

2023

Pseudo-Wellens syndrome: A rare entity associated with cocaine use

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Recommended Citation

Mohamed M, Waheed S, Mahmoud A, Hashem A, Baibhav B, Firoze Ahmed AM. Pseudo-Wellens syndrome: A rare entity associated with cocaine use. *Advances in Clinical Medical Research and Healthcare Delivery*. 2023; 3(3). doi: 10.53785/2769-2779.1164.

ISSN: 2769-2779

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Pseudo-Wellens syndrome: A rare entity associated with cocaine use

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Abstract

Wellens syndrome usually indicates critical left anterior descending artery (LAD) occlusion. Pseudo-Wellens syndrome consists of criteria of Wellens syndrome in the absence of critical LAD occlusion. We report a case of Pseudo-Wellens syndrome related to cocaine use. A 52-year-old male with a medical history of hypertension and diabetes, presented with acute retrosternal chest pain of 3 days duration. Physical examination was unremarkable. EKG on presentation showed deep T-wave inversions in leads V2 to V5. Highly sensitive troponin was elevated. The patient admitted to using cocaine daily for the past two months. Due to concerns for Wellens syndrome, the patient had an immediate coronary angiography which revealed mild disease of the LAD (< 30%) only. Inpatient echocardiogram revealed preserved left ventricular ejection fraction and no segmental wall motion abnormalities. Subsequent EKG at the cardiology clinic showed improvement in T-wave inversion. The patient was advised to abstain from using cocaine. As Pseudo-Wellens syndrome is a diagnosis of exclusion, patients with a history of recent cocaine use presenting with acute chest pain history, evidence of myocardial injury, and EKG findings suggestive of Wellens syndrome should undergo an emergent coronary angiogram to exclude critical LAD occlusion.

Keywords

Pseudo-Wellens syndrome, Wellens syndrome, Cocaine use, Myocardial infarction, Emergency Coronary Angiogram

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Conflict of Interest Statement

None

CASE REPORT

Pseudo-Wellens Syndrome: A Rare Entity Associated With Cocaine Use

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Abstract

Wellens syndrome usually indicates critical left anterior descending artery (LAD) occlusion. Pseudo-Wellens syndrome consists of criteria of Wellens syndrome in the absence of critical LAD occlusion. We report a case of Pseudo-Wellens syndrome related to cocaine use. A 52-year-old male with a medical history of hypertension and diabetes presented with acute retrosternal chest pain of 3 days duration. Physical examination was unremarkable. EKG on presentation showed deep T wave inversions in leads V2 to V5. Highly sensitive troponin was elevated. The patient admitted to using cocaine daily for the past two months. Due to concerns for Wellens syndrome, the patient had an immediate coronary angiography which revealed mild disease of the LAD (<30%) only. Inpatient echocardiogram revealed preserved left ventricular ejection fraction and no segmental wall motion abnormalities. Subsequent EKG at the cardiology clinic showed improvement in T wave inversion. The patient was advised to abstain from using cocaine. As Pseudo-Wellens syndrome is a diagnosis of exclusion, patients with a history of recent cocaine use presenting with acute chest pain history, evidence of myocardial injury, and EKG findings suggestive of Wellens syndrome should undergo an emergent coronary angiogram to exclude critical LAD occlusion.

Key words: Pseudo-Wellens syndrome, Wellens syndrome, Cocaine use, Myocardial infarction, Emergency coronary angiogram

1. Introduction

Wellens syndrome consists of biphasic or inverted T waves in leads V2 and V3, history of anginal chest pain, lack of Q waves, no loss of precordial R-wave progression, no significant ST-segment elevation on 12-lead electrocardiography (ECG), and mild or no serum cardiac marker elevation.¹ However, 12% of patients with Wellens syndrome can have elevated cardiac enzymes.² Wellens syndrome is often caused by critical stenosis of the proximal left anterior descending (LAD) coronary artery.¹ Pseudo-Wellens syndrome is diagnosed when the criteria for Wellens syndrome is met but in the absence of a critical LAD lesion. We present a rare case of Pseudo-Wellens syndrome secondary to cocaine use.

2. Case presentation

52 years old male patient with a medical history of hypertension, diabetes, and cocaine use presented to emergency department with retrosternal chest pain. Chest pain was acute in onset, progressive, radiating to the left arm and back, heaviness in character, and associated with diaphoresis. The patient was hemodynamically stable and physical examination was unremarkable. His ECG showed deep T wave inversions in leads V2 through V5 without ST-segment elevation or Q waves – consistent with Wellens syndrome (Fig. 1). His labs were significant for elevated high-sensitivity cardiac troponin at 0 h and 1 h. Urine drug screening was positive for cocaine.

After receiving 325 mg of aspirin, the patient was taken for emergent coronary angiography due to significant cardiovascular risk factors, ECG changes,

Accepted 6 February 2023.
Available online ■■■

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<https://doi.org/10.53785/2769-2779.1164>
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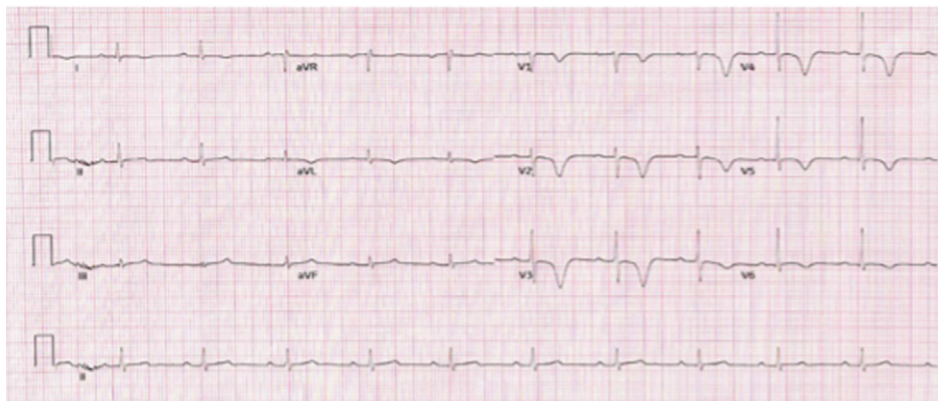


Fig. 1. EKG showing deep symmetrical T wave in leads V2 to V5.

and significant troponin elevation. However, coronary angiogram only revealed mild disease (<30%) of the LAD with no disease in the other coronary arteries (Fig. 2). An echocardiogram showed normal left ventricle dimensions and functions with a left ventricle ejection fraction of 60% and no segmental wall motion abnormality. Given the patient's recent cocaine use, typical anginal chest pain, elevated cardiac enzymes, EKG changes, and normal coronary angiogram, the patient was diagnosed with Pseudo-Wellens syndrome secondary to cocaine use.

The patient was discharged on aspirin and atorvastatin. As the use of a beta blocker agent can worsen coronary vasospasm due to the unopposed alpha-adrenergic effect due to his active cocaine use, the patient was not prescribed a beta blocker to avoid precipitating chest pain.³ The patient was

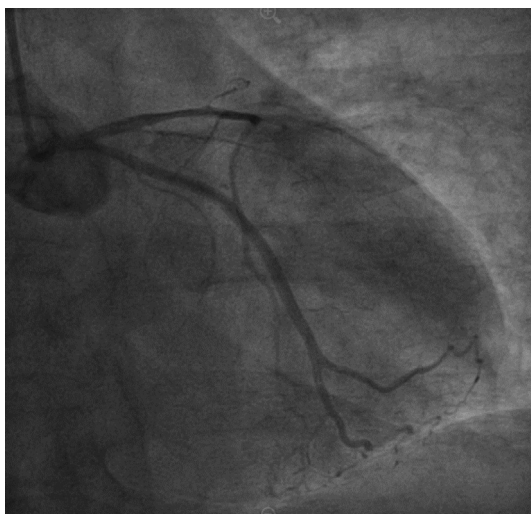


Fig. 2. Coronary angiogram of left coronary system showing Normal LAD.

advised to abstain from cocaine use. Two months later, the patient was seen in the cardiology clinic. The patient did not report chest pain or shortness of breath. Repeat ECG (Fig. 3) showed significant improvement of the T wave inversions in comparison with the ECG the patient had on admission two months ago.

3. Discussion

Wellens syndrome is sub-classified to type A (biphasic T wave in V2–V3) and type B (deep symmetrical T wave inversions in V2–V3).¹ Wellens syndrome usually indicates critical stenosis at the proximal part of LAD coronary artery.¹ Pseudo-Wellens syndrome mimics Wellens syndrome but without critical stenosis of LAD.⁴ Causes of Pseudo-Wellens syndrome include drug use [eg. cocaine, cannabis,⁴ and phencyclidine⁴], pulmonary embolism,⁵ left ventricle hypertrophy due to hypertension,⁶ and severe sepsis.¹

The exact mechanism of Pseudo-Wellens syndrome is still unknown. One possible mechanism is intermittent vasospasm causing transient myocardial ischemia and EKG changes. In our case, cocaine may have caused vasospasm of the LAD coronary artery leading to myocardial ischemia and injury.⁷ Cocaine blocks the presynaptic uptake of catecholamines causing vasoconstriction of the coronary arteries leading to decreased myocardial perfusion.⁷ Cocaine has a positive inotropic and chronotropic effect increasing the myocardial oxygen demand.⁷ This demand-supply mismatch induces chest pain and EKG changes. In patients with chest pain associated with cocaine use, 56%–86% of them have an abnormal EKG but myocardial infarction occurs in only 0.7%–6%.⁸

Differentiating between Wellens syndrome and Pseudo-Wellens syndrome is a clinical dilemma.

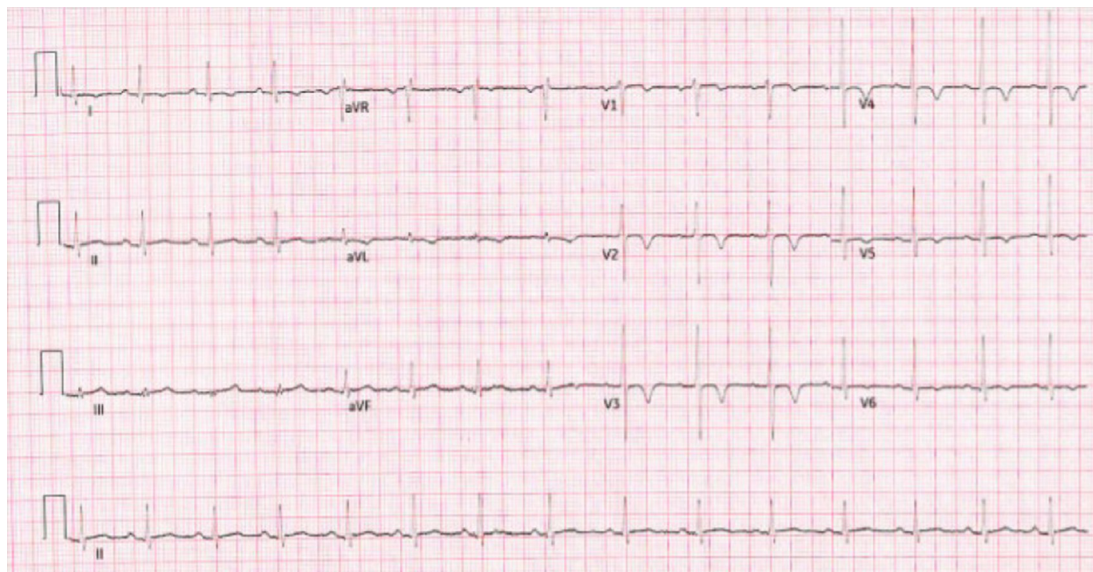


Fig. 3. EKG showing improvement of T wave inversions.

Only coronary angiography can differentiate between the two conditions. Hence, all patients with EKG findings suggestive of Wellens syndrome must be managed as an acute coronary syndrome until coronary artery disease is ruled out.⁷

Acute management of Pseudo-Wellens syndrome due to cocaine abuse includes benzodiazepines to relieve acute distress and calcium channel blockers to relieve coronary vasospasm. Unlike in acute coronary syndrome, beta blockers are contraindicated as unopposed alpha-adrenergic receptors activation can worsen the coronary vasospasm and myocardial ischemia.³ Long-term management includes abstaining from cocaine and other recreational drugs.

Funding

None.

Conflict of interest

None.

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