Electrocardiogram after 2 weeks of diarrhea

D. Luke Glancy, MD, Frank E. Wilklow, MD, and Brent J. Rochon, MD

Figure. Electrocardiogram recorded on admission. See text for explanation.

A 41-year-old man, known to be HIV positive for at least 3 years (CD4 count on this admission, 134/mm³; reference, 228–2290), presented with a 2-week history of sore throat, dysphagia, odynophagia, change in his voice, fatigue, and exertional dyspnea. Only later did he admit to having 4 to 6 large watery stools per day for the same length of time. An electrocardiogram (ECG) recorded on admission showed sinus rhythm, sagging ST segments, and giant U waves, with the T waves appearing as slight protuberances on the upslopes of the U waves near their apices (Figure). The repolarization pattern is typical of severe hypokalemia, and a serum potassium level obtained at the time of the ECG was 1.2 mmol/L (reference, 3.6–5.2).

Diarrhea, the major cause of the hypokalemia, was due primarily to *Clostridium difficile* colitis, and the patient's stool

also was positive for *Cryptosporidium* antigen. Dysphagia and odynophagia, due to thrush and mediastinal and cervical emphysema, led to decreased oral intake that also played a role in causing both hypokalemia and the dehydration that was manifested by admission hypotension (blood pressure, 91/57 mm Hg), hemoconcentration (hemoglobin, 17.9 g/dL; hematocrit, 49%), and kidney failure (serum creatinine, 2.8 mg/dL; blood urea nitrogen, 66 mg/dL). With potassium and fluid replacement and appropriate antimicrobial therapy, the

From the Sections of Cardiology, Departments of Medicine, Louisiana State University Health Sciences Center and the Medical Center of Louisiana, New Orleans.

Corresponding author: D. Luke Glancy, MD, 7300 Lakeshore Drive, #30, New Orleans, Louisiana 70124 (e-mail: dglanc@lsuhsc.edu).

patient improved in all respects and was discharged 8 days after admission.

Although bradycardia, left ventricular hypertrophy, and digitalis can all increase the size of U waves, these never reach the size often seen with severe hypokalemia, as in this patient. Progressive hypokalemia also causes increasing ST-segment depression and a diminution in the size of the T wave (1–4). Because of the frequency of ST depression with ischemic heart disease, the hypokalemic ECG is often misdiagnosed as being the result of ischemia. The ECG that most closely mimics severe hypokalemia, however, is that seen in patients receiving digitalis and quinidine or another Vaughan Williams class IA antiarrhythmic drug (5).

In addition to defacing electrocardiographic repolarization, severe hypokalemia, by increasing automaticity and slowing conduction, can cause a variety of tachyarrhythmias, including fatal ventricular tachycardia/fibrillation and rarely atrioventricular block (1, 2). These arrhythmias occur more frequently in the presence of digitalis because fewer potassium ions allow more digitalis molecules to bind to potassium sites on membrane

sodium-potassium ATPase (3). Severe hypokalemia also can cause a variety of noncardiac problems: fatigue, anorexia, rapid shallow respirations, nocturia, mental aberrations, and muscular weakness that occasionally proceeds to paralysis (1). Although the electrocardiographic manifestations of hypokalemia have been known for over half a century (4), virtually none of the computer programs for interpreting ECGs attempt to read electrolyte disturbances, including hypokalemia. Thus, physicians are on their own in making this important diagnosis.

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