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Case Report

Wellens Syndrome Following Transient ST-Elevation Myocardial Infarction: A Clinical Dilemma of "Treat Now or Later?"

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ABSTRACT

Introduction: Transient ST-segment elevation myocardial infarction (TSTEMI) is often associated with better prognosis due to spontaneous reperfusion, yet its progression to Wellens syndrome indicates severe left anterior descending (LAD) occlusion and raises the risk of anterior infarction.

Case Presentation: We describe a 61-year-old woman with the initial presentation of typical chest pain (numeric rating scale [NRS]: 6/10) and ST-segment elevation at the extensive anterior leads at the referring hospital. The patient was given dual antiplatelet treatment and referred to our hospital. Upon arrival, her symptoms resolved (NRS: 0/10), and her ST-segment elevation was evolved to biphasic T waves in leads V_2-V_6 along with ST elevation-T-wave inversion in leads I and aVL, similar to Wellens syndrome. Cardiac biomarkers were markedly elevated, and echocardiography revealed hypokinetic wall motion abnormalities. Coronary angiography revealed proximal LAD occlusion, a feature of Wellens syndrome. Despite the potential for extensive myocardial damage, the patient was managed under a delayed invasive strategy. She remained stable after the intervention and at 3 months' follow-up at the outpatient clinic.

Conclusions: Conversion of TSTEMI to Wellens syndrome raises clinical dilemma regarding invasive strategy decision-making. TSTEMI occurs due to spontaneous resolution, which can be partial or total, yet the risk of spontaneous reocclusion is also inevitable. TSTEMI requires an early invasive strategy (< 24 h) with close monitoring of new or worsening ischemic signs; however, the urgency may be individualized on a case-by-case basis. ***(Iranian Heart Journal 2026; 27(2): 87-93)***

KEYWORDS: transient ST-changes; door-to-wire time; percutaneous coronary intervention; thrombus; case report

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Transient ST-segment elevation myocardial infarction (TSTEMI) refers to a subset of acute coronary syndrome (ACS) in which patients present with chest pain and ST-segment elevation but subsequently experience rapid symptom relief and greater than 50% resolution of ST elevation on serial electrocardiograms (ECGs) prior to any reperfusion therapy. This spontaneous improvement is often correlated with angiographic evidence of restored coronary flow (Thrombolysis in Myocardial

Infarction [TIMI] flow grade 2–3), suggesting spontaneous reperfusion. Although relatively uncommon, TSTEMI occurs in approximately 4% to 24% of ACS cases, with a prevalence of about 15% among patients initially classified as STEMI.[1, 2] The current guideline suggests that TSTEMI is similar to high-risk NSTEACS, yet the risk of spontaneous reocclusion after spontaneous reperfusion raises a clinical dilemma.

Wellens syndrome is a distinct ACS presentation characterized by T-wave abnormalities either biphasic or symmetrically inverted T waves in leads V_2-V_3. The Wellens pattern is strongly indicative of moderate-to-severe occlusion (50%-99%) at the proximal left anterior descending (LAD) artery. Previously, Wellens syndrome was considered a STEMI equivalent that warranted urgent intervention.[3] Nonetheless, the recent European Society of Cardiology (ESC) guideline in 2023 categorized Wellens syndrome to high-risk NSTE-ACS, requiring intervention under 24 hours.[4] This case report highlights the clinical scenario posed by the occurrence of Wellens syndrome following TSTEMI.

Case Presentation

A 61-year-old woman was referred to our hospital with a chief complaint of chest pain and a diagnosis of extensive anterior STEMI. The chest pain began 8 hours prior to admission to our hospital and described as crushing sensation and penetrating to the back. The initial pain intensity was 6/10 on the numeric rating scale (NRS) and reduced to 0/10 at our hospital. The patient was also nauseous. She had a history of uncontrolled hypertension with amlodipine (5 mg OD). Any history of smoking, previous cardiovascular diseases, diabetes, stroke, kidney disease, or autoimmune disease was ruled out by the patient. Previous treatment from the referring hospital was aspirin (300 mg), ticagrelor (180 mg), isosorbide dinitrate (5 mg sublingual), intravenous ranitidine, and ketorolac.

Physical examination was unremarkable. The patient's vital signs were stable with blood pressure of 130/69 mm Hg, heart rate of 87 bpm, respiratory rate of 20 breaths/min, and body temperature of 37.2 °C. Body mass index was normal (20.81 kg/m^2). Jugular venous pressure was not elevated. No abnormality was found during heart and lung auscultation, nor was there any evidence of congestion or hypoperfusion on peripheral extremity examination.

The initial ECG from the referring hospital showed significant ST elevation in leads V_2-V_6, I, and aVL with reciprocal changes in the inferior leads (Figure 1A). Nevertheless, repeated ECG at our hospital only showed biphasic T waves in leads V_2-V_6, along with ST-elevation-T-wave inversion in leads I and aVL. No ST elevation was noted in any precordial leads, and there was a reduction of 60% in ST elevation in leads I and aVL (Figure 1B). Laboratory findings were within the normal limits, except for elevated serum potassium (5.6) and elevated high-sensitivity troponin I (> 50,000). The patient's chest radiogram was normal. Transthoracic echocardiography revealed hypokinesia at the basal to apical anterior and basal to middle anterolateral portion with a left ventricular ejection fraction of 45.9%. Serial ECG demonstrated no significant changes from the last ECG, and the patient denied any chest pain. Hence, we decided to defer the immediate intervention because of the significant resolution of chest pain and ST elevation The patient was diagnosed as TSTEMI, but considered high-risk NSTEMI owing to Wellens pattern appearance on the latest ECG. She was admitted to the intensive coronary care unit and received atorvastatin (40 mg OD), bisoprolol (2.5 mg OD), and ramipril (2.5 mg OD) after potassium correction with an oral potassium-lowering agent.

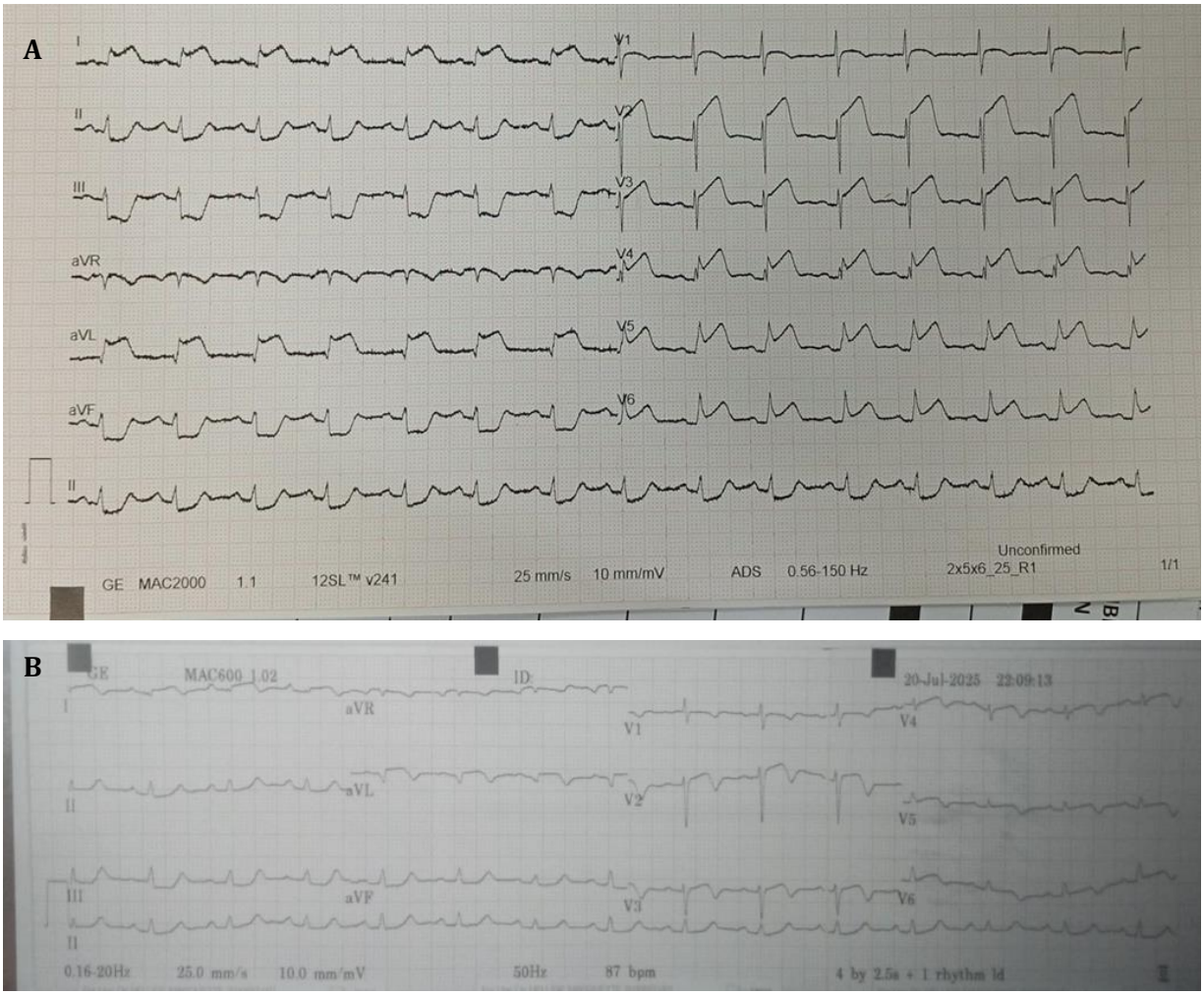

Figure 1. (A) Initial electrocardiograms (ECG) from the referring hospital show ST elevation in leads V_2-V_6, I, and aVL. (B) Admission ECG at our hospital shows resolution of the ST elevation.

Coronary angiography was performed 11 hours later. It showed significant occlusion (90%) at the proximal to middle LAD (Figure 2A) with insignificant stenosis at the osteal (50%) and distal (40%) of the left circumflex coronary artery. While angiography of the right coronary artery (RCA) revealed ectasia from the proximal to distal RCA with significant stenosis at the middle RCA (70%) and insignificant stenosis at the ostial RCA (60%). An ad-hoc percutaneous coronary intervention with a sirolimus stent (2.75 × 23 mm) was performed on the proximal to middle LAD and showed TIMI grade 3 flow (Figure 2B). During observation, the patient exhibited no symptoms, yet serial ECG after intervention showed a consistent biphasic T wave in leads V_2-V_6, I, and aVL without any pathological Q waves (Figure 3). The patient remained stable and was asymptomatic at 3 months' outpatient follow-up.

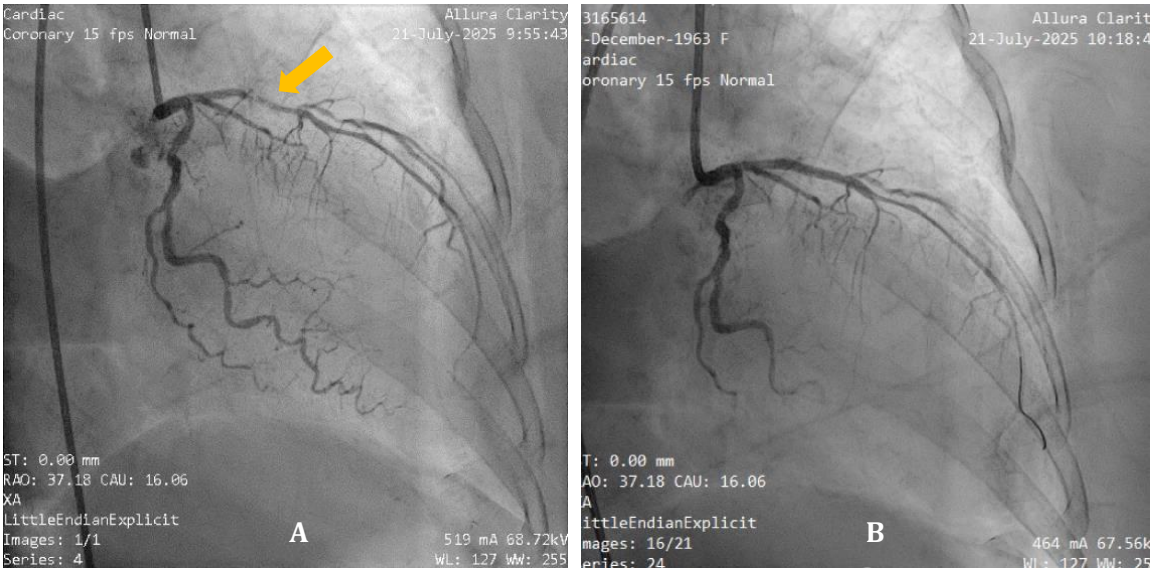

Figure 2. Coronary angiography pre- (A) and post-intervention (B). The arrow shows significant occlusion at the proximal-middle left anterior descending, consistent with the electrocardiographic appearance of anterior extensive ST elevation at the referring hospital that converted to Wellens type A.

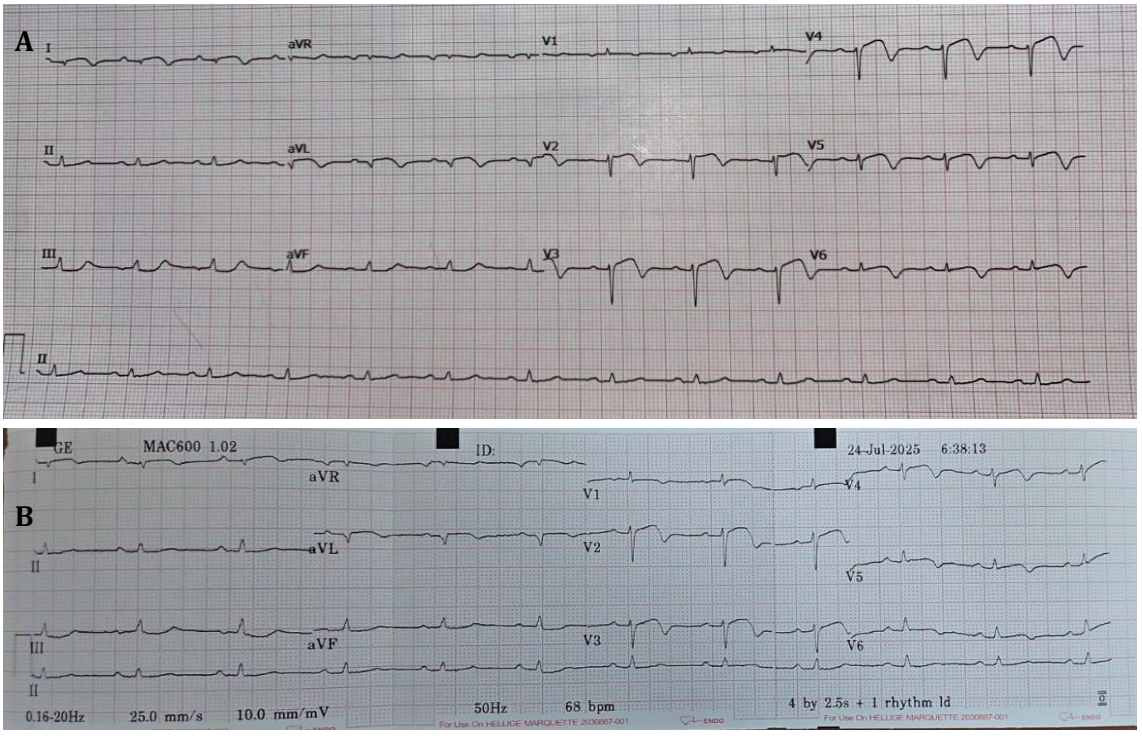

Figure 3. The images illustrate serial electrocardiograms (A) 24 hours and (B) 48 hours following intervention.

DISCUSSION

The primary mechanism underlying TSTEMI is spontaneous reperfusion of a previously occluded epicardial coronary artery.[1] In this condition, coronary blood flow is restored prior to the initiation of reperfusion therapy. The abrupt restoration of blood flow typically results in rapid resolution of ST-segment elevation and

improvement of ischemic chest pain, which is similar to the current case.

Spontaneous reperfusion reflects a dynamic interaction between the thrombotic process and endogenous fibrinolytic activity in the coronary circulation. In TSTEMI, the fibrinolytic activity predominates and leads to enzymatic degradation of fibrin.[5] High shear-stress in a coronary artery with severe luminal occlusion may induce mechanical dislodgement of the thrombus from the vessel wall, facilitating distal embolization, accelerating fibrinolysis, and relieving the occlusion partially or even totally. In this case, the resolution was considered partial rather than total. Despite improving clinical condition along with significant reduction of ST-segment elevation, coronary angiography revealed a severe occlusion at the proximal-to-mid segment of the LAD. In addition, the initial presentation of ST-elevation was theoretically caused by a total occlusion. Other contributing mechanisms include coronary vasospasm and plaque erosion, producing transient thrombotic occlusion.[2]

Wellens syndrome is characterized by distinctive T-wave abnormalities in V_2-V_3 precordial leads, manifesting as either biphasic waves (Type A) or deeply inverted waves (Type B). Wellens syndrome is typically associated with critical occlusion in the proximal LAD, consistent with our case.[3]

A previous study showed that two-thirds of patients with the Wellens pattern had an LAD culprit, and one-third had proximal LAD culprit. Sensitivity and specificity of the Wellens sign to predict the LAD culprit were 24.6% and 96.2%, respectively.[6] The dynamic ECG changes in Wellens syndrome are thought to result from cycles of reperfusion and reocclusion, with the characteristic T-wave pattern often appearing after spontaneous or pharmacologically induced partial reperfusion of the ischemic myocardium. This reperfusion is frequently transient, and the underlying critical LAD occlusion confers a high risk of extensive anterior wall MI.[3]

More often, intrinsic fibrinolytic activity can result in spontaneous resolution, leading to normal ECG and not the Wellens pattern, as was the case in our patient.[2] Patients with TSTEMI that convert to normal ECG demonstrate a more favorable prognosis than those with persistent STEMI or NSTEMI, largely because early spontaneous reperfusion limits myocardial injury. Patients with TSTEMI show smaller intracoronary thrombus lesions and lower thrombus burdens, along with smaller left ventricular infarcts and preserved left ventricular function.[7] The mortality rate was also significantly lower in TSTEMI than in NSTEMI and STEMI. TSTEMI showed 0% in-hospital mortality (vs 2.3% in NSTEMI and 4.2% in STEMI) and 4.8% long-term mortality over 36 months (vs 14.7% in NSTEMI and 14.2% in STEMI), corresponding to hazard ratios of 0.33 and 0.27, respectively.[8] Collectively, these findings underscore how TSTEMI's rapid reperfusion translates into limited ischemic burdens and improved outcomes.

The previous 2018 ESC guideline on myocardial revascularization provided no specific recommendations on the optimal timing of revascularization in patients with TSTEMI. This uncertainty raises an important clinical gap whether patients with TSTEMI should pursue an immediate invasive strategy similar to persistent STEMI or a delayed approach strategy similar to high-risk NSTE-ACS.[9] The TRANSIENT Trial directly addressed this issue by comparing 142 TSTEMI assigned to either immediate intervention (median: 0.3 h) or delayed intervention (median: 22.7 h). Surprisingly, both strategies led to equally infarct size percentages on day 4 (1.3% vs

1.5%; *P* = 0.48) and at 1 year (0.4% vs 0.4%; *P* = 0.79). Left ventricular function was also preserved for both immediate and delayed strategies on day 4 (57.5% vs 58.0%; *P* = 0.66) and at 4 months' follow-up (59.9% vs 59.3%; *P* = 0.63). The incidence of 30-day (2.9% vs 2.8%; *P* = 1.00) and 1-year (4.4% vs 5.7%; *P* = 1.00) major adverse cardiovascular events was also comparable in both strategies. Although a small subset of patients (5.6%) in the delayed arm required urgent intervention because of reinfarction symptoms, this condition was not associated with larger infarcts or worse clinical outcomes.[2,10] Collectively, these findings suggest that the early invasive strategy was not inferior to the immediate strategy, adopted in the recent ESC 2023 guideline with close monitoring for ischemic signs.[4]

While the TRANSIENT trial demonstrated the non-inferiority of the delayed approach, the current case highlights a clinical dilemma in managing TSTEMI that converted to Wellens syndrome probably due to spontaneous (partial) resolution. In this case, the patient was categorized as high-risk NSTEMI, and early revascularization (< 24 h) was warranted to achieve favorable outcome. Our cases also emphasizes that spontaneous resolution of chest pain and ECG should not delay coronary angiography for more than 24 hours. In addition, close monitoring of ischemic signs (eg, chest pain, hemodynamic deterioration, and higher ST elevation) is paramount, as its presence is the indication of the immediate invasive strategy in this context. Finally, identifying the urgency of the invasive strategy in ACS is important to optimize patient outcomes.

CONCLUSIONS

Conversion of TSTEMI to Wellens syndrome raises a clinical dilemma regarding the choice of the invasive strategy. TSTEMI occurs due to spontaneous resolution, which can be partial or total, yet the risk of spontaneous reocclusion is also inevitable. TSTEMI requires the early invasive strategy (< 24 h) with close monitoring of new or worsening ischemic signs. However, urgency may be individualized on a case-by-case basis.

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Consent for Publication
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Ethics Approval and Consent to Participate
Not applicable

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