Addison’s disease (AD) is a metabolic disorder that affects the metabolism of potassium. The hyperkalemia that results from this condition can be reflected in the electrocardiogram, which could confound the diagnosis of other cardiac conditions. Such a challenging situation was encountered when a 73-year-old male with history of AD showed up to the emergency department with complaint of chest pain.

INTRODUCTION

Addison’s disease (AD) is a metabolic disorder that affects the metabolism of potassium. The hyperkalemia that results from this condition can be reflected in the electrocardiogram (ECG), which could confound the diagnosis of other cardiac conditions. Such a challenging situation was encountered when a 73-year-old male with history of AD showed up to the emergency department with complaint of chest pain.

CASE REPORT

A 73-year-old male with history of AD treated with prednisone presented to the emergency department complaining of chest pain while walking. He described the pain as retrosternal, constant, nonradiating and associated with one episode of vomiting. He denied shortness of breath, nausea, palpitations or sweating. The patient had similar episodes of chest pain in the past associated with high serum potassium from AD and improved after correcting the hyperkalemia. Past medical history was significant for chronic renal failure and gastric esophageal reflux disease. He had no known drug allergies and did not smoke.

The patient was in no acute distress. The blood pressure was 127/80 mmHg, heart rate was 74 beats per minute and respiratory rate was 22 respirations per minute. On the exam, there was a normal S1 and S2 and no S3, S4, murmurs or friction rubs were present. There was no jugular venous distention and the peripheral pulses were normal. The remainder of the physical exam was unremarkable. An ECG (Fig. 1a) showed sinus rhythm at 75 beats per minute with low voltage in the limb leads. Peaked T waves were noted in the precordial and limb leads (V2–6, I, II, III, AVF). It also showed subtle ST depression in lateral leads and no Q waves were appreciated. No previous ECGs were available for comparison. A first set of cardiac enzymes showed Troponin I of 0.16 ng/ml, CK-MB 2.7 ng/ml and myoglobin 170 ng/ml. The creatinine was 2.0 mg/dl, potassium 6.3 meq/l, sodium 137 meq/l, leukocyte count 7.3K/µl, hemoglobin 12.8 g/dl and b-type natriuretic peptide 34 pg/ml. The patient was admitted to the telemetry floor for ECG changes related to hyperkalemia from AD and he was treated with calcium gluconate, insulin, furosemide and sodium polystyrene sulfonate. Six hours later a second set of cardiac enzymes showed myoglobin of 1843 ng/ml, Troponin I 11.4 and CK-MB 85.7 ng/ml. A repeat ECG showed new Q waves in leads V2, V3 and V6, with rs complex in v5 (Fig. 1b). The patient remained hemodynamically stable and the chest pain resolved. The potassium decreased to 5.6 meq/l.

The patient was started on aspirin, metoprolol, atorvastatin and intravenous heparin and transferred to the cardiac intensive care unit. Subsequently, he underwent a coronary angiogram, which showed a thrombus in the mid left anterior descending (LAD) artery with 99% stenosis (Fig. 2). A percutaneous transluminal coronary angioplasty of the LAD artery was performed with placement of a drug-eluting stent. The procedure was uneventful and the patient was discharged home 5 days later.
DISCUSSION

The presence of prominent T waves in the ECG can be seen in early repolarization and left ventricular hypertrophy. Furthermore, they also appear in hyperkalemia and the earliest stage of myocardial infarction (MI) [1]. Usually, T waves from hyperkalemia are tall, narrow and symmetrically peaked, whereas those from ST-elevation MI are broad and asymmetrically peaked. Other changes seen with hyperkalemia are ST-segment elevation and widened QRS complex [2, 3], which could further complicate the assessment of a patient with chest pain.

An acute MI is a serious condition that has to be addressed immediately. The ECG is an important tool to identify promptly those who might benefit from early aggressive interventions [4]. In patients with high clinical suspicion for ischemic heart disease, it is of paramount importance to follow up
serial ECGs in order to reveal the presence of dynamic changes of the ST segment and/or T wave [5]. In addition, the use of serial cardiac biomarkers can increase the likelihood of diagnosing an existing ischemic event [6]. Moreover, a bedside echocardiogram, which is a relatively inexpensive and widely available tool, can be used to detect wall motion abnormalities in patients with equivocal changes in the ECG and negative cardiac enzymes. This diagnostic modality can also be used for prognostic purposes and to evaluate those who might benefit from aggressive interventions [7].

CONCLUSION

The presence of broad, asymmetrically peaked T waves can be seen in early stages of ST-segment elevation MI. However, this can be compounded by other conditions, including hyperkalemia, which usually produces tall, narrow peaked T waves on the ECG. In a patient with both conditions, in which the ECG findings might be equivocal, following serial ECGs, along with serial cardiac enzymes and performing a bedside echocardiogram may yield invaluable diagnostic information.

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REFERENCES