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IMAGING VIGNETTE

BEGINNER

ECG CHALLENGE

Not Every ST-Segment Elevation Is a STEMI

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ABSTRACT

Electrocardiogram is fundamental to diagnose ST-segment elevation myocardial infarction in patients with chest pain, other consistent symptoms, and/or echocardiographic abnormalities. Nevertheless, physicians should remember that other conditions can cause an ST-segment elevation myocardial infarction–like electrocardiogram. We exemplify this warning describing the case of an electrocardiogram mimicking ST-segment elevation myocardial infarction caused by severe dysionia. (**Level of Difficulty: Beginner.**) (J Am Coll Cardiol Case Rep 2021;3:283–5) © 2021 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 77-year-old woman was admitted to the emergency department for diarrhea and vomiting in the previous days. She had dyslipidemia and noninsulin-dependent diabetes mellitus, but no cardiovascular history. Her therapy included rosuvastatin, metformin, and low-dose aspirin in primary prevention. On admission, the patient was asymptomatic. Blood pressure was 140/70 mm Hg, heart rate was 75 beats/min. Physical examination was unremarkable. However, the electrocardiogram (ECG) showed ST-segment elevation in leads I, aVL, and V_2, and ST-segment depression in the inferior and precordial leads. Widespread T-wave alterations were also noted (**Figure 1A**). Transthoracic echocardiography demonstrated preserved left ventricular function with neither regional wall motion abnormalities (RWMA) nor significant valve disease. Complete blood count and levels of C-reactive protein and liver enzymes were normal, as was creatinine concentration (61.9 μmol/l). Cardiac troponin I was moderately elevated in 3 serial measurements (0.06, 1.85, and 1.61 μg/l). Conversely, potassium was 2.6 mmol/l (normal range, 3.5 to 5.0 mmol/l), calcium 5.8 mg/dl (normal range, 8.5 to 11 mg/dl), and magnesium 0.3 mg/dl (normal range, 1.9 to 2.5 mg/dl). Ion repletion was immediately started, whereas coronary angiography was deferred based on absence of symptoms and RWMA consistent with ST-segment elevation myocardial infarction (STEMI). On electrolyte reintegration, the ECG gradually improved (**Figure 1B**). Two days later, after normalization of serum electrolytes, coronary angiography was performed, excluding significant coronary artery disease (Supplemental Figures 1 and 2).

DISCUSSION

ECG is fundamental to diagnose STEMI and trigger immediate treatment. Notwithstanding, a STEMI-like ECG may depend on several other conditions, including electrolyte disorders, myocarditis/pericarditis, and pulmonary embolism (1). Interpretation of the ECG in the clinical context becomes then critical.

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ABBREVIATIONS AND ACRONYMS

ECG = electrocardiogram

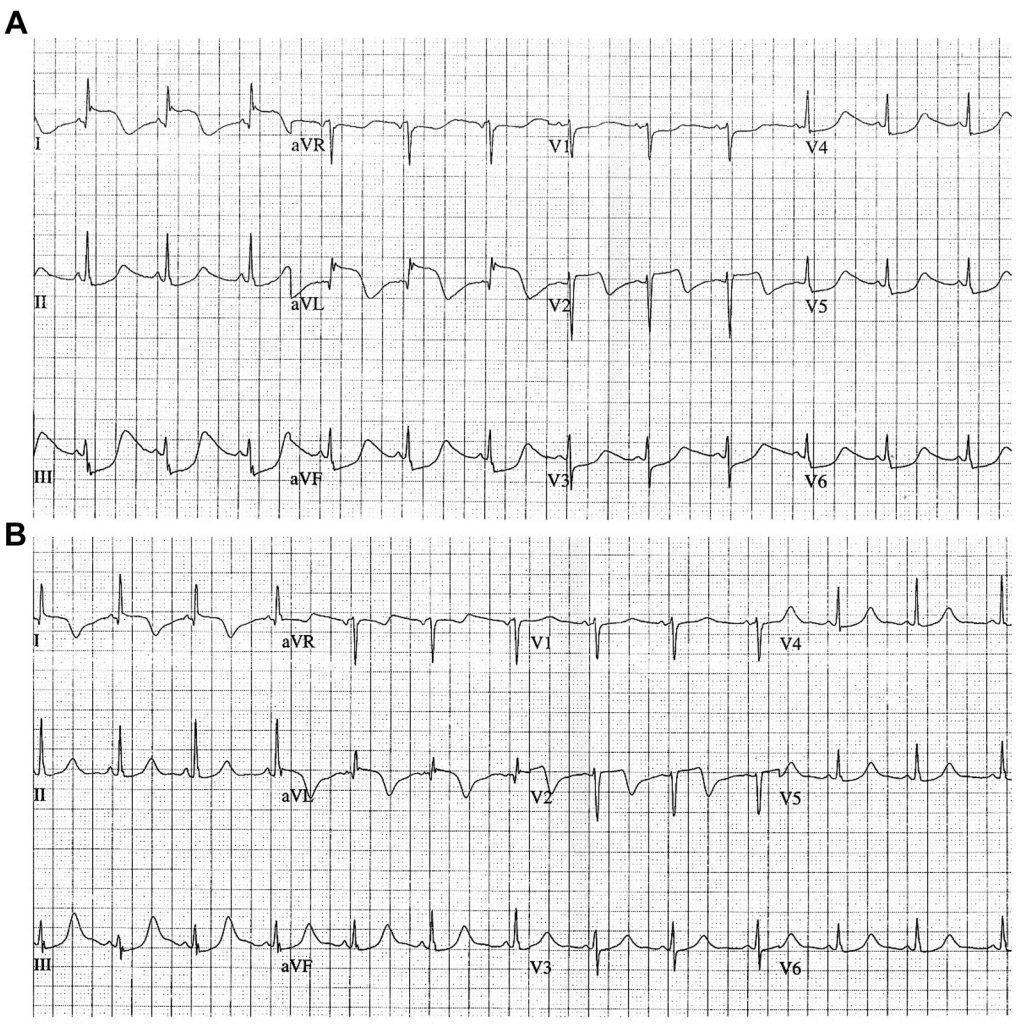
RWMA = regional wall motion abnormalities

STEMI = ST-segment elevation myocardial infarction

This caveat is exemplified by our case. Although the ECG alone raised the suspicion of STEMI, the patient did not have symptoms, signs, and echocardiographic alterations suggesting an acute coronary syndrome. Of note, the ECG displayed ST-segment elevation in leads I, aVL, and V_2 with reciprocal ST-segment depression in the inferior and precordial leads, which supported STEMI diagnosis, but also uncharacteristic nonconvex elevation of the ST-segment associated with asymmetric, diphasic T waves and ST-segment depression in all other leads. Furthermore, the QT interval was prolonged (600 ms), consistently with metabolic rather than ischemic myocardial injury.

Hypocalcemia is the electrolyte alteration primarily leading to ST-segment elevation, although the mechanisms remain unclear (Supplemental Figure 3) (2,3). Plainly, our interpretation of the ECG is speculative, and other causes may have occurred. A transient coronary spasm was suggested by the ECG abnormalities consistent with a coronary territory involvement, although long lasting. Furthermore, hypocalcemia and hypomagnesemia may elicit coronary spasm. By contrast, the coronary-like distribution of the ECG alterations did not support the presumptive diagnosis of myocarditis, as well as the absence of symptoms, increase in

FIGURE 1 Electrocardiographic Evolution During Electrolyte Repletion



(A) Electrocardiogram showing ST-segment elevation in leads I, aVL, and V_2, and ST-segment depression in the inferior and precordial leads. **(B)** Electrocardiogram showing regression of ST-segment elevation after correction of ion imbalances.

inflammatory markers, and RWMA. Thus, cardiac magnetic resonance was not performed. The lack of RWMA also pointed against takotsubo syndrome, which can be triggered by electrolyte imbalances.

In conclusion, the process leading to the diagnosis of STEMI relies on integration of multiple pieces of information, and mental shortcuts prompted by single cues must be avoided: the ECG alone is not a STEMI.

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REFERENCES

1. Wang K, Asinger RW, Marriott HJL. ST-segment elevation in conditions other than acute myocardial infarction. N Engl J Med 2003;349:2128–35.

2. Adeel MY, Clarke JD, Shetty S, Arora A, Buscher MG. Severe hypocalcemia mimicking acute inferior ST-segment elevation myocardial infarction. Oxf Med Case Reports 2018;2018:omy103.

3. Yusuf J, Khan MU, Cheema Y, Bhattacharya SK, Weber KT. Disturbances in calcium metabolism and cardiomyocyte necrosis: the role of calcitropic hormones. Prog Cardiovasc Dis 2012;55:77–86.

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APPENDIX For supplemental figures, please see the online version of this paper.