Hyperkalemia produces various abnormalities in electrocardiogram (ECG): symmetric tall T waves, wide QRS, shortened QT interval, short amplitude of P wave and ST-segment elevation that can simulate a pattern of myocardial infarction.

A 48-year-old man diagnosed of hepatitis C virus cirrhosis, portal hypertension and diabetes secondary to chronic pancreatitis, presented to the emergency room with intense dyspnea, signs of heart failure, hypotension (85/60 mmHg) and chest pain. Admission ECG showed sinus rhythm, wide QRS, Q-wave and ST-segment elevation in inferior leads III and aVF with reciprocal ST depression in leads I and aVL (Fig. 1), as well as ST-segment elevation (3 mm) and negative T-wave in right leads V3r - V4r (Fig. 2), which led to suspicion of inferior infarction and right ventricular involvement.

Analysis showed blood glucose 210 mg/dL, pH 7.33, HCO$_3$ 18 mEq/L, PCO$_2$ 34 mmHg and NT-proBNP 1500 pg/mL. Urgent echocardiography and coronary angiography were normal.

Serum potassium concentration on admission was 10.8 mEq/L, due to acute renal failure associated with type I hepatorenal syndrome. After normalization of serum potassium (4.5 mEq/L), the ECG (Fig. 3) showed sinus rhythm with narrow QRS, and the pseudoinfarction pattern disappeared.

Pseudoinfarction pattern associated with hyperkalemia is a rare ECG manifestation. The few relevant publications describe abnormalities that mimic an acute anteroseptal myocardial infarction. Hyperkalemia can simulate impaired ST-T, probably as a direct effect of increased extracellular potassium, which lowers the resting potential of cardiac fibers, thereby reducing the amplitude of action potential. However, it is not clear how hyperkalemia can produce localized instead of diffuse ischemic ECG patterns.