



## Case Reports

### Pseudo-Wellens syndrome in a cocaine user

de Zwann et al [1] first described a characteristic electrocardiographic (ECG) pattern in the precordial leads that is associated with a critical stenosis of the proximal left anterior descending coronary artery (LAD). These changes, usually but not always, occur during a pain-free interval when other evidence of ischemia might be absent. All of the patients in their 1988 study had 50% or greater narrowing of the LAD [2]. Early angiography with subsequent stenting or coronary bypass surgery is now recommended for these patients. The distinctive ECG pattern and clinical findings suggest critical LAD stenosis with impending massive anterior wall infarction. It is frequently missed with the potential of fatal result [3]. The emergency physician (EP) might approach such a patient aggressively. However, as this case illustrates, there are other causes of these distinctive ECG findings.

A 46-year-old man presented to the ED describing several minutes of severe chest pain associated with palpitations, diaphoresis, and stomach cramps. He subsequently had a 45-minute episode. He did admit to smoking cocaine for the previous 2 days. He had a history of hypertension and was taking 20 mg of Lisinopril daily.

He used of cocaine, marijuana, and alcohol. Physical examination results were as follows: afebrile; pulse, 88; respirations, 18; blood pressure, 178/118; general, not presently in distress; neck, no jugular venous distension; lungs, clear; cardiovascular, regular, no extra heart sounds; abdomen, unremarkable; extremities, no edema; initial laboratory tests: complete blood cell count, electrolytes, and renals, normal. Urine screened positive for cocaine and tetrahydrocannabinol. Troponin was below 0.01 (negative  $\times 3$ ). Creatine kinase and creatine kinase-MB were negative. Initial ECG showed biphasic T waves in leads V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>, and V<sub>5</sub> (Fig. 1). The ECG reverted to normal after 48 hours (Fig. 2).

Patient was taken directly for cardiac catheterization, which showed an ejection fraction of 65% and completely normal coronary arteries.

This patient met the criteria of Wellens, which are 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, biphasic T waves in leads V<sub>2</sub> and V<sub>3</sub>, or symmetric, often deeply inverted, and T-waves in V<sub>2</sub> and V<sub>3</sub>, and was clearly a candidate for emergent cardiac angiography. However, the case is unique in that the patient had normal coronary arteries. The ischemia in the territory of the LAD was likely due

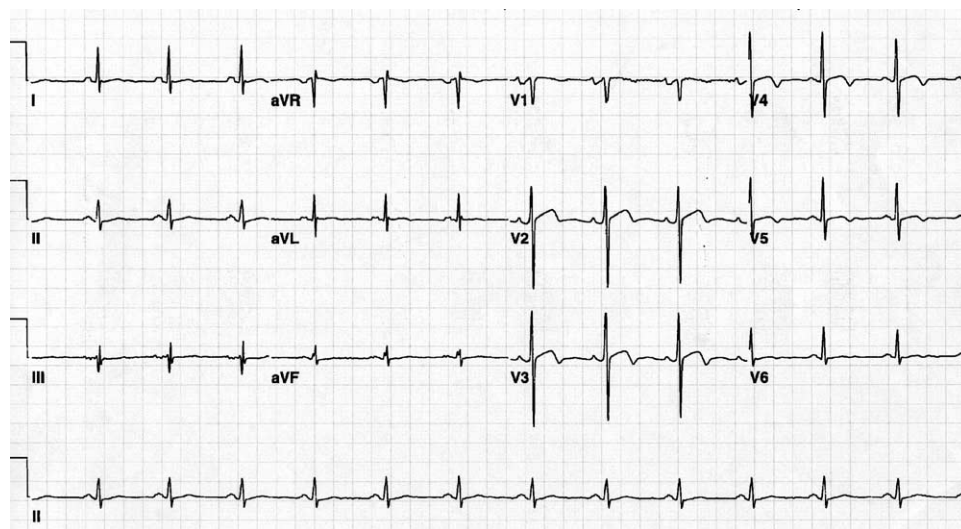
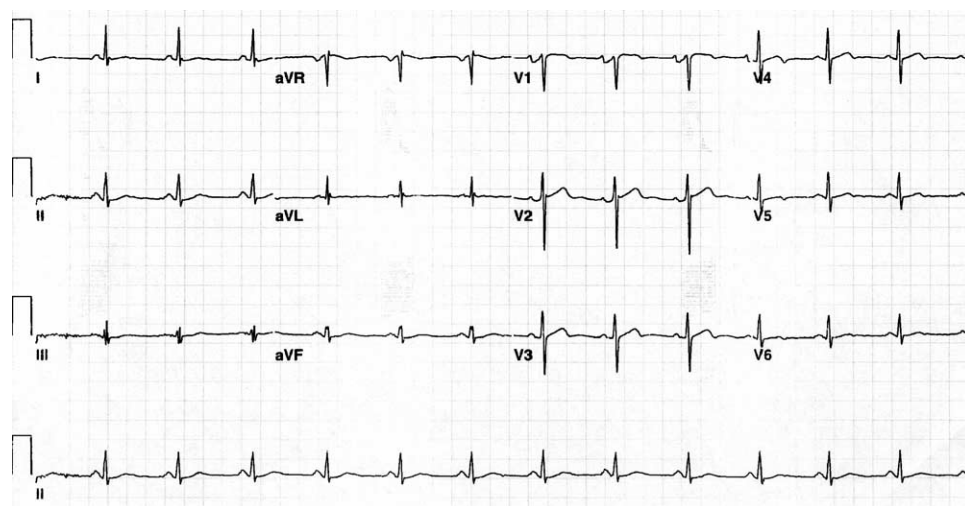


Fig. 1 Initial ED electrocardiogram.



**Fig. 2** Discharge electrocardiogram.

to cocaine-induced coronary artery spasm [4,5]. The EP must be alert that these characteristic ECG findings may be caused by nonobstructive coronary disease. If the EP is unaware of the cocaine etiology of these ECG findings, he might administer  $\beta$ -blockers, as one should with acute coronary syndrome, which could have disastrous results [6].

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## Subarachnoid hemorrhage in survivors of out-of-hospital cardiac arrest: true or not?<sup>☆</sup>

Subarachnoid hemorrhage (SAH) presenting with out-of-hospital cardiac arrest (OHCA) and abnormal electrocardiographic (ECG) findings may be delay- or miss-diagnosed with the prevalence of training programs for advanced cardiovascular life support, which strengthen acute coronary syndromes (ACSs). Unfortunately, most managements for ACS might worsen SAH. Moreover, the diagnosis of SAH may affect the application of therapeutic hypothermia. We reported 2 OHCA survivors both with increased attenuation of the subarachnoid space on early cranial computed tomography (CT) scans after resuscitation and discussed the application of cranial CT scans in identifying possible OHCA causes and guiding further managements.

A 66 year-old man with hypertension was sent to the ED as OHCA. Sudden collapse happened when he was having sexual intercourse. Ventricular fibrillation was converted during resuscitation. Return of spontaneous circulation (ROSC) was achieved after 21 minutes of cardiopulmonary resuscitation (CPR) with a Glasgow Coma Scale score of 3. Symmetrical pupil size without light reflex, areflexia, and hypotonia of all extremities were found. A 12-lead ECG demonstrated diffuse ST-segment depression in leads I, II, and V<sub>2</sub> through V<sub>6</sub>. Emergent cardiac catheterization was performed under the suspicion of ACS but found patent coronary arteries. Then, the cranial CT scan showed diffuse increased density in the subarachnoid space (Fig. 1). Subarachnoid hemorrhage was impressed, and neurosurgeon suggested conservative treatment. The patient died on the 10th day after the event.

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