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Case Report

Unusual Manifestations of Severe Hypokalemia: Arrhythmias and Severe Ischemic ECG Changes-A Case Report

Mahdieh Mehralipoor[1], MD; Asadollah Amiripour[2*], MD; Amirmasoud Rahimi[1], MD; Mohammad Ruzbahani[1], MD; Mohammad Sobhan Amiripour[3]

ABSTRACT

Hypokalemia, characterized by low serum potassium levels, can cause marked alterations in cardiac electrical activity and lead to a range of ECG abnormalities. These changes span from mild findings to severe, life-threatening arrhythmias. A key diagnostic challenge is the resemblance of ECG manifestations of hypokalemia to ischemic heart disease, particularly myocardial infarction with nonobstructive coronary arteries. This overlap can complicate diagnosis, especially in patients presenting with chest pain. This case report describes a patient with severe hypokalemia whose ECG showed changes mimicking ischemic injury, including ST-segment depression, T-wave inversion, and atrial fibrillation. Despite initial concern for myocardial infarction, further evaluation—including normal troponin levels, echocardiographic findings, and selective coronary angiography—ruled out significant ischemia. The patient improved with potassium correction, and subsequent normalization of the ECG supported a diagnosis of hypokalemia-induced changes. This case underscores the importance of rapid potassium correction to prevent adverse cardiac outcomes and highlights the need for careful differential diagnosis when interpreting ECG findings in hypokalemia. ***(Iranian Heart Journal 2025; 26(4): 85-88)***

KEYWORDS: Hypokalemia; ECG; Myocardial infarction; Potassium correction; Electrocardiographic changes

[1] Imam Ali Cardiovascular Research Center, Kermanshah University of Medical Sciences, Kermanshah, IR Iran.
[2] Cardiology Department, Imam Ali Cardiovascular Research Center, Kermanshah University of Medical Sciences, Kermanshah, IR Iran.
[3] Student Research Committee, Kermanshah University of Medical Sciences, Kermanshah, IR Iran.

*** Corresponding Author:** Asadollah Amiripour, MD; Cardiology Department, Imam Ali Cardiovascular Research Center, Kermanshah University of Medical Sciences, Kermanshah, IR Iran.
Email: asadamiri1351@yahoo.com **Tel:** +988337246791

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Variations in serum potassium levels can substantially affect electrical conduction and cardiac cellular function, resulting in ECG abnormalities. These manifestations may appear as mild, incidental findings or progress to severe, potentially life-threatening rhythm disturbances. Although not all ECG abnormalities arise directly from potassium imbalances, the ECG remains an essential tool for assessing the severity of such disturbances and guiding urgent medical intervention. [1, 2] Among the reported changes, ST-segment depression, T-wave inversion, and U waves are most common and may closely resemble ischemic heart disease, creating diagnostic challenges. [3] Patients presenting with atrial fibrillation (AF) and ischemic-appearing ECG changes are frequently managed as acute coronary syndrome, particularly when accompanied by nonspecific symptoms such as chest

discomfort or palpitations. Nonetheless, in elderly patients, electrolyte disturbances such as hypokalemia may underlie these presentations. [4] Recognition of such abnormalities is critical to prevent unnecessary invasive procedures and to promptly treat the reversible cause. This report describes the diagnostic and therapeutic implications of severe hypokalemia in an 85-year-old patient presenting with AF and suspected myocardial infarction with nonobstructive coronary arteries (MINOCA).

CASE REPORT

An 85-year-old woman with a history of coronary angioplasty, diabetes, hypertension, and renal artery stent placement 10 years prior presented to the emergency department with chest pain and cold sweats. She reported 3 days of weakness and lethargy but denied fever, nausea, vomiting, abdominal pain, dyspnea, peripheral edema, or recent antibiotic use. On arrival, the patient was alert with stable vital signs: blood pressure, 140/90 mm Hg; heart rate, 110 beats per minute; temperature, 37.3 °C; and oxygen saturation, 92%. Physical examination revealed clear breath sounds, no cardiac murmurs, and a soft, nontender abdomen.

The initial ECG showed AF with a ventricular rate of approximately 110 beats per minute, ST-segment depressions in the anterior, inferior, and lateral leads, and ST-segment elevations in leads aVR and V_1 (Figure 1). Given concern for myocardial infarction, the patient was transferred urgently to the percutaneous coronary intervention center. Emergency coronary angiography revealed single-vessel disease with moderate stenosis in the mid-right coronary artery and mild ostial stenosis of the left anterior descending artery. Due to suspected aortic dissection, an aortic root injection was performed, which showed no evidence of dissection. In the cardiac care unit, echocardiography demonstrated normal left ventricular size and systolic function, no pericardial effusion or clot, and mild mitral regurgitation. Laboratory test results were as follows: white blood cell count, 7.2 × 10^9/L; hemoglobin, 11.6 g/dL; platelet count, 250 × 10^9/L; partial thromboplastin time, 29 seconds; international normalized ratio, 1; blood glucose, 246 mg/dL; urea, 64 mg/dL; creatinine, 1.4 mg/dL; sodium, 132 mmol/L; potassium, 2 mmol/L; magnesium, 2.2 mmol/L; and troponin, negative.

The patient received intravenous and oral potassium chloride for rapid correction. After 6 hours, the serum potassium level increased to 2.7 mmol/L, and a follow-up ECG showed sinus rhythm at 65 beats per minute without ischemic changes (Figure 2). [1] Treatment for hypokalemia was continued with regular monitoring of potassium levels. The patient's symptoms improved significantly, and after 3 days of hospitalization, she was discharged with a serum potassium level of 4.1 mmol/L. At a 1-week follow-up clinic visit, electrolyte levels were normal, with a potassium level of 3.5 mmol/L.

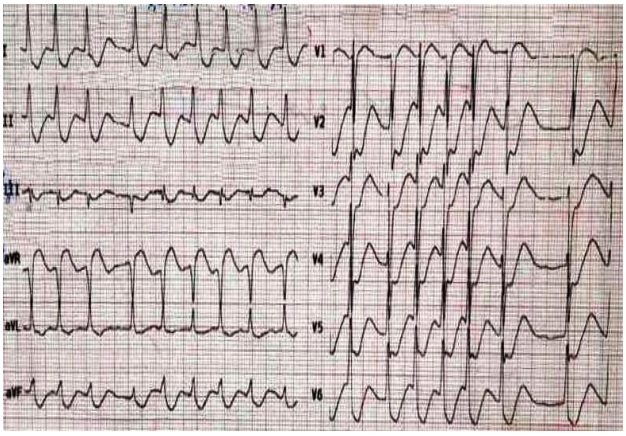

Figure 1. The image presents the patient's admission ECG.

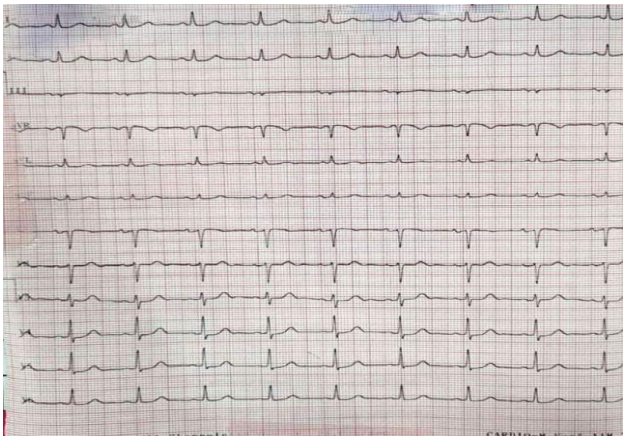

Figure 2. The image presents the patient's ECG after she received intravenous and oral potassium chloride for rapid correction.

DISCUSSION

Hypokalemia is a critical condition that, if left untreated, can lead to serious complications such as reduced cardiac output, hypotension, arrhythmias, and potentially cardiac or respiratory arrest. Recognition of ECG changes associated with hypokalemia is essential because rapid diagnosis and intervention are vital. [5] Pathophysiologically, reduced potassium levels cause cellular hyperpolarization, which increases the resting potential of cardiac cells and accelerates depolarization. This process enhances cardiac automaticity and excitability, producing notable ECG alterations. Hypokalemia also prolongs the action potential and increases the QT interval. [6] In addition, hypokalemia may induce ECG changes via vasoconstrictive effects, as decreased potassium levels are associated with narrowing of blood vessels. [7] Typical ECG features of hypokalemia include ST-segment depression, reduced T-wave amplitude, prominent U waves, and a U-to-T ratio greater than one. Nevertheless, distinguishing between hypokalemia and ischemia can be challenging in patients treated with digitalis, those with ventricular hypertrophy, conduction abnormalities, or central nervous system disorders. [8–10]

Nonischemic causes of ST-segment depression include right and left ventricular hypertrophy with a "strain" pattern and conduction abnormalities such as right bundle branch block and left bundle branch block. In addition, hypokalemia and hypomagnesemia may contribute to ST-segment depression. [11, 12]

Several studies have shown that a substantial proportion of patients with hypokalemia exhibit ECG changes. In a 2014 study, Marti et al. [13] reported ECG abnormalities in 69% of patients with hypokalemia, with U waves (24%) and ST-segment depression (21%) being most common. In 2019, Sokolovic et al. [14] examined the relationship between hypokalemia and MINOCA, noting ECG changes in a patient with chest pain and low potassium; potassium correction reversed the ECG changes despite the absence of coronary occlusion. Further, in 2020, Musa et al. [15] reported improvement of ECG abnormalities, including QT prolongation and U waves, after potassium repletion, even before potassium levels returned to normal. In 2024, Humberto et al. [16] reported a case in which severe hypokalemia mimicked signs of an obstructive lesion of the left main coronary artery, emphasizing the need for rapid potassium treatment when ECG changes resemble ischemia.

In our case, the patient's ST-segment depression, along with AF with rapid ventricular response, was reminiscent of subendocardial injury or myocardial ischemia. Still, given the normal troponin levels, normal echocardiogram, and resolution of ECG abnormalities after potassium correction, the changes were attributed to severe hypokalemia rather than myocardial ischemia.

CONCLUSIONS

Severe hypokalemia can induce significant ECG changes that may be misinterpreted as acute ischemic injury or arrhythmia. Rapid potassium correction is critical to reverse these changes, prevent serious cardiac complications, and avoid misdiagnosis. Prompt treatment can resolve hypokalemia-associated ECG abnormalities, alleviate symptoms, and mitigate the risk of severe cardiac events. Timely potassium repletion also serves a diagnostic role by helping to differentiate between changes secondary to hypokalemia and those caused by primary ischemic events, thereby improving patient management and outcomes.

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