Two Cases of Severe Hyperkalemia with Atypical Electrocardiographic Manifestations

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Introduction

Hyperkalemia is one of the most common electrolyte abnormalities affecting the electrocardiographic changes. It is also the more common acute life-threatening metabolic emergencies seen in the emergency department. Thus, electrocardiography (ECG) often helps physicians to confirm the presence of severe hyperkalemia and to guide therapeutic maneuvers. Serum potassium levels higher than 8 mmol/L (severe hyperkalemia) are almost invariably associated with ECG abnormalities. However, minimal or atypical ECG changes have been observed in some cases of severe hyperkalemia[1, 2].

In this report, we describe two cases of severe hyperkalemia (K^+ ≥8 mmol/L) in which the ECGs showed atypical changes.

Case

Case 1

A 66-year-old woman presented to the Emergency Department complaining of generalized, progressive weakness, fever, and myalgia over the past 7 days. A physical examination revealed an acutely ill appearance and there was an eschar on her neck. She had moderate tenderness in the upper abdomen. Vital signs on admission revealed: blood pressure 100/70 mmHg, temperature 36.9℃, pulse 72 bpm, respiratory rate 22/min. Laboratory findings were as follows: blood urea nitrogen 66 mg/dl, creatinine 3.95 mg/dl, serum potassium 8.5 mmol/L, serum sodium 121 mmol/L, serum bicarbonate 21.3 mmol/L, ionized calcium 0.85 mmol/L. Sera were tested for a serodiagnosis of scrub typhus, which showed 1:320 in titers. An ECG was obtained, revealing ventricular rate 51 bpm, PR interval 184 ms, QRS duration 92 ms, QT/QTc 452/414 ms, sinus bradycardia, and nonspecific ST and T wave changes (Fig. 1). The patient was diagnosed with scrub typhus induced acute renal failure and was stabilized with calcium chloride, insulin/glucose therapy, followed by Kayexalate.

Case 2

A 41-year-old man presented to the Emergency Department complaining of abdominal pain and diarrhea over the past 5 days. His medical history included membranoproliferative glomerulonephritis, tuberous pleurisy, and cerebral infarction. A physical examination revealed an acutely ill appearance and he had entire abdominal tenderness. The intensity and frequency of bowel sound was decreased. Vital signs on admission revealed: blood pressure 80/60 mmHg, temperature 37.5℃, pulse 98 bpm, respiratory rate 22/min. Laboratory findings were as follows: blood urea nitrogen 41 mg/dL, creatinine 7.12 mg/dL, serum potassium 8.2 mmol/L, serum sodium 137 mmol/L, serum bicarbonate 23.6 mmol/L, ionized calcium 0.84 mmol/L. ECG showed ventricular rate 101 bpm, PR interval 148 ms, QRS duration 84 ms, QT/QTc 344/441 ms, and sinus tachycardia (Fig. 2). He was

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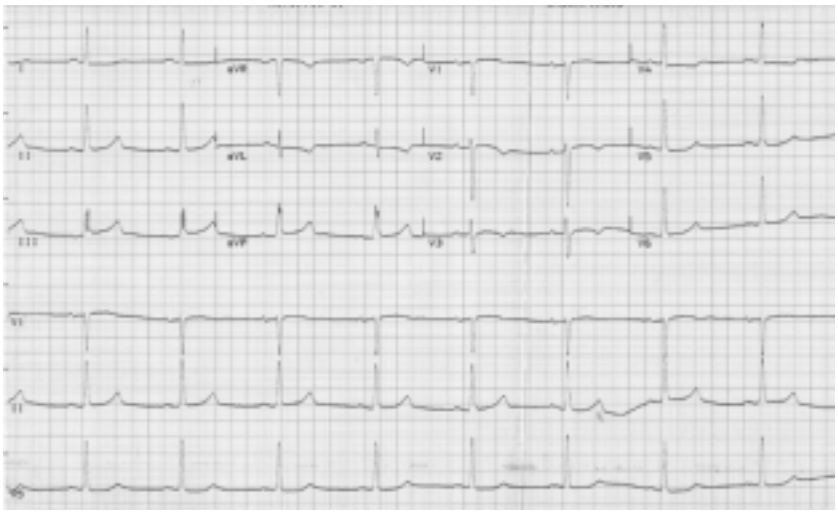

Fig. 1.

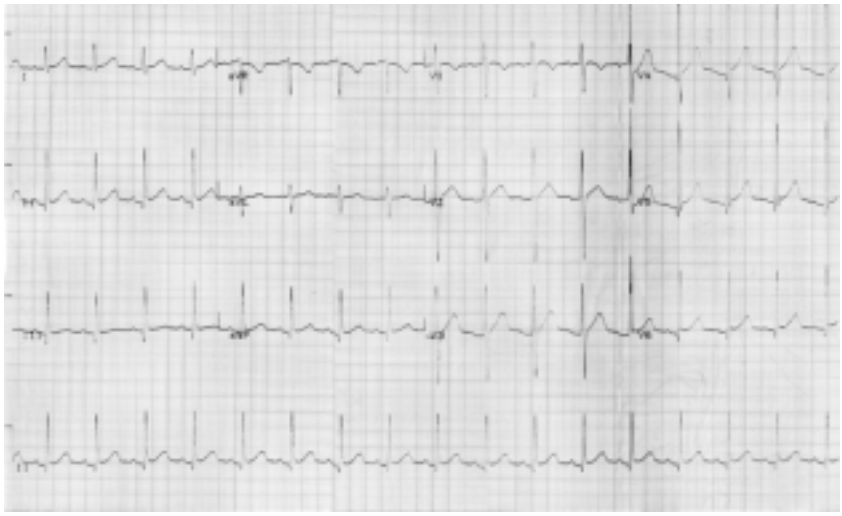

Fig. 1.

treated with calcium chloride, insulin/glucose therapy, Kayexalate, and hemodialysis. Serum potassium returned to normal, but he had a maintenance hemodialysis.

Discussion

Life-threatening hyperkalemia is most frequently diagnosed in patients with known chronic renal failure. Significant hyperkalemia may also be found in patients with many other medical conditions, including severe dehydration with resulting acute renal failure and situation associated with medications that affect kidney function[3]. Because of laboratory delays in obtaining serum potassium levels in patients, early diagnosis and treatment of hyperkalemia is dependent in many cases on ECG findings of hyperkalemia.

With the increased extracellular concentration of potassium, transmembrane permeability is increased, causing an influx of potassium into the cells. There is alteration of the transmembrane potential gradient, a decrease in magnitude of the resting potential, and a decrease in velocity of phase 0 of the action potential. The potassium influx causes a shortening of the action potential and results in delayed conduction between the myocytes. Finally, these changes produce a slowing of conduction[4].

Peaked T-waves in the precordial leads are among

the most common and the most frequently recognized findings on the ECG. Othor classic ECG findings in patients with hyperkalemia include prolongation of the PR interval, flattening or absence of the P-wave, widening of the QRS complex, and a sine-wave appearance at severely elevated levels[5].

However, these cases show that severe hyperkalemia can be seen without the typical ECG findings. There is no obvious explanation for the lack of ECG changes correlated with the severe hyperkalemia in our two cases. Spurious hyperkalemia could be also ruled out completely.

Other reports suggest the lack of ECG changes correlated with the severe hyperkalemia can be associated with the rate of change of serum potassium concentration, hypernatremia, hypercalcemia, and baseline ECG changes such as left ventricular hypertrophy, intraventricular conduction delay, and myocardial ischemia[1, 2].

In conclusion, our cases have the lack of correlation between severe hyperkalemia and ECG findings. Our physicians should be aware that the ECG is not always a reliable indicator of severe hyperkalemia.

References

1) Szerlip HM, Weiss J, Singer I : Profound hyperkalemia without electrocardiographic manifestations. *Am J Kidney Dis* **7:**461-465, 1986
2) Martinez-Vea A, Bardaji A, Garcia C, Oliver JA. Severe hyperkalemia with minimal electrocardiographic manifestations. *J Electrocardiol* **32:**45-49, 1999
3) Aslam S, Friedman EA, Ifudu O : Electrocardiography is unreliable in detecting potentially lethal hyperkalemia in hemodialysis patients. *Nephrol Dial Transplant* **17:**1639-1642, 2002
4) Mattu A, Brady W, Robinson DA. Electrocardiographic manifestations of hyperkalemia. *Am J Emerg Med* **18:**721-729, 2000
5) Diercks DB, Shumaik GM, Harrigan RA, Brady WJ, Chan TC : Electrocardiographic manifestations : electrolyte abnormalities. *J Emerg Med* **27:**153-160, 2004