

THE PRACTITIONER LE PRATICIEN

Country cardiograms case 55: Answer

Charles Helm, MD, CCFP Tumbler Ridge, BC

light sinus arrhythmia is present, with a mean rate of 65 beats/min. The PR interval is normal (0.16 s). The QRS duration is at the top end of normal (0.105 s), the QT interval is normal (0.40 s) and the QRS axis is normal (80°). The P wave morphology is normal; there is no evidence of left atrial abnormality. High QRS voltage is present (S wave of 30 mm in lead V2, R waves of 34 mm in lead V5 and 22 mm in lead II). Down-sloping ST segment depression is present in inferior leads II, III and aVF, and precordial leads V2-V6. The T waves are flattened, and very prominent U waves are present in leads V1-V5.

The abnormal features on this electrocardiogram (ECG) include high QRS voltage, down-sloping ST segment depression, flat T waves and prominent U waves.

Although left ventricular hypertrophy (LVH) is a common cause of high QRS voltage, caution is warranted when diagnosing this based on voltage criteria alone, especially in younger patients with thin chest walls. The presence of left atrial abnormality, left axis deviation, secondary ST-T changes or a wide QRS complex would make a diagnosis of LVH more likely and form the basis for the Romhilt-Estes scoring criteria. In this case, the voltage, in addition to the slightly wide QRS complexes and the ST segment changes, prompt consideration of LVH, supported by the hypertension noted on examination. Caution should be exercised in diagnosing this with certainty, however, considering the patient's young age.

The causes of ST segment depression include secondary changes to LVH or bundle branch block; ischemia; medications, such as digoxin; and hypokale-

mia. In this case, LVH or hypokalemia would be the most likely causes.

Small U waves are often a normal phenomenon, but the large U wave amplitude in this case is unusual and suggests hypokalemia. The flat T waves, rather than the inverted T waves that are often seen secondary to LVH, further raise the suspicion for hypokalemia.

Electrocardiogram diagnosis in this case suggests hypokalemia and possible LVH.

This patient's serum potassium level was 1.5 (normal 3.5–5.1) mmol/L, with associated alkalosis and hyponatremia, and an elevated creatine kinase level. Hypertension in a young person raises the possibility of secondary causes, and hypertension combined with profound hypokalemia (in the absence of diuretic therapy) suggests hyperaldosteronism. Further screening investigations in this case suggested primary hyperaldosteronism, with a grossly elevated aldosteroneto-renin ratio of greater than 7400 (normal < 1500) pmol/L per ng/L/s.

In an urgent situation such as this, the 12-lead ECG potentially provides a rapid diagnosis, enabling appropriate intravenous potassium therapy to be prepared while awaiting serum potassium results.

The "teapot" memory aid may be useful: "no pot, no T" (low potassium levels often produce T wave flattening). If U waves are present, they may be mistaken for T waves, leading to pseudolengthening of the QT interval. If the QT interval seems long, consider hypocalcemia, but also consider the U waves of hypokalemia as a cause.

For the question, see page 145.

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