ECG Mimicry: Hypokalemia Mimicking Myocardial Ischemia

Travis R. Huffman, DO[1], Kris Pfirman, DO[1], Aniruddha Singh, MD[2]
[1]University of Kentucky-Bowling Green, Cardiovascular Medicine Fellowship, Bowing Green, KY
[2] Med Center Health, Western Kentucky Heart and Lung Research Foundation and Education Trust, Bowling Green, KY

Objectives

- Metabolic abnormalities, specifically hypokalemia, alter normal cardiac conduction and produce specific patterns on Electrocardiogram (ECG).
- Severe hypokalemia produces significant ST segment depressions and abnormal T wave morphology, mimicking the pattern present in myocardial ischemia.
- Recognizing severe hypokalemia and its associated ECG findings can help avoid risk associated interventions such as systemic anticoagulation

Case

A 37-year-old male with no significant past medical history presented after a syncopal episode with complaint of generalized fatigue, without chest pain or dyspnea. The patient reported several weeks of loose stools with decreased oral intake preceding presentation.

Physical exam was significant for a cachectic appearing male. Cardiac exam noted a regular rate, regular rhythm, normal S1/S2 without murmurs or rubs, no peripheral edema and 2+ distal pulses bilaterally.

Vital signs were unremarkable. Chest X-ray was without evidence of cardiopulmonary disease. Admission 12-Lead ECG was significant for ≥2mm ST segment depressions in inferior limb leads and anterolateral precordial leads with reciprocal ST segment elevation in aVR, concerning for subendocardial myocardial ischemia (Figure 1).

Admission laboratory studies were remarkable for undetected cardiac enzymes and a serum potassium of 2.0 mmol/L and positive HIV ab/ag testing. ECG obtained after repletion of potassium displayed resolution of ST segment depressions and return of normal T wave morphology with disappearance of previously present U wave (Figure 2). Potassium was 3.4 mmol/L at time of repeat ECG.

Electrocardiogram Comparison

Figure 1. Admission ECG. Serum Potassium 2.0 mmol/L

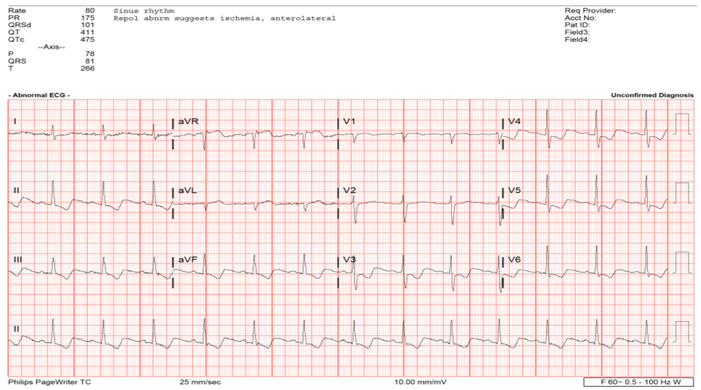
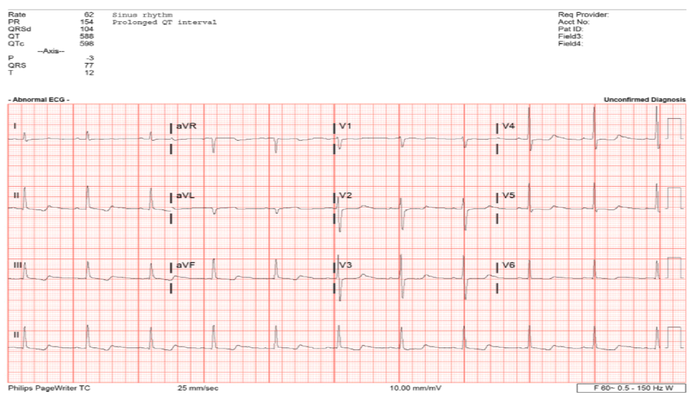


Figure 1. ECG with >2mm ST segment depressions in leads II, III, aVF, V3-V6, biphasic T waves with presence of U waves in the same leads, as well as subtle 1mm ST segment elevation in aVR, mimicking a pattern consistent with myocardial ischemia.

Figure 2. Serum Potassium 3.8 mmol/L



ECG after repletion of potassium; serum K+of 3.4 mmol/L with near complete resolution of ST segment depressions, normal T wave morphology and absence of U wave.

Discussion

Metabolic disorders, specifically hypokalemia, alter cardiac conduction and result in specific ECG patterns. At the cellular level, hypokalemia produces an alteration in resting membrane potential and a prolongation of the cardiac myocyte action potential [1,2]. These alterations at the cellular level are represented on ECG as decreased T wave amplitude, ST segment depressions, prolongation of the P-R interval, QT prolongation and development of a U wave [1,2].

The U wave, a characteristic finding in severe hypokalemia, is a positive deflection following the T wave, best seen in precordial leads. QT interval prolongation is suspected to occur in severe hypokalemia, as it prolongs repolarizations. However, accurate measurement of the QT interval in severe hypokalemia can be challenging as the T wave and U wave often merge[1,2].

While ST segment depressions in the emergent setting require prompt recognition and intervention, these changes are not isolated to cases of myocardial ischemia. Recognizing severe hypokalemia and its associated ECG findings can help avoid an incorrect diagnosis of myocardial ischemia and avoid risk-associated interventions such as systemic anticoagulation and cardiac catheterization.

Potassium repletion is crucial in management as it not only improves surface ECG findings, but more importantly prevents the development of both atrial and ventricular arrhythmias.[3]

References

1. Mirvis DM, Goldberger AL, Electrocardiography. In Zipes, DP, editor. Braunwald's Heart Disease: A Textbook of Cardiovascular Medicine, Eleventh Edition. Elsevier, 2019:117-153
2. Lee, JZ, Asirvatham, SJ. Metabolic disturbances for the electrophysiologist. J Cardiovasc Electrophysiol. 2019; 30: 2147– 2149. https://doi.org/10.1111/jce.14139
3. Rose BD, Post TW. Hypokalemia. In: Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed, Rose BD, Post TW (Eds), McGraw-Hill, New York 2001. p.836.