QUICK QUIZ
WHAT IS THE DIAGNOSIS?

BACKGROUND

A 50-year-old woman presents to the emergency department because of recurrent syncopal episodes over the last 2 days. She has had worsening weakness and weight loss in the last 3 months. She denies having fevers, chills, dyspnoea, nausea, vomiting, abdominal pain, or urinary symptoms. Her medical history is significant for a chronic pain disorder involving multiple joints and her back, with associated headaches. She is being treated with long-term Risedronate, Amitriptyline, Carisoprodol, Buspirone, and Omeprazole.

On physical examination, her blood pressure is 125/80 mm Hg, her temperature is 37.5°C, and her heart rate is about 90 bpm and regular, with no abnormal heart sounds. She weighed 94 lb (35 kg) 6 months ago but has lost 15 lb. She has normal sensation over her body with symmetric deep tendon reflexes and normal cranial-nerve responses, though she has diffuse weakness in all major muscle groups. Her mental status is normal.

ECG is shown below.

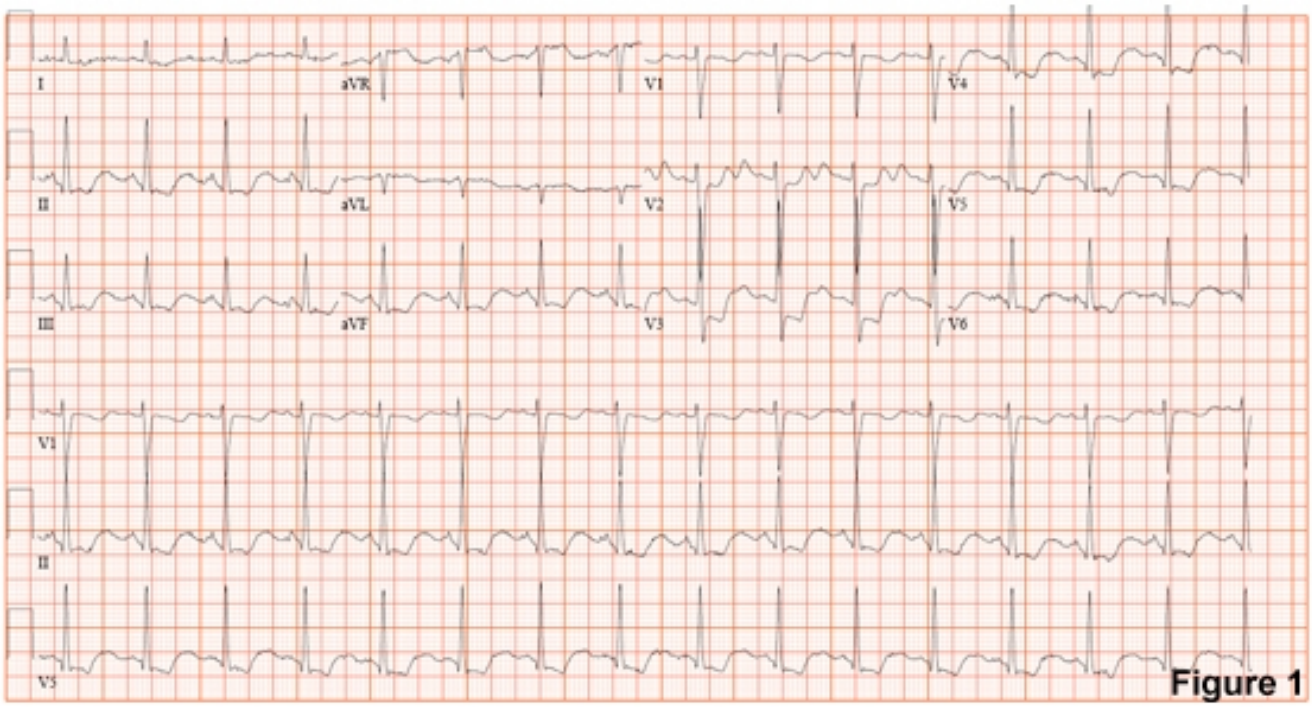

Figure 1

QUESTION

1. What's the diagnosis?

ANSWER

Hypokalaemia: The ECG shows a normal sinus rhythm at a rate of 92 bpm with depression of the ST segment in the inferior and anterior leads and T-wave inversions in the lateral leads. The PR interval is normal at 126 ms, whereas the QT and QTc intervals are prolonged at 506 and 621 ms, respectively. U waves are present in all leads but are most prominent in V_2.

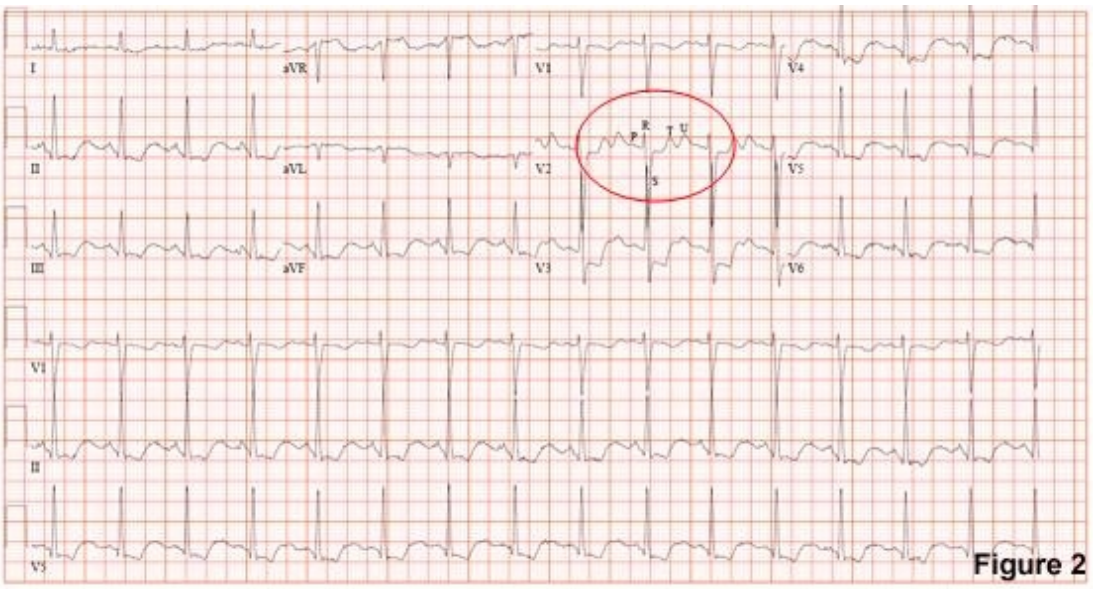

Figure 2

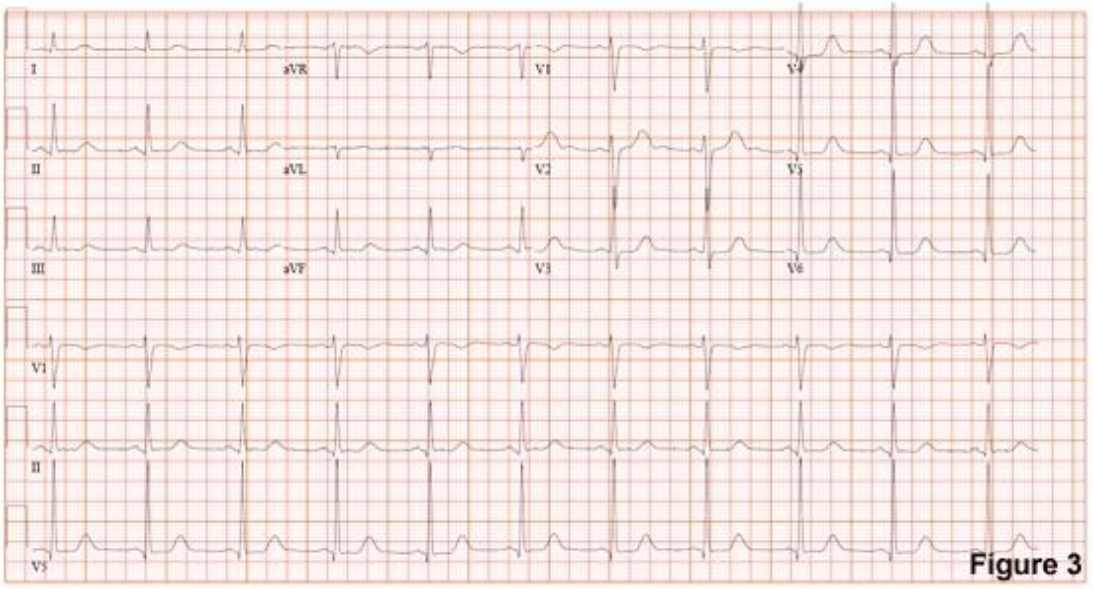

Figure 3

The results of a metabolic panel revealed a K^+ level of 1.6 mmol/L. On further questioning, the patient admitted to a long history of self-induced vomiting, with a recent increase in frequency. The patient was admitted for cardiac monitoring, K^+ supplementation, and psychiatric evaluation. The initial ECG changes (seen in Figure 2) completely resolved when the patient's K^+ level was corrected (see Figure 3). The patient was eventually discharged home for outpatient psychiatric treatment of her eating disorder.

Hypokalemia is defined as a plasma K^+ concentration of less than 3.5 mmol/L and may result from several physiologic processes: decreased net intake of K^+, K^+ shift into cells, or increased net loss of K^+. In this patient, losses from vomiting of gastric secretions could not completely account for her severe hypokalemia. The K^+ concentration of gastric fluid is too low (5-10 mmol/L) to cause the deficit of greater than 400 mmol typical of moderate-to-severe hypokalemia. In cases like this, the primary mechanism is increased renal K^+ excretion due to both metabolic alkalosis and volume depletion resulting from the loss

of gastric fluid.

Clinical manifestations of mild-to-moderate hypokalemia are fatigue, myalgia, and muscular weakness of the lower extremities. Severe hypokalemia may lead to progressive weakness; hypoventilation; and, eventually, complete paralysis, rhabdomyolysis, and paralytic ileus. Characteristic ECG changes of hypokalemia are due to delayed ventricular repolarization and are not well correlated with plasma K^+ concentrations. Early changes are flattening or inversion of the T wave, a prominent U wave, ST-segment depression, and a prolonged QU interval. Severe K^+ depletion may result in a prolonged PR interval, decreased voltage, widening of the QRS complex, and an increased risk of ventricular arrhythmias.

The treatment of hypokalemia is K^+ repletion and correction of the cause to minimize ongoing loss. Patients with severe hypokalemia or those unable to take anything by mouth require intravenous replacement therapy with KCl. The concentration of intravenous K^+ should not exceed 40 mmol/L for administration via a peripheral vein or 60 mmol/L for a central vein. The rate of infusion should not exceed 20 mmol/h unless muscular paralysis or malignant ventricular arrhythmias are present. Ideally, KCl should be mixed in normal saline because dextrose solutions may initially exacerbate hypokalemia as a result of insulin-mediated intracellular movement of K^+.