DOI: 10.7860/JCDR/2025/78572.20973

Case Series

Internal Medicine Section

Unmasking the Silent Danger: A Case Series of Wellens' Syndrome and its Implications for Early Intervention

HARIHARAN NARASAIYAN[1], N SWAMINATHAN[2]

ABSTRACT

Wellens' syndrome represents a critical Electrocardiographic (ECG) pattern indicative of severe proximal Left Anterior Descending (LAD) coronary artery stenosis, demanding urgent diagnosis and treatment. This case series details four patients with diverse clinical presentations and risk factors, all exhibiting Wellens' syndrome. The cases illustrate both classic ECG features-biphasic or deeply inverted T waves in precordial leads-and atypical features, such as biphasic T-wave inversion extending to lateral leads and post-prandial chest pain in a patient with prior right coronary artery Percutaneous Coronary Intervention (PCI). This series underscores the need for clinicians to maintain a low threshold of suspicion for Wellens' syndrome, even with atypical signs and low troponin levels. Each patient underwent coronary angiography revealing significant LAD stenosis, followed by successful PCI and secondary prevention strategies. These cases highlight the importance of recognising Wellens' syndrome and the urgency of diagnosis and treatment to prevent major adverse cardiac events.

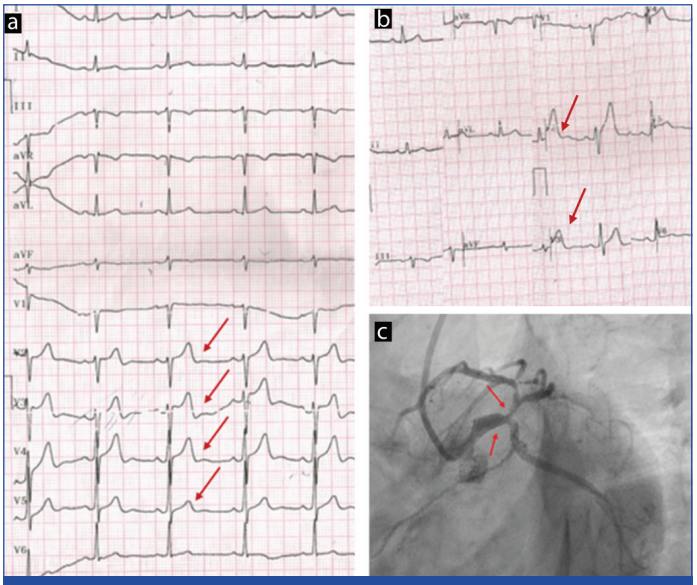
Keywords: Cardiac biomarkers, Left anterior descending artery stenosis, Myocardial infarction, T wave inversion

INTRODUCTION

Wellens' syndrome, first identified in the early 1980s in patients presenting with unstable angina, is characterised by specific ECG T-wave changes associated with severe proximal LAD artery stenosis. This stenosis causes blood flow obstruction followed by reperfusion, potentially progressing to extensive myocardial infarction [1]. ECG changes include minimal ST elevation, followed by characteristic T-wave inversion. Patients may present with chest pain, minimal ST elevation, preserved precordial R waves, and absent precordial Q waves [2,3]. Careful ECG interpretation is crucial in chest pain cases, even with normal cardiac biomarkers. These cases, ranging from classic biphasic T-wave inversion to complex presentations like postprandial angina, highlight the importance of coronary angiography and percutaneous intervention to prevent myocardial infarction. Thorough post-PCI management, including Dual Antiplatelet Therapy (DAPT), lipid-lowering strategies, and lifestyle modifications, is also essential for long-term patient health.

Case 1

A 65-year-old male with hypertension and diabetes presented with intermittent crushing chest pain radiating to the left arm over the preceding 24 hours. Vital signs were stable; cardiac auscultation revealed normal heart sounds without murmurs. Initial ECG showed classic biphasic T waves in leads V2-V3, suggestive of Wellens' syndrome [Table/Fig-1a-c]. Echocardiography excluded pulmonary embolism and aortic dissection. Troponin was negative; coronary angiography revealed distal left main disease and severe (90%) proximal LAD artery stenosis [Table/Fig-1c]. Successful PCI was performed. Symptoms resolved, and the ECG normalised [Table/Fig-1b]. The patient was discharged on aspirin (75 mg/day), clopidogrel (75 mg twice daily), metoprolol (25 mg/day), atorvastatin (40 mg/day), ramipril (5 mg/day), and metformin (1 g twice daily), along with lifestyle modifications. At one-month follow-up, the ECG and echocardiogram were normal, and clopidogrel was reduced to 75 mg once daily. At six months, the patient remained compliant, with all results within normal limits and no further angina.



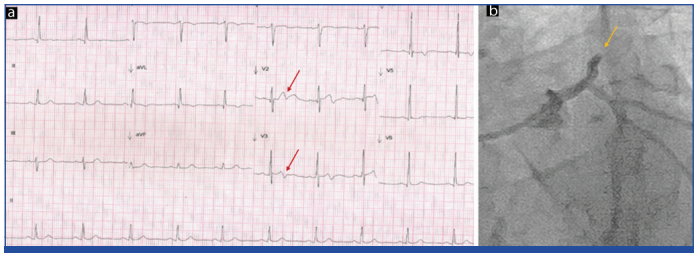
[Table/Fig-1]: (a) Classic biphasic T-waves (red arrow) in leads V2-V5 are suggestive of Wellens' syndrome; (b) After PCI, there was a resolution of the T-wave changes (red arrow); (c) CAG showing Distal left main disease along with critical LAD stenosis (red arrow).

Case 2

A 72-year-old male with coronary artery disease, hypertension, and a history of smoking presented with progressively worsening chest discomfort (6/10), described as chest tightness unrelieved by sublingual isosorbide dinitrate, and mild breathlessness over 48 hours. ECG showed prominent biphasic T-wave inversions in leads V2-V4, consistent with Wellens' syndrome [Table/Fig-2a]. Initial troponin was slightly elevated (2.3 ng/mL), and serum total cholesterol was 380 mg/dL, with a GRACE risk score of 3-10%. Coronary angiography revealed complete LAD occlusion [Table/Fig-2b]. Emergent angioplasty was performed. Symptoms resolved, and ECG changes normalised. The patient commenced DAPT, atenolol, and atorvastatin (40 mg/day). At one-month follow-up, echocardiography showed mild left ventricular dysfunction (ejection fraction 51%), which normalised by four months after adding an ACE inhibitor.

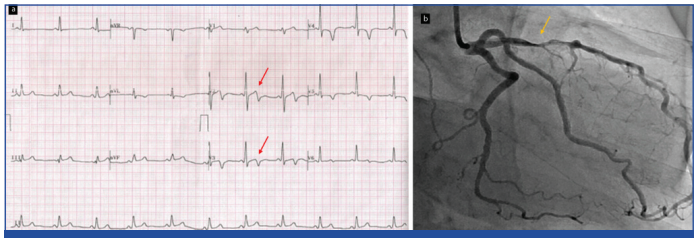
Case 3

A 45-year-old male with a family history of premature coronary artery disease and familial hypercholesterolaemia presented with two days



[Table/Fig-2]: (a) Classic biphasic T-waves in leads V2-V3 (red arrow) suggestive of Wellens' syndrome; (b) Coronary angiogram showing complete occlusion of LAD (yellow arrow).

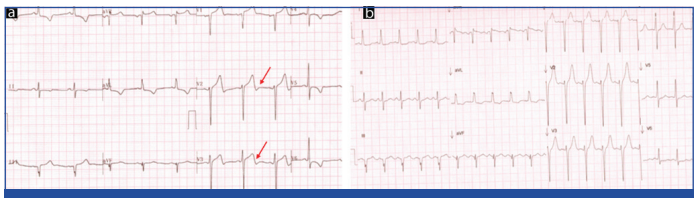
of retrosternal chest pain, initially exacerbated by activity and relieved by rest, but later persistent at rest. Vital signs were stable (heart rate 72 bpm, blood pressure 127/90 mmHg, respiratory rate 16 breaths/minute, SpO_2 98% on room air). LDL cholesterol was 282 mg/dL, and tendon xanthomas were present. Cardiac examination was normal; chest X-ray showed no acute cardiopulmonary disease; two troponin tests were negative. ECG showed typical biphasic T-wave inversions in leads V2-V3, suggesting Wellens' syndrome [Table/Fig-3a]. Coronary angiography revealed 75% proximal LAD artery stenosis [Table/Fig-3b]. Successful PCI with drug-eluting stent placement was performed. The patient commenced DAPT, and aggressive lipid management with high-dose atorvastatin (80 mg/day) and ezetimibe (10 mg/day) was initiated, along with beta-blocker therapy. At three-month follow-up, the patient was asymptomatic, with normal left ventricular function and LDL cholesterol reduced to 150 mg/dL.



[Table/Fig-3]: (a) Biphasic T-waves in leads V2-V3 (red arrow) suggestive of Wellens' syndrome); (b) Coronary angiogram showing 75% occlusion of LAD (yellow arrow).

Case 4

A 60-year-old female with ischaemic heart disease, diabetes, and a history of right coronary artery angioplasty five years prior presented with two weeks of postprandial epigastric and left-sided chest discomfort (burning in character), without radiation or associated symptoms. Laboratory results were normal, including troponin-T and CK-MB (11 U/L). ECG showed deep T-wave inversions in precordial leads (V2-V4), raising suspicion for Wellens' syndrome [Table/Fig-4a]. Echocardiography showed normal left ventricular function; coronary angiography demonstrated 90% proximal LAD artery stenosis, with a patent right coronary artery stent. PCI with stent deployment was performed. The patient received DAPT, a beta-blocker, an ACE inhibitor, and antidiabetic medications (including insulin), along with lifestyle modifications. One- and six-month follow-up visits showed adequate glycaemic control, with normal ECG and echocardiogram results [Table/Fig-4b].



[Table/Fig-4]: (a) classic biphasic T-waves in leads V2-V3 (red arrow) suggestive of Wellens' syndrome; (b) After PCI, the ECG findings were normalised.

DISCUSSION

In 1982, Wellens et al., first described the clinical and ECG criteria for Wellens' syndrome [4]. T-wave inversion is a common ECG abnormality seen in various conditions, including ischaemia, myocarditis, left ventricular hypertrophy, pulmonary embolism, and Wolff-Parkinson-White syndrome [3,5,6]. In ischaemia, T-wave inversions are usually symmetrical and pronounced. In Wellens' syndrome, characteristic biphasic T waves typically begin with a positive deflection before descending below the isoelectric line. These ECG changes are predominantly observed during pain-free intervals, reflecting reperfusion of ischaemic myocardium [7], and may persist for hours to weeks, potentially recurring with angina. Early detection is crucial to prevent myocardial damage. The mechanism is not fully understood, but ECG changes are thought to reflect reperfusion of ischaemic myocardium due to relief of proximal LAD artery spasm [8].

Wellens' syndrome comprises two main ECG patterns: Type 1 (biphasic T waves in leads V2-V3, prevalent in 24% of cases) and Type 2 (deep inverted T waves in leads V1-V4, more common at 76%) [9]. Diagnostic criteria include: (i) Type 1 or Type 2 T-wave pattern; (ii) history of angina; (iii) absence of Q waves; (iv) normal or minimally elevated troponin levels; (v) isoelectric or minimally elevated ST segment (<1 mm) [9]. This case series reinforces the established association between Wellens' syndrome and proximal LAD stenosis, consistent with the work of de Zwaan and Wellens et al., [4]. All four cases showed Type 1 biphasic T-wave inversion. The first three cases (male patients with varying risk factors) illustrate classic Wellens' syndrome, with biphasic Twave inversion and significant LAD stenosis requiring urgent intervention.

This series also reveals atypical presentations. Case 1 showed biphasic T-wave inversion extending to lateral leads, a finding not consistently reported [1,8,10]. Troponin levels varied (normal in Cases 1 and 3, mildly elevated in Case 2), consistent with reports showing Wellens' syndrome even with minimal or absent biomarker elevation [10], suggesting it is a pre-infarction condition. This variability should not deter angiography.

Treatment followed current best practice (DAPT, beta-blockers, statins, ACE inhibitors post-PCI). Case 3 demonstrates a tailored approach to hypercholesterolaemia. Serial ECGs and echocardiograms are vital for monitoring. Case 2 highlights the benefits of prompt intervention. Successful outcomes in all cases mirror the efficacy of revascularisation for LAD stenosis [11].

In Case 4, a 60-year-old female with postprandial chest pain, highlights the importance of recognising Wellens' syndrome despite atypical symptoms (potentially misdiagnosed as gastritis or gastro-oesophageal reflux disease). The presence of the Wellens' pattern and significant LAD stenosis, even with prior intervention, underscores the need for vigilance, even with unusual presentations.

CONCLUSION(S)

This case series supports Wellens' syndrome as a reliable indicator of impending proximal LAD stenosis, highlighting the importance of recognising distinctive ECG patterns. Elevated clinical suspicion is crucial for atypical presentations, such as postprandial chest pain with Wellens' ECG abnormalities. Troponin level variations should not delay coronary intervention when the Wellens' pattern is present. Prompt PCI consistently yielded successful outcomes, emphasising the importance of timely intervention. Comprehensive secondary prevention and evidence-based treatments remain essential for long-term patient well-being.

REFERENCES

[1] Marzlin KM. Wellens syndrome. AACN Adv Crit Care. 2018;29(3):360-64.
[2] Nisbet BC, Zlupko G. Repeat Wellens' syndrome: Case report of critical proximal left anterior descending artery restenosis. J Emerg Med. 2010;39(3):305-08.
[3] Dhawan SS. Pseudo-Wellens' syndrome after crack cocaine use. Can J Cardiol. 2008;24(5):404.
[4] de Zwaan C, Bär FWHM, Wellens HJJ. Characteristic electrocardiographic pattern indicating a critical stenosis high in left anterior descending coronary artery in patients admitted because of impending myocardial infarction. Am Heart J. 1982;103(4, Part 2):730-36.
[5] Ola O, Tak T. Pseudo-Wellens syndrome in a patient with hypertension and left ventricular hypertrophy. Am J Case Rep. 2019;20:1231-34.

[6] Ozdemir S, Cimilli Ozturk T, Eyinc Y, Onur OE, Keskin M. Wellens' Syndrome – Report of two cases. Turk J Emerg Med. 2016;15(4):179-81.
[7] Birnbaum Y, Wilson JM, Fiol M, de Luna AB, Eskola M, Nikus K. ECG diagnosis and classification of acute coronary syndromes. Ann Noninvasive Electrocardiol. 2014;19(1):04-14. Doi: 10.1111/anec.12130.
[8] Sheng FQ, Rong HM, Lin ZM, Ying SG. Wellens syndrome caused by spasm of the proximal left anterior descending coronary artery. J Electrocardiol. 2015;48(3):423-25.
[9] Al-Assaf O, Abdulghani M, Musa A, AlJallaf M. Wellen's syndrome: The life-threatening diagnosis. Circulation. 2019;140(22):1851-52.
[10] Lindsell CJ, Anantharaman V, Diercks D, Han JH, Hoekstra JW, Hollander JE, et al. The Internet Tracking Registry of Acute Coronary Syndromes (i*trACS): A multicenter registry of patients with suspicion of acute coronary syndromes reported using the standardized reporting guidelines for emergency department chest pain studies. Ann Emerg Med. 2006;48(6):666-77, 677.e1-9.
[11] Stone GW, Kappetein AP, Sabik JF, Pocock SJ, Morice MC, Puskas J, et al. Five-year outcomes after PCI or CABG for left main coronary disease. N Engl J Med. 2019;381(19):1820-30.

PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Electrophysiology and Pacing, Christian Medical College, Vellore, Tamil Nadu, India.
2. Professor (Rtd), Department of Cardiology, Madras Medical College, Chennai, Tamil Nadu, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Hariharan Narasaiyan,
Block: B1, Floor 2, Door B3 (64-2B3), CMC Kannigapuram Residential Campus, Rathinagiri, Kilminnal Post, Walaja Taluk Ranipet, Vellore-632517, Tamil Nadu, India.
E-mail: narasaiyanhariharan@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Feb 15, 2025
- Manual Googling: Mar 15, 2025
- iThenticate Software: Mar 22, 2025 (7%)

ETYMOLOGY: Author Origin

EMENDATIONS: 6

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Feb 14, 2025**
Date of Peer Review: **Mar 07, 2025**
Date of Acceptance: **Mar 24, 2025**
Date of Publishing: **May 01, 2025**