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Severe Hypokalemia Masking Myocardial Ischemia

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Introduction

Variations in serum potassium levels can impact cardiac cell conduction significantly and can result in alterations to electrocardiograms.[1] These alterations may cause incidental abnormalities on the 12-lead ECG or trigger dysrhythmias that may be fatal. The electrocardiogram is a helpful screening tool for determining the severity of the blood potassium abnormality and the urgency of the therapeutic response, even though in some cases ECG abnormalities do not accompany serum potassium abnormalities.ST segment depression brought on by hypokalemia might mimic subendocardial damage or ischemia, and hypokalemia's ECG correlates can be mistaken for myocardial ischemia.[2] Differential diagnosis with myocardial ischemia can be challenging in many clinical practice scenarios, particularly when chest pain is present along with ST-T alterations.[3]

Case report

A 30-year-old previously healthy male arrived at the Emergency Department with a two-day history of progressive weakness and chest discomfort. He attempted to walk on the day of presentation but fell due to his weakness. The electrocardiogram (ECG) indicated sinus rhythm at 60 per minute with significant ST-segment depression in multiple leads, leading to an initial diagnosis of non-ST elevation myocardial infarction. Following admission to the Cardiology Department, transthoracic echocardiography revealed normal findings with no evidence of regional wall motion abnormalities. Further inquiry unveiled a recent intake of unidentified food during the holiday, accompanied by upper abdominal pain, diarrhea, and vomiting. Physical examination revealed dehydration, hypotension, and hypokalemia with normal troponin levels. A slow gait was noted on neurological examination, while abdominal

examination was unremarkable. Given the clinical picture, acute gastroenteritis was suspected. Immediate potassium supplementation and continuous electrocardiographic monitoring were initiated, alongside intravenous potassium chloride infusion. Serum potassium levels were closely monitored, and as electrolyte correction ensued, ECG abnormalities resolved. Upon discharge, electrolyte levels and ECG findings were within normal limits.

Discussion

Hypokalemia poses a critical medical emergency, as its delayed resolution can lead to diminished cardiac output and peripheral perfusion, culminating in dysrhythmias and hypotension that may progress to cardiac and respiratory arrest.[4] Timely recognition and intervention are imperative due to the associated high morbidity and mortality rates.[5]While laboratory tests are considered the gold standard for diagnosing electrolyte imbalances, delays in obtaining results can occur. Thus, clinicians must be adept at identifying electrocardiographic signs of hypokalemia, which include ST-segment depression, decreased T-wave amplitude, prominent U waves, and a U-wave to T-wave ratio exceeding 1.[6]Distinguishing hypokalemic ECG patterns from those induced by digitalis or ischemia requires precision.[7]The hypokalemic ECG index has been proposed as a predictive tool, evaluating the sum of ST depression and U wave in specific leads.[8]Interpretation of ST-segment depression necessitates careful consideration to differentiate non-ischemic causes, such as ventricular hypertrophy or digoxin therapy, from ischemic etiologies.[9] Conduction abnormalities, mitral valve prolapse, central nervous system disorders, as well as hypokalemia and hypomagnesemia, can all manifest as ST depression on electrocardiography.[10]

Conclusion

ST-segment depression caused by hypokalemia can closely resemble myocardial ischemia, posing a challenge in differential diagnosis, particularly when accompanied by chest discomfort. The electrocardiographic findings in this case underscore the potential for severe hypokalemia to mimic or even precipitate myocardial ischemia.

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