Evolving Electrocardiographic Indications for Emergent Reperfusion

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KEYWORDS

• ECG • ACS • STEMI • Coronary

KEY POINTS

- Specific high-risk electrocardiogram (ECG) patterns may represent acute myocardial infarction (AMI) or identify impending AMI that will benefit from early diagnostic coronary angiography.
- The ECG continues to play a critical role in determining which patients require urgent coronary angiography and revascularization.
- It is important to be familiar with obvious indications for emergent cardiac catheterization, and ECG patterns in which urgent coronary angiography and revascularization may improve patient outcomes.

INTRODUCTION

Chest pain or other symptoms concerning for acute coronary syndrome (ACS) continues to remain a major reason for presentation to the emergency department. However, there is significant heterogeneity in the spectrum of risk severity of these patients, ranging from patients presenting either with cardiac arrest or cardiogenic shock to those patients presenting with benign noncardiac chest pain. The electrocardiogram (ECG) remains a critically valuable tool in the physician's arsenal to diagnose patients and help with risk stratification. Although the management of ST-segment elevation myocardial infarction (STEMI) is straightforward and requires rapid reperfusion, there are multiple other high-risk ECG findings that are also suggestive of adverse outcome and may benefit from rapid transfer for coronary angiography. This article reviews specific high-risk ECG patterns that may represent acute myocardial infarction (AMI) or identify impending AMI that benefits from early diagnostic coronary angiography.

WELLENS SYNDROME

This syndrome is a characteristic pattern present in the precordial leads of the ECG in patients presenting with ACS that signifies the presence of a critical stenosis of the proximal left anterior descending (LAD) coronary artery. Wellens T waves were first described in 1982 from a series of 26 of 145 patients presenting with unstable

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angina who had initial negative cardiac enzymes. Wellens T waves are characterized by biphasic T-wave inversions in leads V_2 or V_3 with an initial positive deflection and subsequent terminal negative deflection (type A, ~25% of patients) or symmetric, often deep (>2 mm), T-wave inversions in the anterior precordial leads (type B, ~75% of patients).[1] Although these changes are present in V_2 and V_3, they are found across the precordial leads, commonly involving V_4 but much less frequently seen in V_1.[2] These characteristic changes typically develop after the resolution of chest pain. However, patients may develop either ST-segment elevation or normalization of the ST segment and T wave with recurrence of chest pain.[2] An example of Wellens T waves progressing to anterior STEMI is found in **Fig. 1**. Wellens syndrome also requires that there be absence of electrocardiographic criteria for myocardial infarction, such as pathologic Q waves or absence of R waves, because the T-wave morphology seen with Wellens syndrome mimics the T-wave inversion pattern found following a recent anteroseptal myocardial infarction.

The presence of Wellens syndrome carries significant diagnostic and prognostic value. In the initial series, the value of Wellens T waves was not recognized until most developed anterior AMI. Of the 13 patients that underwent coronary angiography in this initial group, 12 were found to have greater than or equal to 90% stenosis of the proximal LAD.[1] Because the characteristic T-wave changes are a harbinger of a critical stenosis in the proximal LAD, the prognostic importance was highlighted by the high percentage of patients that develop AMI and death. A subsequent prospective study in 1260 patients with unstable angina by the same group demonstrated that 180 of the patients (14%) presented with Wellens T waves, all of whom were shown to have greater than or equal to 50% stenosis of the LAD on coronary angiography.[3]

Clinical management of patients with Wellens syndrome requires rapid recognition because it signifies a large impending anterior AMI. Although medical therapy for ACS is obviously required, rapid transfer for urgent coronary angiography and possible revascularization is imperative. Patients treated conservatively with medical therapy but without revascularization progress to AMI and potentially death.[1] Therefore, although Wellens syndrome is not considered a STEMI equivalent, it is our clinical practice to take these patients urgently or emergent to the cardiac catheterization laboratory to avoid the development of anterior STEMI and associated pathologic consequences.

The clinical context of patients presenting with Wellens syndrome is critical because there are a variety of ECG patterns that mimic Wellens syndrome. It is essential because "pseudo-Wellens T waves" can also occur in a variety of clinic conditions. In the case of cocaine and intracranial hemorrhage, the cause of the pseudo-Wellens T waves may be the result of coronary spasm involving the proximal LAD. Among patients with intracranial hemorrhage, the ECG can frequently demonstrate inverted T waves and QT prolongation.[4–6] Patients with deep inverted T waves have also been shown to have a characteristic

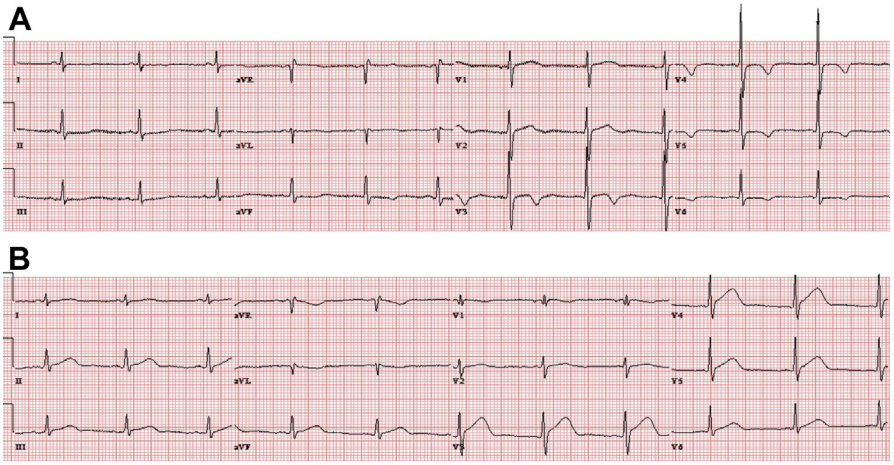

Fig. 1. Wellens syndrome converting to anterior STEMI. A 73-year-old woman with hypertension and hyperlipidemia presented to the emergency department with 2 hours of intermittent chest pain. (*A*) Her initial ECG demonstrated Wellens syndrome (type II) with T-wave inversions in leads V_2 and V_3. (*B*) Repeat ECG was performed after return of chest pain and demonstrated anterior STEMI. She underwent emergent coronary angiography, which demonstrated a subtotal occlusion of the proximal LAD (99% stenosis).