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THE CALM BEFORE THE STORM: WELLENS SYNDROME REVISITED

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ABSTRACT
Wellens syndrome is a characteristic electrocardiographic pattern indicating a critical stenosis of the left anterior descending (LAD) coronary artery and is a warning sign for an impending anterior myocardial infarction. Early recognition is crucial, as this pattern typically appears when the patient is pain-free and cardiac biomarkers are standard, which can potentially mislead clinicians. This case report aims to highlight the importance of recognizing the pattern of Wellens Syndrome through the clinical description of patient Mr. A, a 61-year-old male with hypertension and chronic kidney disease who presented with chest pain that has subsided. We present a 61-year-old Melanesian man with a history of hypertension and stage 3 chronic kidney disease, who had recent anginal chest pain that resolved upon evaluation. On examination, blood pressure was 154/80 mmHg, heart rate 78/min, respiratory rate 20/min, temperature 36.7°C, oxygen saturation 99% on room air, and minimal pitting oedema in both legs. The ECG obtained showed biphasic T waves in leads V2–V4 with the patient feels no chest pain at all, consistent with a type A Wellens pattern. Troponin T was negative (< 40 ng/L), and no ST elevation or pathologic Q waves were observed. Chest X-ray showed cardiomegaly. Coronary angiography revealed a critical stenosis in the proximal LAD. The patient underwent percutaneous coronary intervention with stent placement to the proximal LAD. Wellens syndrome represents a "pre-infarction" state that should be identified to prompt urgent revascularization before extensive myocardial infarction occurs. This case underscores the importance of recognizing Wellens syndrome as "the calm before the storm" and avoiding conservative management that could lead to adverse outcomes.

Keywords: coronary angiography; electrocardiographic; wellens syndrome

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INTRODUCTION

Wellens syndrome is a collection of clinical symptoms and specific ECG findings that indicate the presence of critical stenosis in the proximal left anterior descending (LAD) coronary artery, generally in patients with a history of recent angina that has subsided. The characteristic ECG pattern of Wellens syndrome consists of biphasic T wave or deep, symmetrical, inverted T waves in precordial leads (especially V_2–V_3) during the pain-free phase. This pattern is particularly significant for the stenosis of the proximal LAD (Zhang & Shen, 2025). Patients with Wellens syndrome typically present without active chest pain and with normal or only slightly elevated cardiac enzyme levels (Zhou et al., 2022). Therefore, this syndrome is easily missed if clinicians are not vigilant.

Risk factors for Wellens syndrome align with those for coronary artery disease in general, including hypertension, diabetes mellitus, dyslipidemia, smoking, obesity, and chronic kidney disease. Patients with Wellens syndrome typically have a history of new-onset or worsening angina within days or weeks before presentation (unstable angina), which then resolves. The exact incidence of Wellens syndrome varies. A 2024 cross-sectional study in Sudan reported a

prevalence of the Wellens pattern of approximately 15% among patients with acute coronary syndrome. Another study in China found an incidence of approximately 5.7% in ACS patients, with most Wellens cases ultimately diagnosed as mild NSTEMI (without significant ST elevation) due to high troponin sensitivity levels (Mohamed & Abdelaziz, 2024). Two forms of the Wellens ECG pattern have been identified: Type A and Type B. Type A is characterized by a biphasic T wave (two phases: positive then negative) primarily in leads V_2–V_3, while Type B shows a deep and symmetrical negative T wave in V_2–V_3 (may extend to V_4).[7] Type B is the more common form (approximately 75% of cases) compared to Type A (Mathew et al., 2022; Mohamed & Abdelaziz, 2024)

Historically, Wellens and colleagues first reported this ECG pattern in 1982 in 18% of patients with unstable angina; 75% of these patients developed anterior myocardial infarction within weeks despite optimal conservative therapy. This suggests that Wellens syndrome is a highly predictive sign of myocardial infarction. Early detection of the Wellens pattern and aggressive coronary intervention have been shown to reduce the risk of extensive infarction and mortality.[4] Recent studies indicate that with early recognition and rapid invasive strategies, the long-term prognosis of Wellens patients can improve and is not significantly different from other NSTEMI patients (Abdeldayem et al., 2025). Therefore, current management of Wellens syndrome emphasizes the need for immediate reperfusion via angiography and PCI before total coronary artery occlusion occurs. This case report aims to highlight the importance of recognizing the pattern of Wellens Syndrome through the clinical description of patient Mr. A, a 61-year-old male with hypertension and chronic kidney disease who presented with chest pain that has subsided.

METHOD

This study is prepared in the form of a case report with a narrative descriptive approach. The main objective of this report is to systematically and in-depth describe the clinical presentation, ECG interpretation, diagnostic support results, interventions, and clinical outcomes in a patient with Wellens Syndrome. This case report aims to provide clinical insight and education on the importance of early recognition of this syndrome in order to prevent extensive acute myocardial infarction. This case report is prepared based on the management of patients treated in the Emergency Unit, catheterisation laboratory (cath lab) and coronary intensive care unit of UKI General Hospital, Jakarta, May 2025. The patient who is the subject of the report is a 61-year-old Melanesian man, with a history of hypertension and chronic kidney disease (CKD) stage 3. The patient came with complaints of anginal chest pain that had subsided during clinical evaluation. Case selection was carried out purposively based on clinical and electrocardiographic findings that were consistent with Wellens Syndrome type A, and confirmed by coronary angiography.

Data were collected through review of patient medical records, structured interviews with patients and families, and documentation of supporting examination results such as: 1) Electrocardiography (ECG); 2) Cardiac biomarkers (Troponin, CK-MB); 3) Coronary angiography as confirmation of diagnosis; 4) Records of drug therapy and/or interventions given during treatment. The analysis was conducted descriptively by compiling a case chronology and interpreting clinical and diagnostic data based on the latest literature on Wellens Syndrome. Interpretation of the results was compared with the American College of Cardiology (ACC)/American Heart Association (AHA) guidelines regarding the management of acute coronary syndrome without ST elevation (NSTEMI) and the latest publications on Wellens Syndrome. The data are presented in the form of a chronological narrative, accompanied by ECG images and coronary angiography results. The discussion is intended to highlight the importance of recognizing typical ECG patterns, the risk of massive anterior myocardial infarction if not intervened early, and the appropriate diagnostic and therapeutic approach.

CASE REPORT

A 61-year-old Melanesian man, came to the cardiac clinic with typical chest pain as the chief complaint. He complained of sudden chest pain that had started seven days before he was came to the cardiac clinic. The pain was felt in the retrosternal area, radiating to the left arm, with severe intensity (pain scale 7/10) that occurred during activity and improved with rest. Cold sweats accompanied the complaint without nausea or shortness of breath. Each pain episode lasted approximately 10 minutes and happened twice in the past 48 hours. The patient then taken to the emergency room (ER) for initial treatment and supporting examination. The patient arrived at the ER in a pain-free state. Other complaints, such as fever, cough, runny nose, and history of trauma, were denied by the patient. A history of coronary artery disease, hypertension, and diabetes mellitus was denied. The patient had a risk factor of heavy smoking (1 pack per day). There was no family history of coronary artery disease. On physical examination, the patient appeared moderately ill, alert and oriented. Blood pressure was 154/86 mmHg, pulse rate was 74 beats per minute, regular, respiratory rate was 20 breaths per minute, temperature was 36.7°C, and oxygen saturation was 99% room air. The conjunctiva was not anemic, and the sclera were not icteric. Cardiac examination: S1 and S2 sounds were single and regular, with no murmurs or gallops. No jugular vein distension was noted. The lungs were symmetrical, with vesicular breath sounds and no rales or wheezing. The abdomen was unremarkable, with no palpable hepatosplenomegaly. The extremities were warm, with minimal pitting oedema visible in the bilateral lower extremities. Laboratory results show standard quantitative troponin T (<40 ng/dL), supporting the diagnosis of Wellens syndrome. Hemoglobin level is 12.7 g/dL, hematocrit 39.4%. Urea level was 56 mg/dL, and creatinine 2.14 mg/dL. The estimated glomerular filtration rate (eGFR) was 34 mL/min/1.73 m² (stage 3b CKD).

Table 1.
Laboratory Results

Examination	Results
Haemoglobin	12,7 g/dL
Hematocrit	38,4%
Platelet	301.000/uL
Leukocyte	9.3 rb/uL
Troponin T	<40 ng/dL
Urea	56 mg/dL
Creatinine	2,14 mg/dL
eGFR	34 ml/minute/1,73m2
Sodium	139 mmol/L
Potassium	3,7 mmol/L
Chloride	110 mmol/L

The ECG (Figure 1) shows a sinus rhythm of 72 bpm with biphasic T waves in leads V2–V4. This ECG pattern is consistent with Wellens syndrome type A. The anteroposterior chest X-ray (Figure 2) reveals an enlarged heart (CTR > 50%), consistent with cardiomegaly.

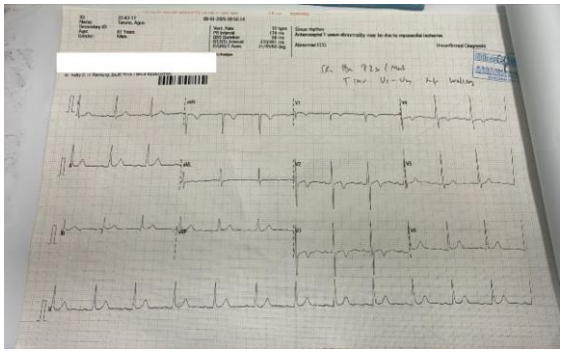

Figure 1. Electrocardiogram

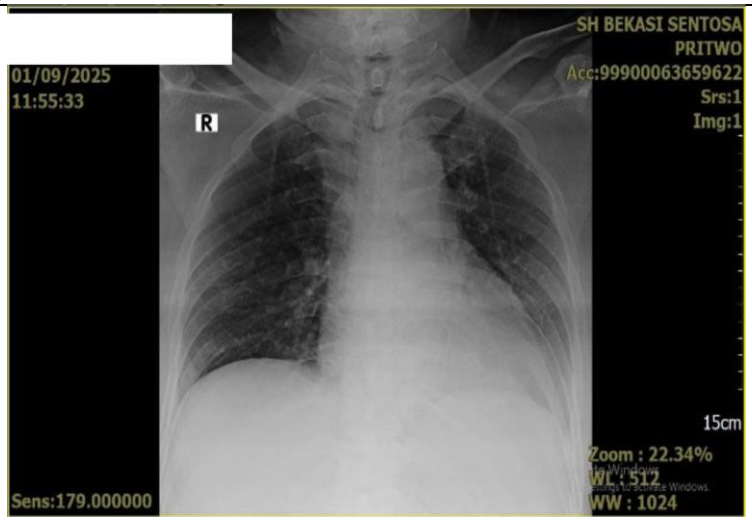

Figure 2. Chest X-ray

After initial treatment in the ED (dual antiplatelet loading, anti-ischemic and statin), the patient was taken to the cath lab for urgent invasive evaluation. Coronary angiography confirmed a critical stenosis in the proximal segment of the LAD (Figure 3). Given the high-risk lesion, the decision was made to proceed with PCI procedure. A drug-eluting stent was successfully deployed in the proximal LAD with TIMI flow 3, effectively treating the lesion (Figure 4). The patient was started on optimal medical therapy (dual antiplatelet therapy, beta-blocker, statin, and ACE inhibitor) and had an uneventful recovery.

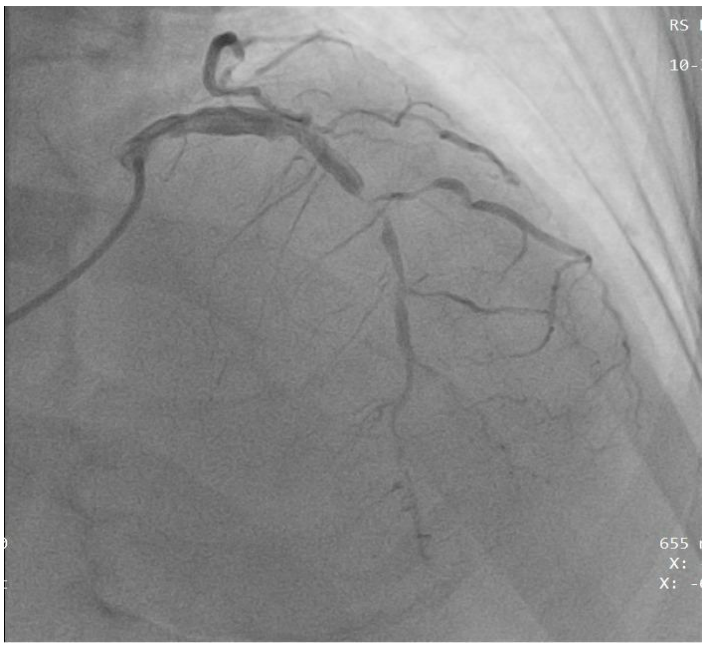

Figure 3. Coronary angiography

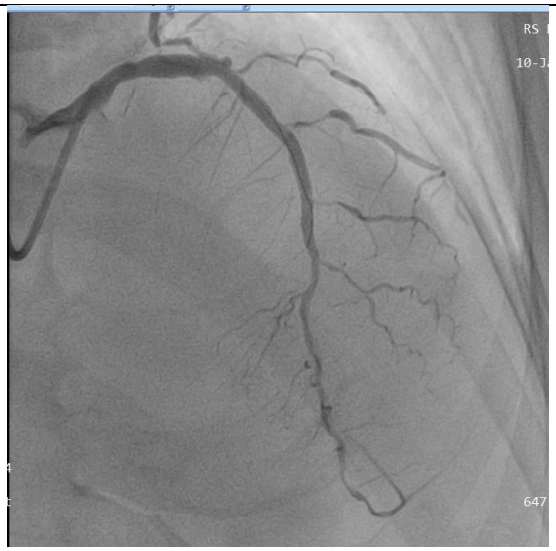

Figure 4. Post stent placement

DISCUSSION

This patient exhibited clinical characteristics typical of Wellens syndrome. Angina-like chest pain appeared in the last few days and had subsided at the time of evaluation, so the patient appeared stable. This phenomenon is consistent with the literature, stating that Wellens syndrome patients typically present pain-free after a previous angina episode, with minimal or normal cardiac enzyme levels (Blanch & Freixa-Pamais, 2021; Mathew et al., 2022). The absence of pain and regular cardiac markers can be misleading, making Wellens syndrome difficult to recognize initially (Coutinho Cruz et al., 2017; Mathew et al., 2022)(Blanch & Freixa-Pamais, 2021; Zhou et al., 2022). In this case, the patient's history of heavy smoking is highly relevant as a significant risk factor. Smoking is a major cause of coronary heart disease, contributing to approximately 30% of deaths from heart disease (Clemente et al., 2024; Miner et al., 2021). Chronic exposure to cigarette smoke triggers atherosclerosis through endothelial dysfunction, inflammation, and increased thrombosis, thereby accelerating the formation of plaques in the coronary arteries.

The patient's ECG pattern is highly characteristic of Wellens syndrome type A, characterized on the electrocardiogram (ECG) by a distinctive biphasic T-wave pattern, most prominently seen in precordial leads V2 and V3, and sometimes extending to V4 and V5 (Okobi et al., 2022; Zhang & Shen, 2025). Type B is typically seen in patients with cardiac risk factors (mean age 50-65), cases occur across age groups, including younger adults (Wang et al., 2018) The absence of ST elevation, loss of the R wave, or appearance of pathological Q waves on the ECG confirms no transmural infarction (Ramires et al., 2018). These abnormal T wave findings are not merely nonspecific changes; pathophysiologically, the Wellens pattern reflects severe ischemia in the anterior region, typically caused by critical stenosis in the proximal segment of the left anterior descending artery (widow maker lesion). Previous research by de Zwaan et al. showed that most patients with the Wellens pattern would progress to extensive anterior wall infarction within a few weeks without intervention. Therefore, recognition of this ECG pattern is crucial to enable timely therapy before the "storm" of myocardial infarction occurs. According to current recommendations, identification of the Wellens pattern should be immediately followed by coronary angiography and revascularization, as prompt action can prevent further myocardial damage and reduce mortality (Zhang & Shen, 2025).

The clinical data of this patient meets the diagnostic criteria for Wellens syndrome according to the literature: there is a recent history of angina that has resolved, the ECG shows biphasic T wave in the anterior precordial leads during pain-free periods, without ST-segment

elevation or Q waves, and normal troponin T levels (Mathew et al., 2022). The absence of troponin elevation, in this case, is consistent with the characteristics of Wellens syndrome as a precursor to myocardial infarction (pre-infarction syndrome) with ischemia that has not yet caused significant myocardial necrosis (Zhang & Shen, 2025). In addition to cardiac markers, other supportive tests also support the diagnosis and management. The patient had an eGFR of approximately 34 mL/min/1.73 m^2, indicating stage 3b renal function impairment. The presence of chronic kidney disease (CKD) is essential to note because CKD is associated with poorer prognosis in ACS; studies show that ACS patients with moderate CKD (stages 3a–4) have a two- to threefold higher risk of cardiovascular mortality compared to patients without CKD (Abdeldayem et al., 2025). Decreased kidney function is also a consideration in the selection and dosage of contrast agents during angiography/PCI and prophylaxis for contrast-induced nephropathy. On electrolyte testing, the patient showed no abnormalities (sodium and potassium levels were typical). Maintaining electrolyte balance is crucial in patients with acute coronary syndrome to prevent malignant arrhythmias related to ischemia. Meanwhile, the patient's chest X-ray revealed cardiomegaly. The 2023 ESC guidelines emphasize that in cases of Wellens' T wave findings, which are indicative of significant LAD occlusion, patients should be treated as very high-risk ACS patients requiring immediate coronary angiography (within 24 hours, even <12 hours) to prevent *pending myocardial infarction* (Miner et al., 2021). In this case, the decision to proceed directly to angiography without prior stress testing was shown to prevent infarction.

The management of this case highlights the recommended approach for Wellens syndrome. The 2023 European Society of Cardiology guidelines emphasize that patients with Wellens T-wave patterns (suggesting significant LAD occlusion) should be treated as very high-risk ACS patients, warranting immediate (within 12–24 hours) invasive evaluation and revascularization. (Miner et al., 2021). In this case, we appropriately avoided any stress testing and proceeded directly to diagnostic angiography, recognizing that a stress test in the setting of Wellens syndrome is contraindicated. Provoking increased cardiac demand with exercise or pharmacologic stress in a patient with a critical LAD stenosis could precipitate complete occlusion of the artery and result in a potentially fatal anterior MI (Wang et al., 2018). Instead, prompt invasive management was undertaken. The angiographic findings of a critical proximal LAD lesion in our patient were consistent with the ECG changes and confirmed the diagnosis. We performed PCI with stent placement to restore coronary perfusion, which is the definitive treatment to prevent an impending infarction. Early revascularization in Wellens syndrome has been shown to improve outcomes. In one cohort study, a higher proportion of patients with the Wellens pattern underwent early PCI compared to patients without the Wellens pattern, and after two years of follow-up, the aggressively treated Wellens group did not have significantly higher rates of major adverse cardiac events or mortality compared to the non-Wellens group (Sahitra & Haizil, 2022) This suggests that with rapid recognition and intervention ("managing the calm before the storm"), the long-term prognosis of Wellens syndrome can approach that of other ACS patients who did not have the ominous Wellens warning on their ECG. This patient case illustrates the importance of identifying the Wellens pattern promptly and performing revascularization before it progresses to a catastrophic anterior myocardial infarction (Okobi et al., 2022; Romero et al., 2023).

CONCLUSION

Wellens syndrome is a clinical electrocardiographic syndrome in unstable angina that indicates critical stenosis of the left anterior descending artery (LAD) and a high risk of impending anterior myocardial infarction. This case report of a 61-year-old Melanesian man highlights the characteristic features of Wellens syndrome, namely abnormal precordial T waves during pain-free episodes with normal cardiac enzymes, serving as an early warning sign before the onset of extensive myocardial infarction ("the calm before the storm").

Recognition of the Wellens ECG pattern is crucial as it requires aggressive coronary angiography and immediate revascularization management. This case discussion compares the patient's clinical findings with current literature: history of resolving chest pain, smoking risk factors, Wellens ECG pattern type A, supportive laboratory and radiographic findings, and confirmation of critical LAD stenosis on angiography. Appropriate management (early PCI) in Wellens syndrome can prevent progression to myocardial infarction and achieve optimal outcomes. Through this report, it is hoped that clinicians awareness of Wellens syndrome will increase, thereby promptly preventing the "storm" of myocardial infarction.

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