CASE REPORT

Wellens' Syndrome with Positive T Wave in Lead aVR Suggesting Severe Three-Vessel Coronary Arterial Disease

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Abstract : Non-ST-elevation myocardial infarction (NSTEMI) showing biphasic or deep negative T waves in lead V2-3 suggests the presence of a severe stenotic lesion in the proximal left anterior descending artery, called Wellens' syndrome. We report a case of NSTEMI with a positive T wave in lead aVR and widespread negative T waves in the precordial and inferior leads, presumed to be Wellens' syndrome, resulting in three-vessel severe coronary artery disease. This report highlights the importance of identifying a positive T wave in lead aVR to avoid overlooking severe organic stenosis of the coronary arteries. J. Med. Invest. 72 : 451-454, August, 2025

Keywords **:** *Wellens' syndrome, global ischemia, positive T wave in lead aVR*

INTRODUCTION

Acute coronary syndrome (ACS) is primarily caused by plaque rupture in the coronary arteries and is accompanied by chest pain or oppression ; however, some patients do not show these typical symptoms (1). Therefore, a comprehensive diagnosis should be made based on clinical symptoms, biomarkers, and electrocardiography (ECG) readings. During screening for ACS, symptoms are often absent during presentation, particularly in patients with non-ST-elevation myocardial infarction (NSTEMI). Hence, ECG is the primary tool used for the screening and diagnosis of ACS.

NSTEMI showing biphasic or deep negative T waves in V2-3 suggests the presence of a severe stenotic lesion in the proximal left anterior descending artery (LAD), and this condition is called Wellens' syndrome (2). Patients presenting with ST-segment elevation in lead aVR accompanied by widespread ST-segment depression in the precordial and inferior leads are typically indicative of global subendocardial ischemia of the left ventricle, most commonly associated with left main coronary artery (LMCA) stenosis or three-vessel coronary artery disease (3VD) (3). However, the significance of a positive T wave in lead aVR without ST-segment elevation in patients with NSTEMI has not yet been established, although a small-scale study demonstrated that it has predictive value for 3VD or LMCA stenosis (4). In this study, we report a case of NSTEMI with a positive T wave in lead aVR and widespread negative T waves in the precordial leads, presumed to be Wellens' syndrome, accompanied by 3VD.

CASE REPORT

An 88-year-old man experienced chest pain for 10 min with cold sweats at rest at night 2 weeks prior, followed by chest pain with the same symptoms every 3 days at night. He had no symptoms on exertion, even after the onset of chest pain at rest. The patient's family physician suspected unstable angina pectoris ; therefore, he presented to our hospital for further examination. An ECG at the time of presentation showed negative T waves in leads V1–V5, II, III, and aVF, as well as a positive T wave in lead aVR. These findings were not present on the ECG obtained 7 years earlier. Atrial fibrillation, which had been noted previously, was also observed (Fig. 1). He had a history of hypertension, dyslipidemia, and chronic atrial fibrillation ; underwent radiotherapy for nasopharyngeal cancer ; and was medicated with candesartan, rosuvastatin, and apixaban. He had a history of smoking but quit 38 years ago. The patient's height was 163 cm, weight was 61 kg, and BMI was 23 kg/m², blood pressure was high at 169/82 mmHg, and his pulse was irregular at 71 beats per minute. There were no signs of heart failure, including jugular vein distention or lower leg edema. The patient's laboratory data at the time of presentation are summarized in the Table : slightly elevated high-sensitivity troponin I levels (60 pg/mL ; upper limit, 27 pg/mL) and white blood cell count (11,600/μL) ; however, the levels of other myocardial enzymes, including creatine kinase MB, lactate dehydrogenase, and aspartate aminotransferase, were within normal ranges. Low-density lipoprotein cholesterol (74 mg/dL) was well controlled with 5 mg rosuvastatin, prescribed for the primary prevention of coronary artery disease. Chest X-ray revealed cardiomegaly with an increased cardiothoracic ratio of 53%, although no signs of pulmonary congestion were observed (Fig. 2). Echocardiography demonstrated diffusely reduced wall motion with a left ventricular ejection fraction of 50%.

Coronary angiography was performed after admission, which revealed severe stenosis in the middle LAD with a slow flow in the first septal branch as well as stenosis in the distal circumflex branches of the left coronary artery and posterior descending branch of the right coronary artery (Fig. 3). Percutaneous coronary intervention (PCI) was subsequently performed for the stenotic lesion in the LAD. On the day after PCI, the negative T waves in V1-2 persisted ; however, the T wave in the lead aVR had become negative. Two weeks later, the ECG demonstrated positive T waves in leads V3 to V6, along with a positive T wave in lead aVR.

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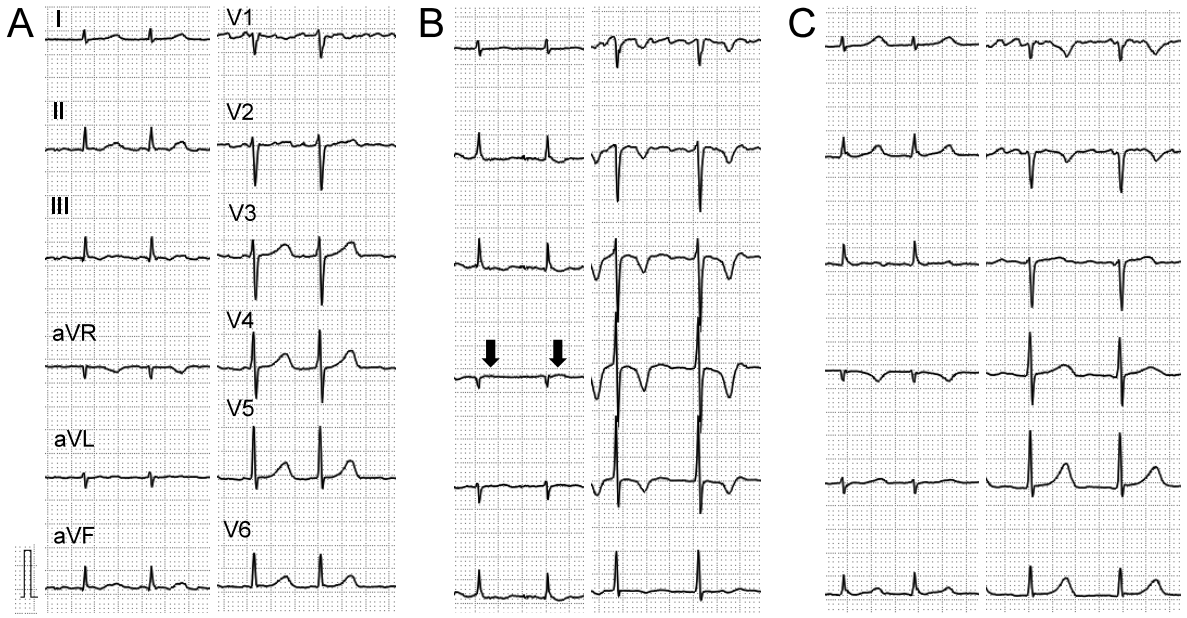

Fig 1. Images of ECG readings
(A) Electrocardiogram recorded 7 years prior to admission, showing atrial fibrillation without significant ST-T changes. (B) ECG at presentation showing negative T waves in leads V1–V5 and a positive T wave in lead aVR (arrows). (C) ECG obtained 2 weeks after percutaneous coronary intervention for the left anterior descending artery, showing normalization with positive T waves in leads V3–V6 and a negative T wave in lead aVR.

Table. The results of blood tests at the time of presentation.

Item	Value	Unit
White blood cell count	11600	/μL
Hemoglobin	16.1	g/dL
Platelet count	208	$\times 10^3/\mu L$
Aspartate aminotransferase (AST)	25	U/L
Alanine aminotransferase (ALT)	19	U/L
Lactate dehydrogenase (LDH)	118	U/L
Creatine kinase (CK)	65	U/L
Creatine kinase MB	1.9	ng/mL
Blood urea nitrogen (BUN)	16	mg/dL
Creatinine (Cre)	0.84	mg/dL
Sodium (Na)	135	mEq/L
Potassium (K)	4.4	mEq/L
C-reactive protein (CRP)	1.08	mg/dL
Triglycerides (TG)	39	mg/dL
Low-density lipoprotein cholesterol (LDL-C)	74	mg/dL
High-density lipoprotein cholesterol (HDL-C)	84	mg/dL
Blood sugar (BS)	121	mg/dL
Hemoglobin A1c (HbA1c)	5.4	%
B-type natriuretic peptide (BNP)	18	pg/mL
High-sensitivity troponin I	60	pg/mL

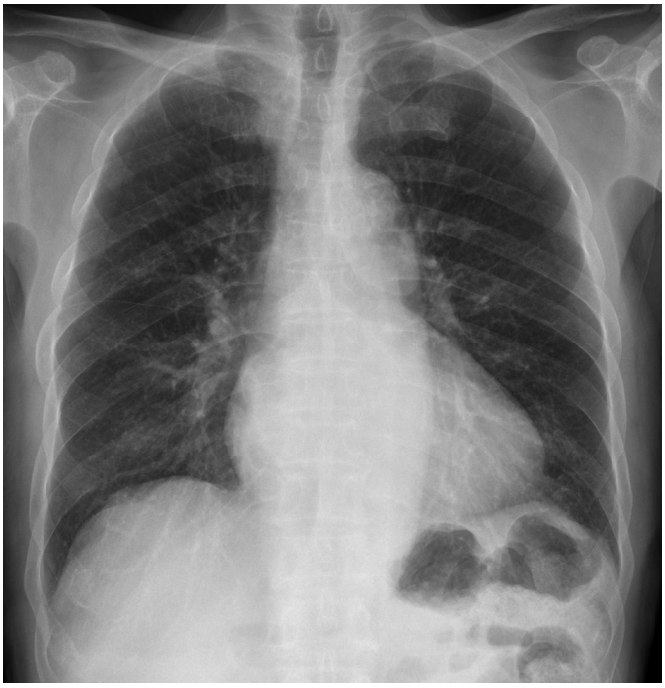

Fig 2. Images of chest X-ray
Chest X-ray revealed cardiomegaly with an increased cardiothoracic ratio of 53%, although no signs of pulmonary congestion were observed.

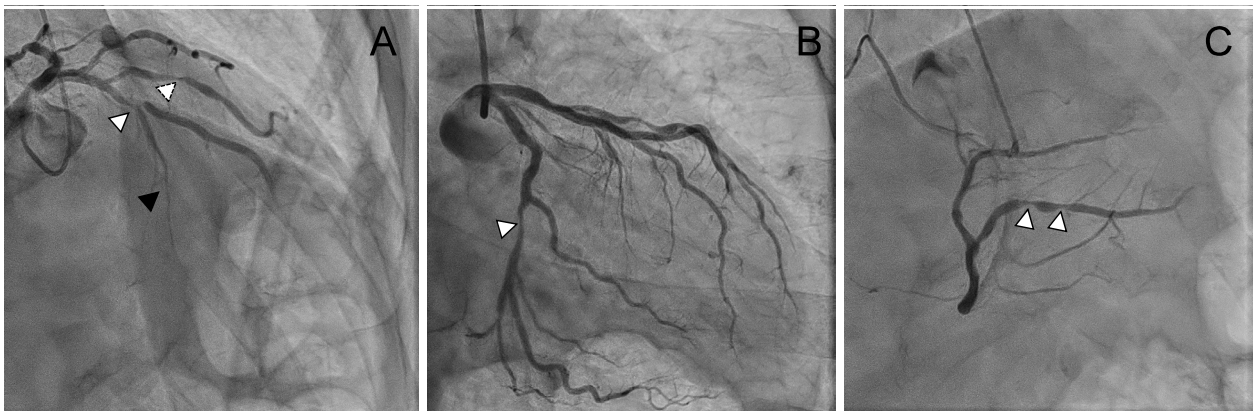

Fig 3. Images of coronary angiography
Severe stenosis was observed in the mid-portion of the left anterior descending artery (A, white arrowhead), accompanied by slow flow in the first septal branch (A, black arrowhead), severe stenosis of the diagonal branch (A, dashed white arrowhead), and severe stenosis in the distal segment of the circumflex artery (B, white arrowhead), as well as in the posterior descending branch of the right coronary artery (C, white arrowhead).

One month later, PCI was performed on the right and circumflex coronary arteries. The patient's symptoms were stable, and no symptoms were observed in the outpatient setting.

DISCUSSION

Herein, we report a case of a positive T wave in lead aVR accompanied by negative T waves in the precordial and inferior leads in a patient with NSETMI who presented with atypical symptoms of chest pain at rest but not on exertion.

Wellens' syndrome is a type of unstable angina pectoris or NSTEMI, first described by Wellens in 1982 (2). It is characterized by asymptomatic negative or biphasic T waves in the precordial leads and is associated with a high risk of progression to myocardial infarction. Previous studies have reported that 75% of patients with severe stenotic lesions in the proximal portion of the LAD develop anterior wall infarction within 2–3 weeks. The clinical and electrocardiographic criteria for diagnosing Wellens' syndrome are as follows (5) : symmetric and deeply inverted T waves in leads V2 and V3, occasionally in leads V1, V4, V5, and V6, or biphasic T waves in leads V2 and V3 plus isoelectric

or minimally elevated (<1 mm) ST segment ; no precordial Q waves ; a history of angina ; pattern present in a pain-free state ; and normal or slightly elevated cardiac serum markers.

In 1983, Haines reported that, among 118 cases of unstable angina, 25 (86%) of 29 cases with negative or biphasic T waves in the precordial leads showed significant stenosis in the LAD and that the sensitivity and specificity for detecting significant LAD stenosis were 69% and 89%, respectively (6). Therefore, paying attention to negative T waves is important to avoid overlooking severe organic stenosis of the coronary arteries, even when symptoms are atypical and suggest coronary spasm, as in the present case, where the patient experienced symptoms only at rest, not on exertion.

In a study examining the relationship between negative T waves and magnetic resonance imaging findings in Wellens' syndrome, it was found that an increase in negative T-wave depth in precordial leads after the onset of chest pain was associated with myocardial stunning at the apex (7). In ischemia–reperfusion injury, oxidative stress, mitochondrial dysfunction, endoplasmic reticulum stress, and activation of proteolytic enzymes such as calpain by excess intracellular calcium ions may disrupt contractile proteins and ion channels, triggering myocardial stunning and leading to the occurrence of a negative T wave (8).

Lead aVR is generally considered to provide limited information, as changes in lead aVR are believed to reflect reciprocal changes in the lateral, inferior, and precordial leads. However, accumulating evidence has shown that ST elevation or positive T waves in lead aVR are associated with global subendocardial ischemia or localized ischemia at the basal region of the heart. A prospective cohort study of 400 patients with NSTEMI reported that a positive T wave in lead aVR is predictive of 3VD or LMCA stenosis (odds ratio, 3.747 ; 95% confidence interval, 2.058–6.822 ; p <0.001) (4). In the present case, coronary angiography revealed 3VD accompanied by 99% stenosis of the first septal branch, suggesting that basal transluminal and global subendocardial ischemias contributed to the occurrence of a positive T wave in lead aVR.

Kosuge *et al*. reported that the presence of a positive T wave in lead aVR combined with the absence of a negative T wave in lead V1 is characteristic of Takotsubo cardiomyopathy and can differentiate it from reperfused anterior myocardial infarction, with a specificity of 95% and a sensitivity of 94% (9). This is thought to occur because lead aVR indirectly reflects the apical region of the heart. In Takotsubo cardiomyopathy, extensive wall motion abnormalities centered on the apex are likely to result in T-wave polarity changes in lead aVR. In the present case, a negative T wave was observed in lead V1 after the chest pain episode, raising suspicion for reperfused myocardial ischemia rather than Takotsubo cardiomyopathy.

In conclusion, an extensively negative T wave accompanied by a positive T wave in lead aVR indicates basal or global left ventricular ischemia. A positive T wave in lead aVR should always be evaluated in patients with suspected unstable angina or NSTEMI.

DECLARATION OF COMPETING INTEREST

The authors state that they have no conflict of interest.

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