



## LETTER TO THE EDITOR

## Myocardial bridge as a cause of pseudo-Wellens' syndrome



## KEYWORDS

pseudo-Wellens' syndrome;  
Wellens' syndrome;  
myocardial bridge

In 1982, Wellens and colleagues identified a group of patients with a history of angina and characteristic ECG changes in the precordial T waves.<sup>1</sup> This pattern was named Wellens' syndrome and has been associated with critical stenosis of the proximal left anterior descending (LAD) artery, signifying an imminent risk of an anterior wall myocardial infarction (MI).<sup>2</sup> Myocardial bridging, on the other hand, is a congenital abnormality of a coronary artery (the vast majority concerns the LAD), in which a segment of the epicardial coronary artery takes an intramuscular course.<sup>3</sup> Myocardial bridging may be associated with exertional angina, acute coronary syndrome (ACS), cardiac arrhythmias, syncope, and sudden cardiac death.<sup>3</sup> As ACS may present with several forms,<sup>4</sup> in this paper, we report the case of a 55-year-old male with a myocardial bridge who presented with a characteristic Wellens' ECG.

A 55-year-old man, with no history of coronary artery disease (CAD), presented to the emergency department (ER) due to recurrent episodes of retrosternal pain during the last 3 days. The episodes lasted 10 to 15 minutes and were accompanied by sweating. The patient reported that the symptoms occurred mainly during exercise or under psychological stress. His medical history was significant for hypertension and dyslipidemia. He was an ex-smoker, denied any illicit drug use, and had no family history of CAD or sudden death. He had visited a cardiologist 3 days before his presentation to the ER, after experiencing the first episode of pain. At that time, the ECG performed during chest pain had no signs of ischemia (Figure 1a). However,

3 days later, the patient had a new episode of chest pain. The patient visited his cardiologist again, and a new ECG was performed, which showed signs of ischemia (Figure 1b), even though the pain had subsided. Therefore, the patient was immediately referred to our hospital. On physical examination, he had no fever, his blood pressure was 160/80 mmHg, his heart rate was 80 beats/min, and his cardiopulmonary auscultation was insignificant. The ECG was almost identical to the one performed a few hours before his arrival at the ER (Figure 1c). Echocardiographic evaluation revealed normal left ventricular contractility (ejection fraction: 60%), with no regional wall motion abnormalities. Laboratory tests were within normal range, except for a small increase in white blood cells. High-sensitivity troponin I was also within normal limits. Because of the high probability of Wellens' syndrome, the patient underwent coronary artery angiography (CAA), which demonstrated normal coronary arteries, except for the presence of a myocardial bridge in the middle segment of the LAD (Figure 1d, 1e, and Supplementary Videos 1–4). The patient was discharged the next day with a Ca-channel blocker and instructions to avoid intense physical exercise and psychological stress.

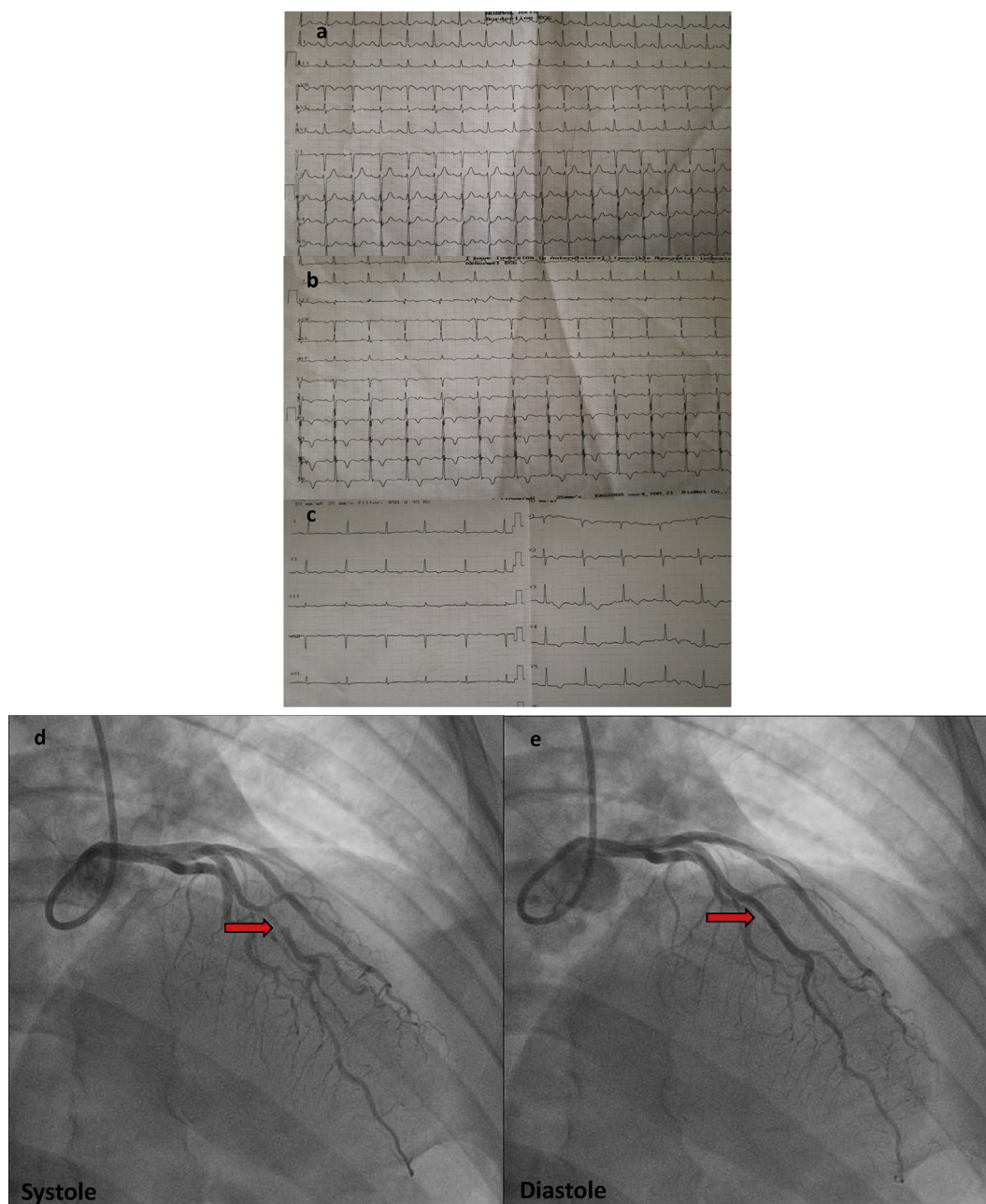
Supplementary video related to this article can be found at <http://dx.doi.org/10.1016/j.hjc.2017.07.003>

Wellens' syndrome is a pattern of electrocardiographic T-wave changes in patients with a history of angina and is associated with critical, proximal LAD stenosis.<sup>1</sup> Criteria for Wellens' syndrome include (a) history of anginal chest pain, (b) minimal or no elevation of cardiac enzymes, (c) no significant ST segment elevation (<1 mm), (d) no pathological precordial Q waves, (e) no loss of precordial R-wave progression, and (f) deeply inverted or biphasic T waves mainly in leads V2 and V3 and sometimes in leads V1, V4, V5, and V6.<sup>2</sup> This pattern is present in 14–18% of patients admitted with angina.<sup>2</sup> It is worth noting that the ECG changes occur in a pain-free period in most of the patients. There are two types of Wellens' syndrome<sup>5</sup>: Type I, which is recognized by biphasic T waves in precordial leads V2 and V3 and represents 24% of Wellens' syndrome, and Type II in 76% of the cases, with deeply inverted T waves mainly in

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**Figure 1** (a) Initial ECG during chest pain. There are no signs of ischemia. (b) ECG on second cardiologist visit. The patient is now free of pain, but the ECG is suspicious for Wellens' syndrome with deeply inverted T waves in V1–V6. (c) ECG on emergency department presentation. Same findings as shown in Figure 1b. (d) Presence of myocardial bridge in the mid-segment of LAD (red arrow). Systole. (e) Presence of myocardial bridge in the mid-segment of LAD (red arrow). Diastole. LAD: Left Anterior Descending

leads V2 and V3. Patients with Wellens' syndrome require early CAA and intervention because of the inherent high risk of extensive MI and sudden death. The risk results from the fact that up to 86% of patients who meet Wellens' syndrome criteria have a significant (>70%) LAD stenosis, with complete or near-complete occlusion in 59%.<sup>6</sup> Therefore, the recognition of this pattern is especially important because it is a preinfarction condition, heralding imminent catastrophic results, should it be underestimated by clinicians, especially those working in the ER.

Myocardial bridge is mostly benign; however, in some cases, it can be responsible for ACS, cardiac arrhythmias, syncope, or sudden cardiac death.<sup>3</sup> Patients who experience symptoms due to myocardial bridging have usually good response to medical therapy with beta blockers and non-dihydropyridine Ca-channel blockers.<sup>3</sup> Surgical therapy (myotomy or CABG) seems to be safe and effective in symptomatic patients with myocardial bridging refractory to maximal medical therapy.<sup>3</sup> However, ischemia-guided revascularization using drug-eluting stents may be

considered on a case-by-case basis for symptomatic patients refractory to maximal medical therapy and who are not optimal surgical candidates because medical therapy appears to be superior to percutaneous coronary intervention.<sup>3</sup>

The pseudo-Wellens' syndrome caused by coronary artery spasm, mostly due to illicit drug use, has also been rarely reported in the literature.<sup>7-10</sup> Severe stenosis is not the only cause of coronary flow interruption; vasospasm could have the same effect, thus causing characteristic T-wave changes.<sup>10</sup> Given that myocardial bridging increases the risk of coronary spasm,<sup>11</sup> we postulate that our patient presented with a characteristic Wellens' ECG due to coronary spasm that affected the blood flow through the pre-existing myocardial bridge.

To our knowledge, this is the first report of a myocardial bridge mimicking Wellens' syndrome. In the case presented, our patient met all the Wellens' syndrome criteria, but CAA revealed the presence of a myocardial bridge in the middle segment of LAD, and no intervention was required.

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## Disclosures

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