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Case Presentation

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Wellens' Syndrome: Importance of Recognizing Specific EKG Patterns in Atypical Chest Pain: A Case Report

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Abstract

Wellens' Syndrome is an abnormal electrocardiogram reading that is important to identify because the severity of a patient's condition is often undermined by their presentation, as they are often without severe symptoms. These patients can present with normal or mildly elevated troponins, however they are at high risk for complications due to severe left anterior descending artery stenosis. This case of a 77-year-old male underscores the importance of recognizing Wellens' syndrome in patients presenting with atypical or resolved chest pain. This patient was taken for cardiac catheterization and showed significant stenosis in multiple cardiac arteries, and he improved following the procedure. Characteristic anterior T-wave abnormalities on electrocardiography should warrant urgent coronary angiography rather than conservative management or stress testing. Early diagnosis and revascularization are crucial to prevent progression to potentially lethal anterior wall myocardial infarction.

Keywords: Acute coronary syndrome; Atypical chest pain; Critical coronary artery stenosis; Electrocardiogram; Wellens's syndrome

Abbreviations & Acronyms: Wellens' Syndrome (WS); electrocardiogram (EKG); Acute Coronary Syndrome (ACS); Coronary Artery Disease (CAD); Hypertension (HTN); Benign Prostatic Hyperplasia (BPH); Percutaneous Coronary Intervention (PCI); Right Anterior Oblique (RAO); Spontaneous Coronary Artery Dissection (SCAD)

Introduction

Wellens' Syndrome (WS) is described as an abnormal Electrocardiogram (EKG) reading that shows deeply inverted T waves in leads V2 and V3, which are secondary to proximal LAD stenosis [1]. It was first described by de Zwaan and Wellens in 1982 [2]. The typical patient presentation is often unassuming, as they report no pain, and cardiac enzymes can even be normal or slightly elevated. It is important to be able to recognize this pattern because patients with WS are at a high risk for myocardial infarction of the anterior wall and subsequent fatal complications. Incidence of WS is quite low, ranging from 10-15% of Acute Coronary Syndrome (ACS) [3]. While the mechanism for WS is largely unknown, the risk factors are similar to those of traditional Coronary Artery Disease (CAD), including obesity, smoking, Hypertension (HTN), and more. It is important to recognize the clinical presentation and imaging features associated with WS, as this can often lead to death if left untreated. This case report aims to bring attention to the clinical features of patients who present with WS for more accurate diagnosis and faster initiation of treatment.

Case Presentation

A 77-year-old male with a past medical history of CAD, HTN, and Benign Prostatic Hyperplasia (BPH) presented to the emergency department with 2-3 weeks of intermittent, non-radiating, exertional substernal chest pain associated with some shortness of breath that resolves with rest. He has no smoking history and does not drink alcohol or use illicit drugs. His family history is significant for atherosclerotic cardiovascular disease in his father. He is very active and walks 3 miles a day. On arrival at the emergency department, he was asymptomatic. On physical examination, he was noted to have a regular S1 and S2 with an S4. There was also a grade I/VI basal systolic ejection murmur. His lipid profile was notable for elevated triglycerides of 190 mg/dL and low HDL of 25 mg/dL.

An EKG was performed, and it showed sinus bradycardia, first-degree AV block, nonspecific ST and T wave abnormalities, and voltage criteria for left ventricular hypertrophy. He was ruled in for an anterior wall acute non-ST-segment elevation myocardial infarction with 4 elevated high-sensitivity troponin levels, all in the same range. A transthoracic echocardiogram was performed and showed preserved left ventricular ejection fraction estimated at 65-70% and mild interventricular septum thickening. A chest X-ray was done as well, which was unremarkable. He was given high-dose aspirin and intravenous heparin and recommended for a cardiac catheterization **(Figure 1)**.

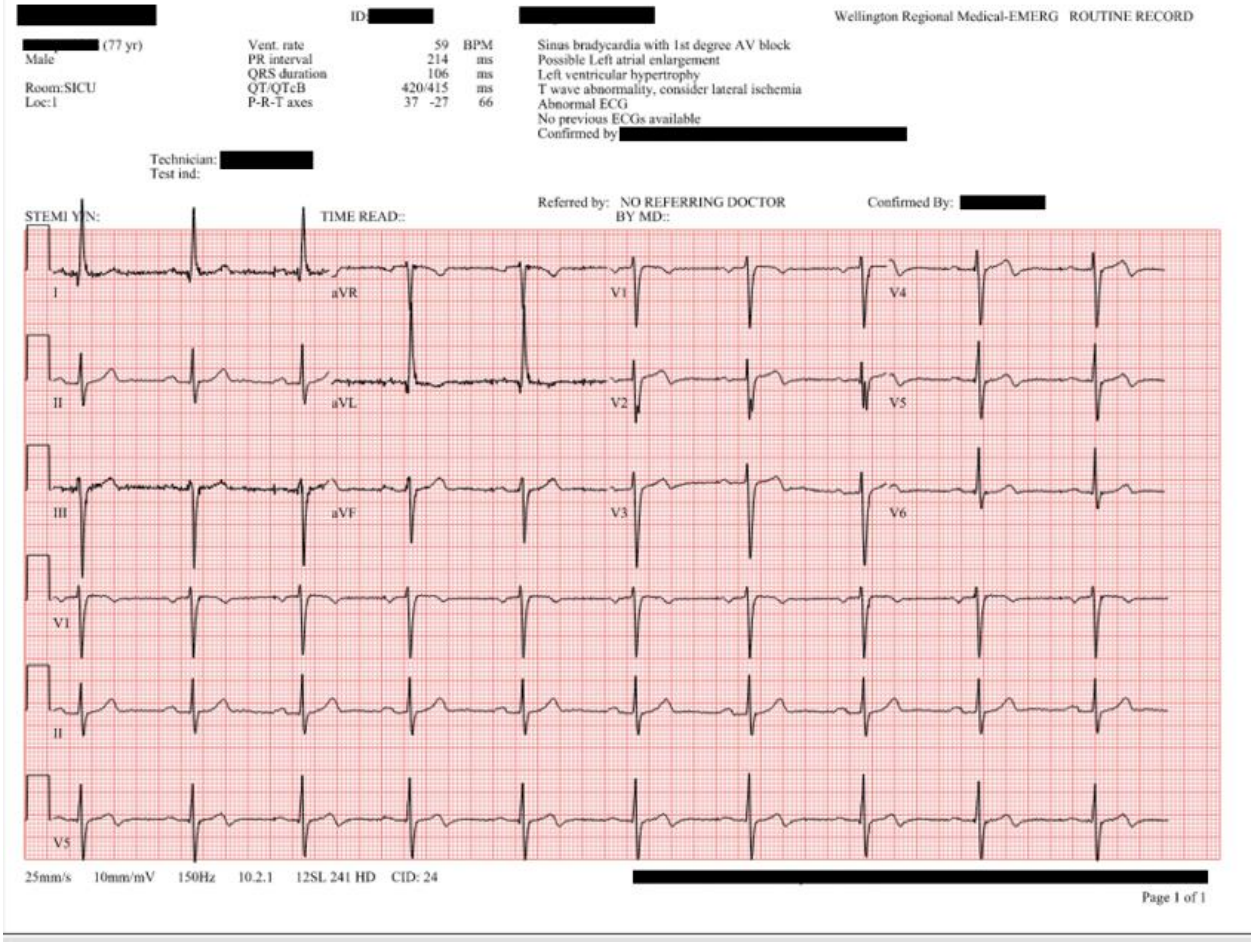

Figure 1: EKG revealing ST and T wave abnormalities.

The patient was taken for a cardiac catheterization the following morning. Just proximal to the diagonal artery, there was an eccentric, irregular, ulcerated, 90% discrete stenosis. The distal left anterior descending coronary artery, after 2 tortuous angulated segments, contained an 80% stenosis. The left circumflex coronary artery branched into a moderate-sized left posterolateral branch and a large left posterior descending coronary artery. The right coronary artery is a small-caliber non-dominant vessel. It was 100% occluded in its proximal portion. Left ventriculography was performed in the Right Anterior Oblique (RAO) projection and revealed overall well-preserved left ventricular systolic function with left ventricular hypertrophy. There was mild mid-anterior wall hypokinesis. The overall ejection fraction was in the 65% range with 1+ mitral regurgitation.

The left main coronary artery was engaged using a guiding catheter. Due to the 2 significant mid angulations, the wire could not be directed into the very distal portion of the left anterior descending coronary artery, so the 80% distal stenosis could not be treated with Percutaneous Coronary Intervention (PCI). The wire would only repeatedly go into the diagonal branch and not the left anterior descending coronary artery distally. Therefore, the attention was focused on the proximal ulcerated culprit lesion.

Intravascular ultrasound interrogation was performed to help in stent sizing. Based on the angiography and the intravascular ultrasound, a 4.5 x 12 mm Medtronic Resolute drug-eluting stent was chosen, which was deployed in the proximal vessel at high pressures. Post-dilation was performed with a 4.5 x 8 mm NC Euphora balloon in the proximal portion of the stent. A 0% residual stenosis was the result with Thrombolysis in Myocardial Infarction (TIMI) grade III flow into the distal vessel.

The patient recovered well from the procedure and was discharged the following day. He was advised to continue antiplatelet medications, aspirin and ticagrelor, and statin medication atorvastatin, and to follow up with his cardiologist **(Figure 2)**.

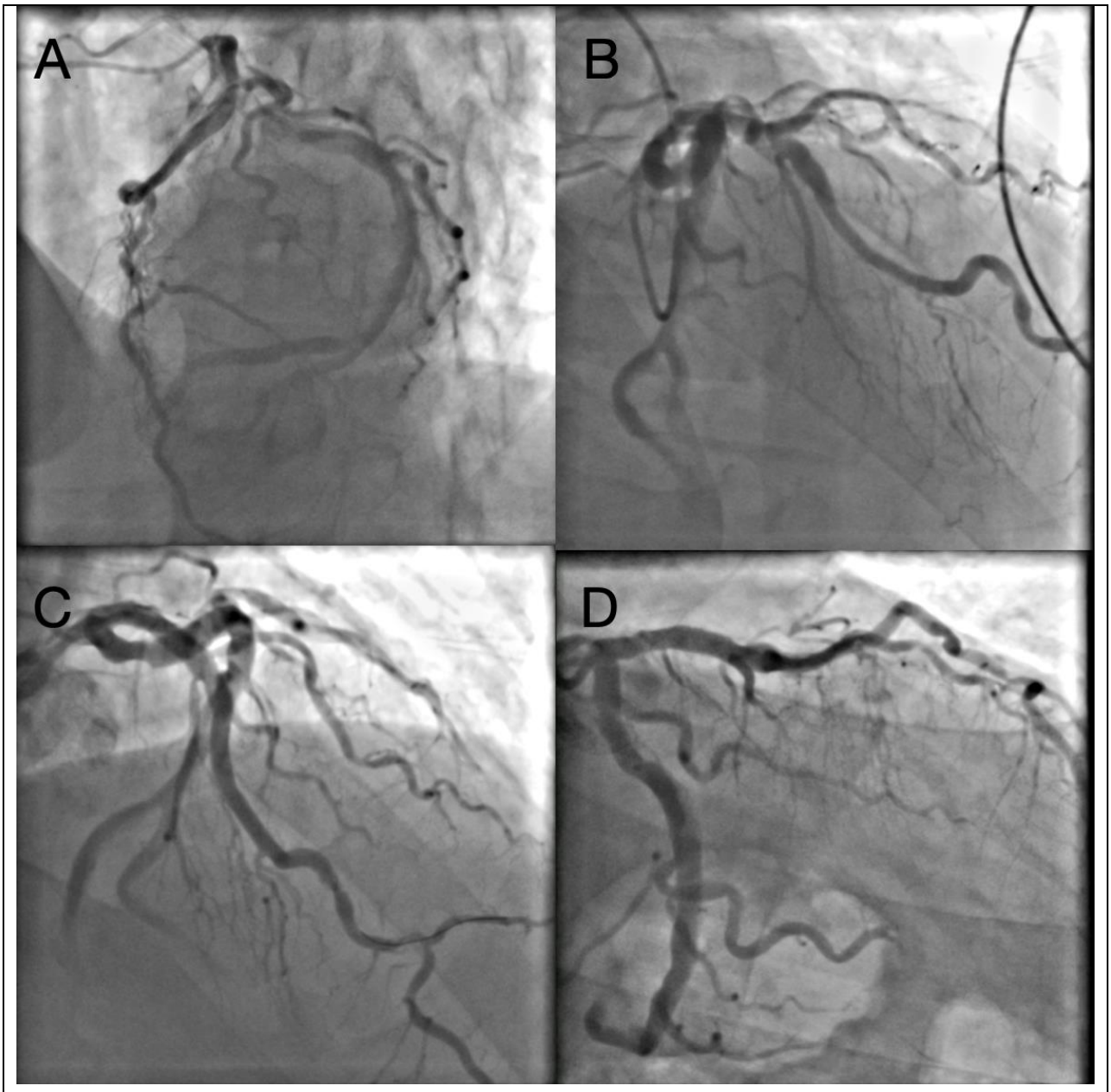

Figure 2: Images from cardiac catheterization procedure.

Discussion

WS is generally characterised by a history of angina, minimal or absent ST-segment elevation, normal or only mildly elevated cardiac biomarkers, preserved R-wave progression, and, most recognisably, either biphasic ("Type A") or deeply inverted ("Type B") T-waves in the anterior precordial leads [4]. In our patient, the ECG findings aligned with the classical description of WS, underscoring its diagnostic importance. Recognition of the characteristic T-wave morphology is paramount because conservative medical management alone is associated with a high risk of progression to frank anterior MI: early series reported that up to 75% of such patients developed anterior MI within days if revascularization was not undertaken [5].

From a pathophysiological standpoint, WS appears to reflect a state of unstable myocardial ischaemia due to a critical but not yet occlusive LAD lesion. The transient relief of chest pain in many patients, often at the time the ECG is recorded, suggests that the T-wave abnormalities may represent reperfusion or intermittent occlusion, followed by ischaemic myocardium that remains at risk [6]. The diagnostic criteria of WS emphasise several features: (1) recent history of angina, (2) minimal or no ST elevation, (3) normal or near-normal cardiac enzymes at presentation, (4) typical T-wave changes, and (5) preserved R-wave progression without pathologic Q-waves [6]. In the current case, the presence of symptoms and findings strongly supported the diagnosis of WS. Given the life-threatening potential of progression to infarction, early coronary angiography and revascularization are recommended over stress testing alone, which may provoke infarction or arrhythmias [5].

Recent literature suggests that WS may be more common than historically appreciated. A descriptive cross-sectional study found that WS criteria were fulfilled in approximately 15% of patients presenting with ACS, and notably, up to 14–18% in certain cohorts [7]. That study also highlighted that WS can present even in the absence of classic coronary risk factors and that stenosis may be located in the mid-LAD or even involve other vessels, not only the proximal LAD [8]. Therefore, our case contributes to the growing recognition that WS must be considered in a broad spectrum of patients, including those with atypical risk profiles or younger age.

Therapeutically, prompt PCI in patients with WS has shown favourable outcomes. For example, a recent case report documented urgent PCI in a 71-year-old male with type 2 WS (deep T-wave inversion in V1–V4) with excellent recovery [9]. Our case similarly underwent cardiac catheterization, with a resultant 4.5 x 12 mm drug-eluting stent, which was deployed in the proximal LAD at high pressures. Post-dilation was performed with a 4.5 x 8 mm NC Euphora balloon in the proximal portion of the stent. A 0% residual stenosis was the result with thrombolysis in myocardial infarction (TIMI) grade III flow into the distal vessel, emphasising the critical nature of timely invasive management. Beta-blocker therapy was initiated at discharge to conform with guideline-directed medical therapy for ACS despite no history of beta-blocker prescription and first-degree atrioventricular block, as he remained hemodynamically stable without higher-grade conduction abnormalities, supporting safe implementation of guideline-directed medical therapy [10].

It is also important to acknowledge limitations and complexities. First, WS may not always present fully

classically: some patients may have non-specific T-wave changes, may present with ongoing pain, or may have biomarker elevation [11]. Second, there are recognized "pseudo-Wellens" phenomena, including etiologies such as cocaine-induced spasm, myocardial bridging, Takotsubo cardiomyopathy, and Spontaneous Coronary Artery Dissection (SCAD), in which the ECG pattern mimics WS but the underlying pathology differs [12]. Therefore, it is essential to interpret the ECG in the full clinical context, including risk factors, biomarker trends, and imaging.

In summary, the present case underscores several key teaching points relevant to WS: the necessity of high clinical suspicion when characteristic T-wave abnormalities appear in a patient with chest pain; the danger of misclassifying the presentation as benign due to normal biomarkers or absence of ST-elevation; and the imperative for urgent angiographic assessment and revascularization to prevent a large anterior MI. Recognition of WS remains a critical responsibility for clinicians evaluating chest pain, and this case further expands the evidence base by showing that symptoms may be minimal even with multiple arteries that are severely occluded. Future work should aim to refine protocols for rapid identification and management of WS, including exploring the prognostic significance of T-wave evolution and the outcome of different revascularization strategies.

Conclusion

This case highlights the critical importance of early recognition and management of WS in patients presenting with atypical chest pain. Even in the absence of ongoing symptoms or markedly elevated cardiac biomarkers, the presence of characteristic T-wave changes in the anterior precordial leads should immediately raise concern for a high-grade lesion in the left anterior descending artery. Prompt diagnosis and urgent cardiac catheterization can prevent progression to a potentially fatal anterior wall myocardial infarction.

Our patient's presentation reinforces that WS may not always manifest with classic symptoms or risk factors. The combination of exertional chest discomfort, transient symptom relief, and specific EKG findings underscores the need for heightened clinical vigilance. Misinterpretation of these patterns as benign can delay intervention and result in catastrophic outcomes.

This case also emphasizes the value of integrating EKG pattern recognition into standard chest pain protocols. Clinicians should maintain a high index of suspicion for WS in patients with recent angina episodes and typical T-wave inversions, even if pain-free at presentation. Early angiography and revascularization remain the cornerstones of management, as medical therapy alone is insufficient to prevent infarction.

In summary, WS represents a unique yet under recognized clinical entity that demands prompt identification and decisive management. This case adds to the growing awareness that vigilance in interpreting subtle EKG changes can directly translate into life-saving outcomes.

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