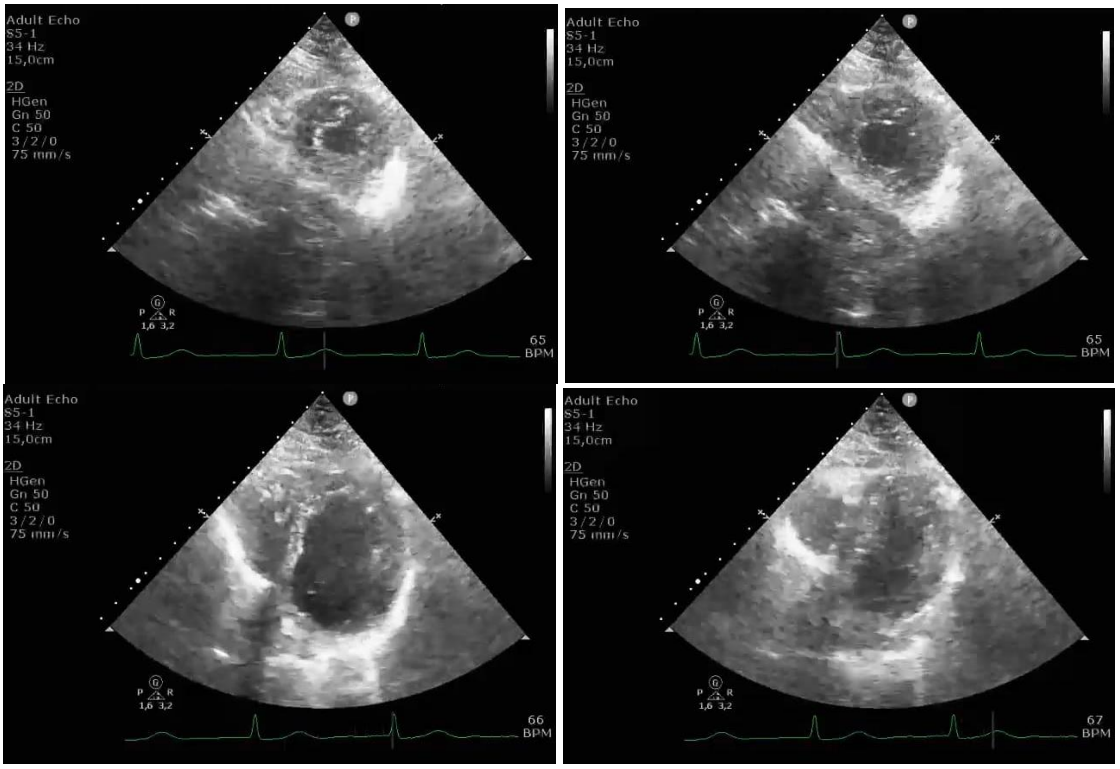

Figure 3. Echocardiographic examination

Echocardiographic Findings

Transthoracic echocardiography (TTE) performed on hospital referral demonstrated eccentric left ventricular (LV) hypertrophy with preserved global systolic function (LV ejection fraction, 51%) and mild anterior wall hypokinesis, consistent with LAD territory ischemia. Diastolic function was mildly impaired (grade I). Cardiac chamber sizes and valvular structures were normal, with no intracardiac thrombus or pericardial effusion. Hemodynamic assessment showed normal cardiac output and index with mildly elevated systemic vascular resistance. Overall findings were consistent with acute LAD-related coronary artery disease with regional dysfunction and preserved LV systolic function.

Multiview TTE assessment showed eccentric LV hypertrophy, mild anterior wall hypokinesis, and preserved global systolic function with an LV ejection fraction of 51%.

Laboratory, Imaging, and Initial Management Findings

Initial laboratory evaluation showed mild leukocytosis and hypokalemia, preserved renal function, and markedly elevated high-sensitivity troponin I levels consistent with acute myocardial injury. Chest radiography was unremarkable. Coronary computed tomography angiography demonstrated severe (>70%) mixed-plaque stenosis of the proximal-to-mid LAD artery with otherwise nonobstructive disease, compatible with high-grade single-vessel coronary disease and ECG features of Wellens syndrome. The patient was diagnosed with high-risk non–ST-elevation myocardial infarction (NSTEMI) and assessed as having low bleeding risk and moderate thrombotic risk. Guideline-directed medical therapy was initiated, including dual antiplatelet therapy, anticoagulation, high-intensity statin therapy, nitrates, electrolyte correction, and supportive care. Given the critical LAD anatomy and dynamic ischemic changes, the

patient underwent emergent transfer to a tertiary center for immediate coronary angiography and ad hoc percutaneous coronary intervention (PCI).

PCI Technical Details

PCI was performed via right radial access using a 6F guiding catheter. The proximal and mid-distal LAD lesions were sequentially predilated with compliant and semicompliant balloons, followed by deployment of 2 overlapping drug-eluting stents (everolimus-eluting distally and sirolimus-eluting proximally). High-pressure postdilatation with a noncompliant balloon ensured optimal stent expansion and apposition. Final angiography demonstrated Thrombolysis in Myocardial Infarction (TIMI) grade III flow with complete lesion coverage and no residual stenosis or procedural complications. Postprocedural risk assessment indicated low bleeding risk and moderate thrombotic risk, supporting continuation of dual antiplatelet therapy.

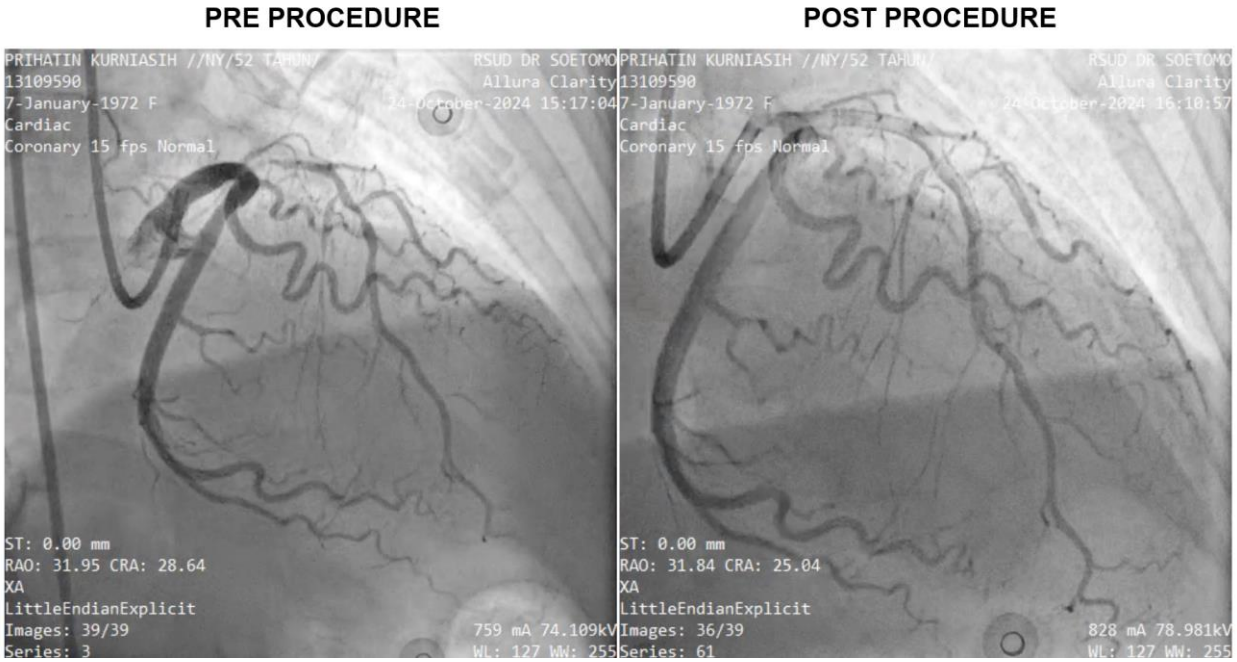

Figure 4. Coronary angiography and post–percutaneous coronary intervention results.

Preprocedure: Critical 99% stenosis in the proximal LAD with TIMI grade 2 flow before intervention.

Postprocedure: Post-PCI result demonstrating restoration of TIMI grade III flow after dual stent placement.

PCI: percutaneous coronary intervention; TIMI: Thrombolysis in Myocardial Infarction

Post-PCI Management and Clinical Course

Following successful PCI, the patient was observed in the intensive cardiac telemetry unit under continuous monitoring and remained hemodynamically stable. Guideline-directed medical therapy was initiated, including 12 months of dual antiplatelet therapy with ticagrelor and aspirin, along with high-intensity statin therapy, a beta-blocker, and an angiotensin II receptor blocker. The clinical course was uncomplicated, with no bleeding events, recurrent ischemia, or arrhythmias. Cardiac biomarkers showed appropriate downtrending, and follow-up ECG demonstrated resolution of anterior T-wave abnormalities. The patient was discharged on

day 4 in stable condition with preserved LV function, a cardiac rehabilitation referral, and planned outpatient cardiology follow-up.

DISCUSSION

This case illustrates a high-risk presentation of Wellens syndrome characterized by dynamic ECG evolution, critical proximal LAD artery stenosis, and successful prevention of extensive anterior MI through early recognition and urgent revascularization.[6, 7] The hallmark finding was a real-time transition between type A and type B T-wave patterns within a 5-hour interval, a clinical phenomenon that is seldom captured in the medical literature but remains consistent with prior clinical observations and ECG-based descriptions of Wellens syndrome.[3, 8] This temporal ECG evolution over a short timeframe reflects the dynamic continuum of evolving myocardial ischemia and underscores the critical importance of serial ECG monitoring in patients with suspected Wellens syndrome.

The transition between type A and type B patterns represents progressive worsening of myocardial ischemia secondary to critical LAD stenosis rather than discrete, unrelated entities, and both morphologic patterns carry similarly grave prognostic implications necessitating emergent coronary intervention.[9]

The observed ECG progression reflects a continuum of ischemia-reperfusion injury rather than distinct disease entities. Foundational and contemporary studies describe how critical LAD stenosis with transient occlusion and spontaneous reperfusion initiated calcium overload, oxidative stress, mitochondrial dysfunction, and myocardial edema, resulting in altered ventricular repolarization during pain-free intervals.[10] Biphasic T waves represent early electrical heterogeneity during reperfusion, whereas progressive myocardial edema and transmural repolarization gradients give rise to deep, symmetric T-wave inversions over time.[1, 3] The rapid type A-to-type B evolution observed in this patient supports this mechanistic model and emphasizes that worsening ECG abnormalities during symptom resolution should not be misinterpreted as clinical improvement.[6, 11]

This case exemplifies the limitations of the traditional STEMI/NSTEMI framework. Despite the absence of ST-segment elevation, coronary imaging demonstrated a near-occlusive (99%) proximal LAD lesion with impaired flow, physiologically equivalent to acute coronary occlusion.[12, 13] Emerging literature increasingly supports the occlusion myocardial infarction (OMI) paradigm, which prioritizes coronary anatomy and pathophysiologic mechanisms over ECG criteria alone.[14] Within this framework, Wellens syndrome is recognized as an OMI equivalent requiring immediate invasive management regardless of troponin elevation or ST-segment changes, a position explicitly endorsed by the 2023 ESC Guidelines.[2] Application of this paradigm in the present case justified urgent angiography and PCI, preserving LV function and preventing irreversible myocardial injury.[6, 15]

The dynamic nature of Wellens syndrome further reinforces the necessity for serial ECG evaluation. Prior reports demonstrate that ECG abnormalities may be transient or rapidly evolving, and reliance on a single ECG may lead to missed or delayed diagnosis.[16] In this case, serial ECGs revealed rapid morphological progression that directly informed escalation of care. Importantly, cardiac biomarkers may remain within normal limits or exhibit only minimal elevation early in the disease course and should not delay invasive evaluation when characteristic ECG features are present.[17]

Multimodal diagnostic integration played a pivotal role in risk stratification. TTE demonstrated LAD-territory ischemia through regional wall motion abnormalities

while preserving global systolic function-findings known to correlate with infarct risk and myocardial salvage potential.[14, 18] Coronary computed tomography angiography enabled rapid confirmation of critical LAD stenosis and exclusion of significant nonculprit disease, aligning with guideline-supported use of coronary computed tomography angiography for anatomical clarification in selected acute coronary syndrome presentations.[2] Nevertheless, in confirmed or strongly suspected Wellens syndrome, adjunctive imaging should complement-but never delay-definitive invasive coronary angiography.[19]

This case also underscores persistent sex-based disparities in acute coronary syndrome care. Large registries and consensus statements consistently demonstrate that women experience longer diagnostic delays, lower rates of invasive management, and higher adjusted mortality compared with men.[8, 13] Sex-based variations in clinical presentation frequently result in women exhibiting nontraditional manifestations of acute coronary ischemia, thereby increasing the risk of underrecognition of high-risk entities such as Wellens syndrome.[16, 17] The relative underrepresentation of women in published Wellens syndrome case series likely reflects underdiagnosis rather than true epidemiologic rarity.[1] The favorable outcome observed in this female patient underscores the need for equal diagnostic vigilance and equivalent urgency in management irrespective of sex.[12]

From a clinical practice perspective, several key principles are reinforced. Anterior biphasic or deeply inverted T waves observed during pain-free intervals should immediately raise suspicion for Wellens syndrome.[6, 20] Serial ECG monitoring is essential when initial findings are subtle or evolving. Exercise or pharmacologic stress testing is contraindicated because of the risk of precipitating complete LAD occlusion, malignant arrhythmias, and sudden cardiac death.[19] Clinicians should adopt an anatomy- and physiology-driven approach consistent with the OMI paradigm, recognizing that the absence of ST-segment elevation does not equate to lower risk.[18, 19]

Ultimately, urgent coronary angiography and revascularization remain the only definitive life-saving interventions, as conservative medical therapy alone is associated with progression to extensive anterior MI and poor clinical outcomes.[1, 6]

In conclusion, this case demonstrates Wellens syndrome as a dynamic and unstable manifestation of critical LAD disease best understood within the OMI framework. Early recognition, meticulous serial ECG assessment, and immediate invasive management are essential to preventing catastrophic outcomes—particularly in populations at heightened risk for underdiagnosis, such as women.[2]

CONCLUSIONS

This case underscores Wellens syndrome as a dynamic and highly unstable manifestation of critical coronary artery disease that must be recognized as a true STEMI equivalent requiring immediate invasive management, irrespective of ST-segment elevation or initial biomarker levels. The documented rapid progression of T waves from type A to type B pattern within 5 hours highlights the inherent instability of this syndrome and reinforces the necessity of serial ECG monitoring. Framing Wellens syndrome within the OMI paradigm provides a more physiologically accurate approach to risk stratification and clinical decision-making. Furthermore, this case draws attention to persistent sex-based disparities in acute coronary care, emphasizing the need for equal diagnostic vigilance and timely intervention in women presenting with

suggestive symptoms or ECG abnormalities. Ultimately, prompt coronary angiography and revascularization remain the only definitive, life-saving therapy capable of preventing extensive anterior MI and preserving long-term ventricular function. Early integration of clinical suspicion, ECG expertise, multimodality imaging, and guideline-directed intervention is essential to achieving optimal clinical outcomes in patients with Wellens syndrome.

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