

Inferior ST-Elevation on the ECG

What Is the Diagnosis?

ECG CHALLENGE

A 62-year-old woman presented to another hospital with a 36-hour history of uninterrupted, nonspecific chest pain. Her medical history included long-standing hypertension and diabetes mellitus for which she took metoprolol, perindopril, felodipine, metformin, and glimepiride. She had stopped smoking 10 years earlier, and did not drink alcohol or use illicit drugs. The ECG on the admission to the other hospital is shown in Figure 1. Laboratory results at the other hospital were all normal, with the exception of mild hyperglycemia; cardiac troponin I was also normal. Based on the ECG in Figure 1, the patient was transferred to our unit for emergency coronary angiography. Clinical examination on admission to our hospital was unremarkable. A repeated ECG on admission in our hospital is disclosed in Figure 2. Based on the history of uninterrupted chest pain in a patient with cardiovascular risk factors and the aspect of the ECG in Figure 1, the on-call cardiology team in our unit agreed with the indication for emergency coronary angiography. This, however, disclosed no significant coronary artery disease. Over the next 48 hours, serial ECGs remained unchanged (Figure 2), and repeated blood tests (including serial troponin I levels) remained unremarkable. Echocardiography disclosed mild left ventricular hypertrophy with preserved ejection fraction and no wall motion abnormality.

What is the most likely explanation of the ECG in Figure 1?

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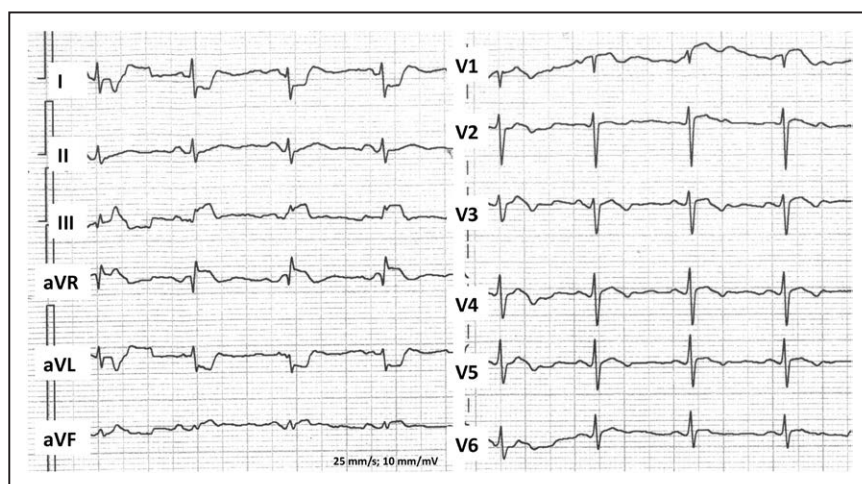


Figure 1. Index ECG performed at the other hospital.

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Figure 2. Follow-up ECG in our center.

Please turn the page to read the diagnosis.

RESPONSE TO ECG CHALLENGE

The most likely explanation for the index ECG of the patient (Figure 1) is artefactual ST-segment deviation mimicking ST-segment-elevation myocardial infarction.

In the limb leads, ST-segment deviation occurs only for the first ≈ 120 ms, whereas the last part of the ST segment is not deviated. The transition between these 2 distinct parts of the ST segment (first deviated, then nondeviated) is sharp and nonphysiological (Figure 3, arrows). Also, similarly timed nonphysiological deflections are present in all precordial leads (Figure 3, arrowheads). However, these abnormal deflections are absent and the ST segment is completely normal in lead II (Figure 3, box). There are also other signs of artefacts, such as sharp nonphysiological deflections of opposite polarities in opposite leads (Figure 3, asterisk), which are also absent in lead II. This ECG pattern, namely abnormal ST-segment deflections in all leads except 1 limb lead, is highly suggestive of a malfunctional limb electrode (in this case, the left-arm electrode).¹

The Einthoven triangle is defined by 3 equal bipolar limb leads (I, II, and III). At its center (the Wilson central terminal), the sum of the measured voltages is almost zero. Thus, the Wilson central terminal is used as a neutral electrode for all unipolar leads (aVL, aVR, aVF, and all precordial leads). A malfunctional limb electrode (eg,

microfractures, interrupted cable, poor electrode contact) may cause sharp changes in electric impedance, thus impairing the filtering processes of the ECG machines and distorting electric signals.² These distorted signals may manifest as phase shifts that become more apparent after high-amplitude signals (such as ST deviation after QRS complexes).^{1–3} Because the Wilson central terminal is used to generate the signals of all unipolar leads, the artefacts would be reflected in all leads except the bipolar lead that does not use the malfunctional electrode (in this case, lead II does not use the presumably malfunctional left-arm electrode).^{1,3}

The nonspecific stable T-wave changes seen across the precordial leads of the follow-up ECGs in this patient (Figure 2) may represent repolarization abnormalities in the context of mild left ventricular hypertrophy and long-standing diabetes mellitus.

ECG signs suggesting artefactual ST-segment deviation mimicking ST-segment-elevation myocardial infarction must be recognized by physicians to avoid: (1) the significant risks associated with contemporary invasive cardiology procedures and treatments (such as coronary angiography), and (2) establishing in error severe diagnoses (eg, acute coronary syndrome with normal coronary arteries) that may also adversely impact the social life of the patients (eg, driving, employment, health, and life insurances).

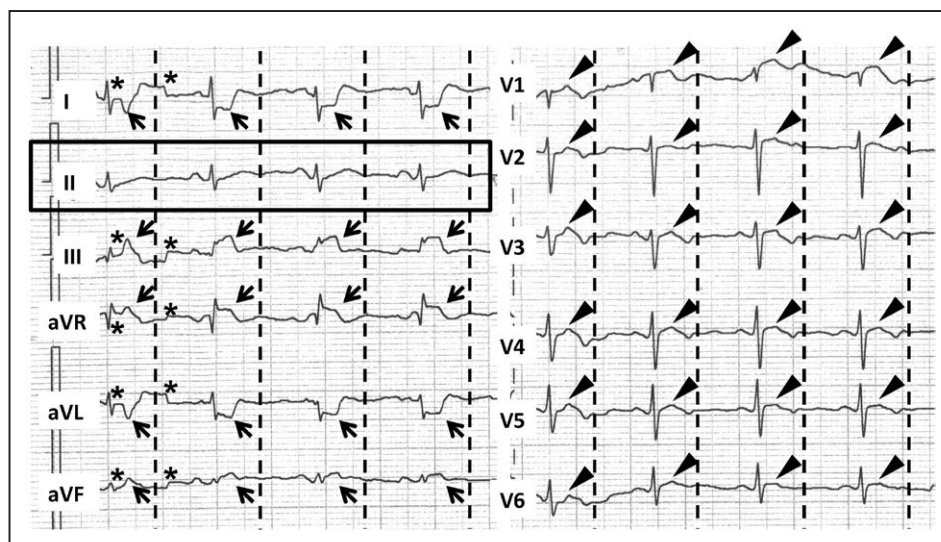


Figure 3. Artefactual ST-segment elevation mimicking ST-segment-elevation acute myocardial infarction.

In the limb leads, ST-segment deviation lasts only ≈ 120 ms, an interval that is too short to account for the entire ST segment, because it would correspond to a QTc interval of ≈ 230 ms. However, the QTc interval is normal (≈ 450 ms), as can be inferred from lead II (vertical dashed lines mark the end of the T wave). Thus, it becomes apparent that, in the limb leads, the ST segment incorporates a deviated and nondeviated part, and that the transition between these distinct parts of the ST segment is sharp and nonphysiological (arrows). In addition, similarly timed nonphysiological deflections of the ST segment are also present in all precordial leads (arrowheads), and other sharp nonphysiological deflections of opposite polarities are also present in opposite leads (asterisk). In lead II, however, the tracing is completely normal, with no abnormal deflections seen in any of its components, including the ST segment (box). The ECG pattern of abnormal deflections (most notably ST-segment deviations) in all leads except 1 limb lead is highly suggestive of a malfunctional limb electrode. See text for further details.

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DISCLOSURES

None.

FOOTNOTES

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