Wellens' Syndrome

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Received for publication April 30, 1998. Revision received August 24, 1998. Accepted for publication October 8, 1998.

Assertions and opinions herein are the views of the authors and should not be construed as official or as reflecting the views of the Department of Defense or the Department of the Navy.

Reprints not available from the authors.

47/1/96041

We describe a patient with Wellens' syndrome. In view of the large area of myocardium at risk, the importance of recognizing the significance of this ECG pattern is of critical importance for the emergency physician, especially those involved in the evaluation of patients at emergency department chest pain centers. Wellens' syndrome, the criteria for diagnosis, and a discussion of its implications are presented.

[Tandy TK, Bottomy DP, Lewis JG: Wellens' syndrome. *Ann Emerg Med March* 1999;33:347-351.]

INTRODUCTION

In 1982, H. J. J. Wellens and his group first described a characteristic ECG pattern of T waves in the precordial leads that were associated with a critical stenosis of the proximal left anterior descending coronary artery.[1] Recognition of this pattern, and appropriate intervention, prevents a potentially devastating anterior wall myocardial infarction. Importantly for the emergency physician, Wellens' T-wave changes usually occur during a pain-free interval when other evidence of ischemia or unstable angina may be absent. Although these patients may initially respond well to medical management, they ultimately fare poorly with conservative therapy and require aggressive treatment. There are 2 variants of Wellens' syndrome. The first, a more common pattern also known as the "left anterior descending coronary T-wave syndrome," has been previously described in the emergency medicine literature.[2,3] However, a less common, less well-described, and potentially more lethal variant of Wellens' syndrome exists that is the subject of this case report.

CASE REPORT

A 39-year-old white man presented to an outlying hospital with a complaint of intermittent, sharp, substernal chest pain of several weeks' duration; the pain occurred at rest

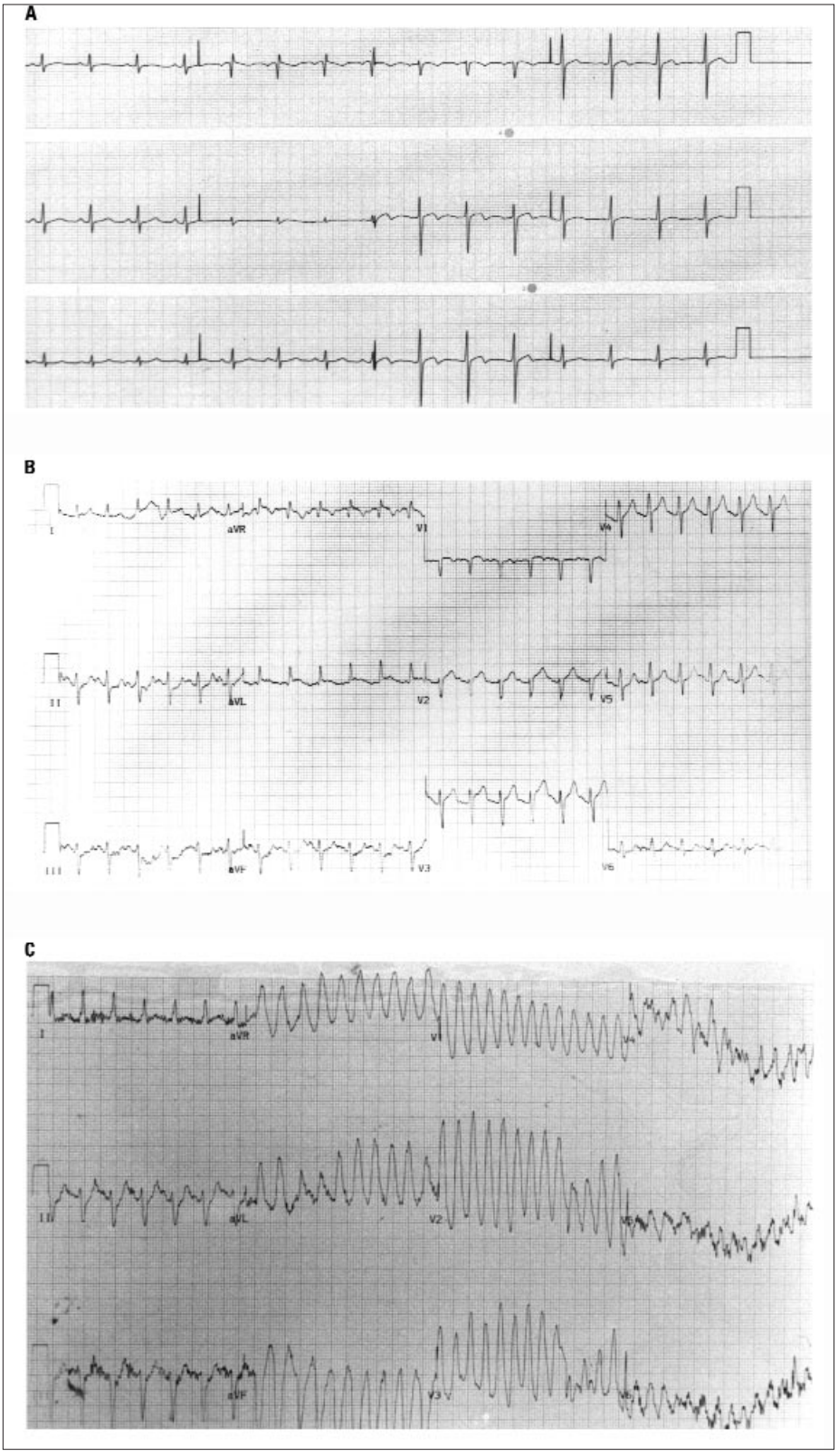

Figure 1.
A, *Baseline ECG shows Wellens' T waves in leads V_2 and V_3.* **B**, *ST-segment elevation during EST.* **C**, *Ventricular tachycardia during EST.*

and with exercise. He had a history of hypertension and diabetes, smoked, had a positive family history for cardiac disease, and his cholesterol level was unknown. He had normal physical examination findings, blood electrolyte levels, ECG (unavailable for review), and cardiac enzyme determination done 2 weeks previously as an outpatient. An ECG obtained 1 hour before his outpatient exercise stress test (EST), when the patient was pain-free, is shown in Figure 1A. Retrospectively, Wellens' T waves in leads V_2 and V_3 are clearly present. Note that the computerized interpretive algorithm failed to diagnose Wellens' syndrome. The decision was made for this patient to undergo an EST.

After 3 minutes of the EST, ST-segment elevation developed in the anterior leads (Figure 1B), with subsequent development of ventricular tachycardia (Figure 1C). The patient died within an hour despite thrombolytic therapy and Advanced Cardiac Life Support protocols. Autopsy revealed 80% occlusion of the left main and 90% to 95% occlusion of the proximal left anterior descending coronary arteries.

DISCUSSION

In 1982, Wellens and his colleagues first published the clinical and ECG criteria of a subgroup of patients with myocardial ischemia that later came to be known as Wellens' syndrome.[1] Recognition of this ECG pattern allowed the identification of patients who had a critical stenosis of the proximal left anterior descending coronary artery and hence were at risk for an extensive anterior wall myocardial infarction. In Wellens' study, the ECG changes were not rare. Of the initial study group of 145 consecutive patients admitted for unstable angina, 26 (18%) showed this ECG pattern, half on presentation and half within the next 24 hours.[1] In a later prospective study, 180 of 1,260 patients (14%) had the typical ECG pattern, with 108 of 180 (60%) within 24 hours.[4] Before fully realizing the importance of his findings, Wellens noted that 75% of these patients had an anterior wall myocardial infarction, usually within a matter of days despite relief of symptoms with medical management. These findings were later corroborated by an independent study.[5] Consequently, early cardiac catheterization with subsequent angioplasty or coronary bypass surgery is now recommended for these patients.

Two variants of precordial ST-T–wave abnormalities are seen in Wellens' syndrome (Figure 2). The first pattern (Figure 2A) consists of biphasic T waves in leads V_2 and V_3 and represents a smaller group (24%) of cases. The larger group (76%) of cases (Figure 2B) has deeper, symmetrically inverted T waves in leads V_2 and V_3, often in leads V_1 and V_4, and occasionally in leads V_5 and V_6.

The simplified criteria for Wellens' syndrome are as follows:

- Prior history of chest pain
- Little or no cardiac enzyme elevation
- No pathologic precordial Q waves
- Little or no ST-segment elevation
- No loss of precordial R waves

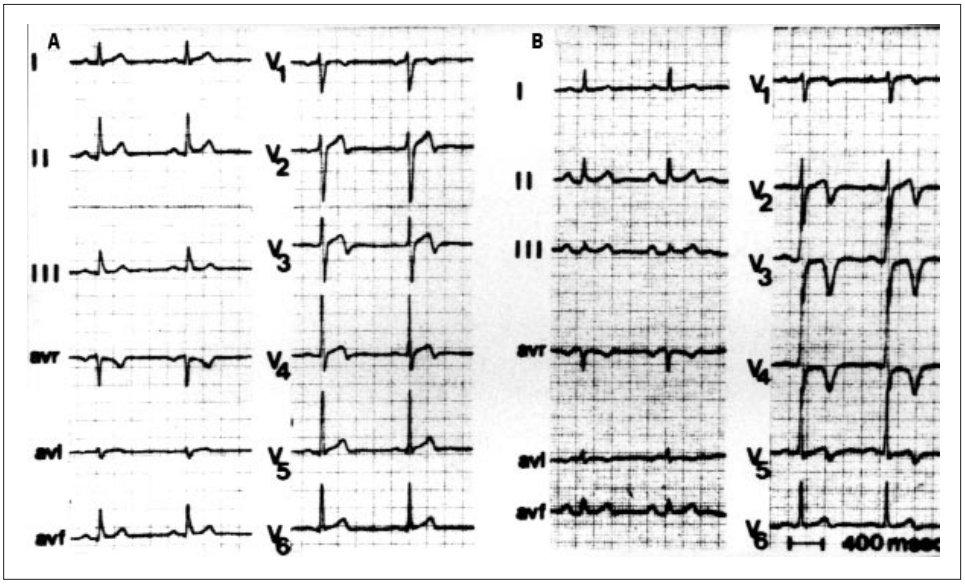

Figure 2.
*Wellens' waves. **A**, Less common pattern showing biphasic T waves in the precordial leads. **B**, More common pattern showing deeply inverted T waves in the precordial leads.*

From de Zwaan C, Bär FW, Janssen JH, et al. Angiographic and clinical characteristics of patients with unstable angina showing an ECG pattern indicating critical narrowing of the proximal LAD coronary artery. *Am Heart J* 1989;117:657-665. Used with permission.

- Biphasic T waves in leads V_2 and V_3 (Figures 1A and 2A) or symmetric, often deeply inverted T waves in leads V_2 and V_3 (Figure 2B).

Wellens' criteria are quite specific for left anterior descending artery disease. All of the patients (n=180) in his 1988 study had more than 50% narrowing of the left anterior descending artery (mean=85% narrowing) with complete or near-complete occlusion in 59%.[4]

Inverted T waves caused by myocardial ischemia are classically narrow and symmetric. However, many processes may cause T-wave changes and many times they are "nonspecific" changes. T-wave inversions may be caused by both Q-wave and non–Q-wave infarctions, as well as by noninfarctional causes such as myocarditis, pulmonary embolism, stroke, left ventricular hypertrophy, Wolff-Parkinson-White syndrome, juvenile T-wave patterns, digitalis, and others.[3] In the past, patients with the obvious, deep, symmetric T-wave inversions of Figure 2B have been referred to as having the "left anterior descending T-wave syndrome" and the need for aggressive treatment noted.[2,3] However, the biphasic T waves of Figures 1A and 2A, despite their importance, are subtle and their significance may be underappreciated. These T waves have a characteristic upsloping then sharply downsloping pattern leading into a T-wave inversion that seems to be different than the T-wave inversions of other causes. This particular pattern of biphasic T waves in leads V_2 and V_3 should alert the emergency physician of the potential for severe coronary disease. Unfortunately, the incidence, sensitivity, and specificity of this ECG finding in the ED chest pain population all await studies. In our experience, the incidence is low but the specificity is high for left anterior descending coronary artery lesions. Further studies may show that biphasic Wellens' waves are one example of a more generalized phenomenon found in T waves of other ECG leads with corresponding coronary lesions. Thorough discussions of T-wave changes are available in the literature for review.[6-8]

There are several other important caveats for the emergency medicine physician. The ECG abnormalities of Wellens' syndrome usually occur during a pain-free interval and for a limited period when other evidence of ischemia or angina may be absent. This is problematic in the "snapshot" ED setting or in a patient with an atypical history. In fact, when a patient has pain, the ST-T–wave abnormalities of Wellens' syndrome usually normalize or the ST segments are elevated.[4] The origin of the T-wave changes remains unclear, but they can be transient or persist for months and tend to resolve with appropriate intervention.

As chest pain centers evolve, the role of emergency physicians in the diagnosis and treatment of myocardial ischemia continues to expand. The Wellens' ECG pattern, especially the more subtle pattern in Figures 1A and 2A, represents a potential pitfall for emergency physicians attempting to evaluate patients in chest pain centers. The biphasic T-wave pattern of Wellens' syndrome in leads V_2 and V_3 may be unfamiliar to some emergency physicians who may diagnose them as being "nonspecific" ECG changes,[9] which they are not. A review of several chest pain center triage protocols reveals that a pain-free patient with "nonspecific" ECG changes, an atypical history, and normal cardiac enzyme levels may be triaged to a protocol culminating in an EST.[10-12] ESTs are contraindicated in the presence of suspected left main or left main–equivalent lesions.[13] As our case report illustrates, this test may have an unfortunate result in a patient with Wellens' syndrome. Instead, timely angiography should be considered in these patients and provocative testing, if done at all, should be done in conjunction with the cardiology department.

In the past, ECG changes similar to those presented have been diagnosed as nontransmural or subendocardial ischemia/infarction and these patients were offered conservative therapy. Accurate identification of patients with Wellens' syndrome allows more rapid and appropriately aggressive management. As chest pain centers evolve, it becomes imperative that the emergency physician recognize these patients to prevent prolonged ED stays, inappropriately conservative therapy, or ESTs. A patient with Wellens' syndrome may be destined for a massive anterior wall myocardial infarction, and must be managed accordingly.

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