A 56-year-old woman with chest pain and emesis is brought to the hospital by ambulance. En route, the pain resolves after nitrates, morphine, and aspirin are administered. Results of a physical examination are normal. A 12-lead ECG is obtained; the tracing is shown here. Serum markers are measured every 4 hours; all levels are normal. Although the patient remains pain-free, the findings on a second ECG are unchanged. A nuclear cardiac stress imaging study is ordered.

Which of the following best explains the ECG findings?
A. Acute pulmonary embolism.
B. Wellens syndrome.
C. Persistent juvenile T-wave pattern.
D. Left ventricular hypertrophy.
E. Subarachnoid hemorrhage.

**DISCUSSION**
The ECG shows inverted T waves in leads V2 through V6 but normal ST segments (Figure 1). T waves are deeply inverted in V3 through V5. In a pain-free patient who has normal serum marker levels, these findings suggest **Wellens syndrome (B)**, in which the T-wave changes in the precordium are associated with critical, proximal left anterior descending (LAD) artery stenosis. In Wellens and colleagues' first description of this syndrome in a subgroup of patients hospitalized for unstable angina who were at high risk for anterior wall myocardial infarction (MI), a prominent, common feature in these patients was the pattern of ECG changes that involved the T wave, with occasional abnormality of the ST segment. There were 2 basic patterns of ECG changes:

- An isoelectric or minimally elevated ST segment leading into a symmetric, deeply inverted T wave (Figures 1 and 2).
- Biphasic T waves (Figure 3).

Approximately 20% of patients in the initial study demonstrated these ECG patterns. Moreover, all of the patients with these ECG changes had significant disease of the proximal LAD artery. Seventy-five percent of these patients did not undergo coronary revascularization, and extensive anterior wall infarction developed in these patients within a few weeks after admission. In a second, prospective investigation, Wellens and colleagues found that approximately 15% of patients with unstable angina presented with these characteristic ECG changes. In all patients in whom such changes were evident, urgent coronary angiography demonstrated significant LAD artery obstruction. **Features of Wellens syndrome.** The criteria for Wellens syndrome include T-wave changes plus a history of ischemic chest pain without serum marker abnormalities (Table 1). These criteria essentially describe a pain-free patient with continued ECG abnormalities who has not had an acute MI. Additional features of the syndrome may include:

- T-wave abnormalities (deeply inverted or biphasic T waves) in the right to mid precordial leads.
- Minimal or no ST-segment elevation.
- Normal precordial R-wave progression.
- Absence of Q waves.

The T-wave abnormalities are very apparent on the ECG and are a key feature of this ECG syndrome.
The T-wave changes may have 1 of 2 patterns:

- T waves are deeply inverted and symmetric in contour. As the ST segment terminates, the T wave assumes a very negative angle relative to the isoelectric baseline; this angle may approach 90 degrees (see Figures 1 and 2). This is the more common of the 2 patterns and is seen in approximately 75% of affected patients.
- T waves are biphasic (see Figure 3). This is the less common variant, seen in 25% of patients with Wellens syndrome.1,2

The T-wave changes are present in leads V2 and V3; in certain cases, the changes may also involve leads V1 and V4. T-wave abnormalities may persist for hours to weeks despite the resolution of chest discomfort. In the second study by Wellens and associates2, 60% of patients in whom the syndrome was diagnosed demonstrated the characteristic ECG changes on admission. After admission, the classic ECG abnormalities developed in a majority of the remaining patients. Neither significant ST-segment elevation nor Q waves are evident on the ECGs of patients with Wellens syndrome. The ST segment itself is often normal (ie, isoelectric). If the ST segment is abnormal, it is minimally elevated—usually less than 1 mm, with a high take-off from the QRS complex (see Figure 3). Elevated ST segments are usually either convex or obliquely straight; however, concave morphologies are also seen.1 Differential diagnosis of inverted T waves. Interpreting the ECG in the context of the clinical presentation can help the clinician distinguish between Wellens syndrome and a number of other causes of T-wave inversions. In this woman, acute pulmonary embolism (A) can be ruled out because of the lack of dyspnea and tachycardia; cerebrovascular accident (B), because of the lack of headache and focal neurologic issues; and a persistent juvenile pattern (C), because of her age. Left ventricular hypertrophy (LVH) (D) can be excluded because of the absence of prominent QRS complexes consistent with LVH on this patient’s ECG. Other entities in the electrocardiographic differential diagnosis of inverted T waves are listed in Table 2. Of these, acute myocarditis and pericarditis can often be distinguished from Wellens syndrome on the basis of the clinical picture. In acute myocarditis, acute congestive heart failure is normally present. In pericarditis, the typical chest pain pattern is different from that seen in Wellens syndrome. Wolff-Parkinson-White (WPW) syndrome and bundle-branch block (BBB) can usually be differentiated from Wellens syndrome based on the presence of additional ECG abnormalities not usually seen in the latter entity (delta waves and widened QRS complexes in WPW syndrome and an abnormal QRS morphology and width in BBB) Past MI cannot be distinguished from Wellens syndrome on the basis of either clinical information or electrocardiographic features. In patients in whom Wellens syndrome is a possible diagnosis, past MI should be considered as well. Always keep in mind, however, that this ECG pattern, with T-wave abnormalities in the right to mid precordial leads, is strongly associated with proximal LAD artery obstruction and anterior wall MI. Significance of Wellens syndrome. The importance of this syndrome lies in its clinical presentation and natural history. Patients have had recent chest discomfort but appear well; ECG abnormalities continue after the pain resolves. The natural history is essentially anterior wall infarction with associated morbidity and mortality. The ECG findings that characterize Wellens syndrome may be the only indication of an impending extensive anterior wall acute MI in an otherwise asymptomatic patient. Thus, it is vital to recognize these changes and to be aware of their association with critical LAD artery obstruction and significant, short-term risk of anterior wall MI. Stress imaging may not be the most appropriate testing modality in patients with Wellens syndrome; such testing may precipitate an acute MI. Definitive management of the stenosis (through either catheter-based or surgical therapy) leads to resolution of the ECG changes. Outcome of this case. After a cardiology consultation, the diagnostic plan was altered. Cardiac catheterization revealed a near-total occlusion of the LAD artery. Angioplasty with stent placement was performed successfully. The patient was discharged without incident, and 1 year later she remains in good health.

References: REFERENCES:

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