Case Report

Profound Hyperkalemia and the Electrocardiogram. Lack of Correlation : A Case Report

Tarek Abdel Hamed Mostafa Dowod, Jaffer Ismail Ali, Sajid Burud
Department of Medicine, Al-Adan Hospital, Kuwait

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ABSTRACT

We report a case of severe hyperkalemia as a result of treatment with potassium sparing diuretics, digoxin and angiotensin receptor antagonist valsartan in the presence of renal insufficiency. Inspite of a maximal serum potassium concentration of 10.3 mmol/l, only non-specific ECG changes were found. The patient survived after an uneventful dialysis. Thus severe hyperkalemia may present without typical ECG changes, and values exceeding 10.3 mmol/l may not necessarily be fatal.

KEYWORDS: electrocardiogram (ECG), hyperkalemia

INTRODUCTION

The effects of hyperkalemia on cardiac electrophysiology have been extensively investigated in humans and animals both *in-vivo* and *in-vitro* and are well described in several reviews[1-4] and medical textbooks[5-6].

The most serious consequence of hyperkalemia is slowing of electrical conduction in the heart. The ECG begins to change when serum potassium (SK) concentration reaches 6.0 mmol/l and it is nearly always abnormal when serum potassium reaches 8.0 mmol/l[7].

The earliest change in the ECG is a tall tapering 'tented' T wave that is most evident in precordial leads (V2-V3). Similar peaked T waves have been observed in metabolic acidosis. As the hyperkalemia progresses, the Pwave amplitude decreases and the PR interval lengthens. The P wave eventually disappears and the QRS duration prolongs. The final event is ventricular asystole[8].

Although it is generally recognized that the ECG is not a reliable indicator of moderate hyperkalemia (SK < 7.0 mmol/l); more severe elevations of serum potassium (SK > 8.0 mmol/l) almost invariably exhibits ECG abnormalities[9]. The ECG is often used by physicians to confirm the presence of severe hyperkalemia and to guide therapeutic maneuvers in such instances.

This report describes a case of severe hyperkalemia (SK > 10.3 mmol/l) in which ECG revealed none of the abnormalities classically associated with hyperkalemia.

CASE REPORT

A 56-years-old woman with a history of ischemic cardiomyopathy, coronary artery bypass graft, diabetes mellitus, hypertension and chronic renal insufficiency was admitted with one week history of general ill-health, anorexia, nausea, body aches and reduced exercise tolerance.

The patient was taking insulin mixtard, moduretic (hydrochlorothiazide 50 mg + amiloride 5 mg) once daily, spironolactone 25 mg twice daily, valsartan 80 mg once daily, digoxin 0.125 mg once daily, atenolol 100 mg once daily and isosorbide mononitrate 40 mg twice daily.

On examination: vital signs revealed a regular pulse rate of 93 bpm, blood pressure of 130/80 mmHg, temperature of 36.8 ºC, a respiratory rate of 14/min and oxygen saturation of 100% on room air by pulse oximetry. There was clinical evidence of mild dehydration; otherwise systemic examination was unremarkable.

Investigation results were as follows: CBC: WBC = 7.8 X 10^9/l, Hb = 11.2 g/dl, Platelets= 212 X 10^9/l. Biochemical profile: SNa = 135 mmol/l, SK = 9.6 mmol/l, blood urea = 20.6 mmol/l, S creatinine = 229 mmol/l, S Mg = 0.83 mmol/l, S PO4 = 1.05 mmol/l, SCl = 113.8 mmol/l, blood sugar = 9.2 mmol/l, pH = 7.17, HCO3 = 11.7 mmol/l, base excess = -15.5 mmol/l, PO2 = 12.1 KPa and PCO2 = 4.3 KPa.

The blood sample was not hemolyzed and was obtained without mechanical difficulty or prolonged ischemia. Repeated determination of the S.

Address correspondence to:
Dr. Tarek Dowod, P. O. Box 47854, Fahaheel, Kuwait. Tel: +965-6052897, +965-3718245, E-mail: doriya70@hotmail.com

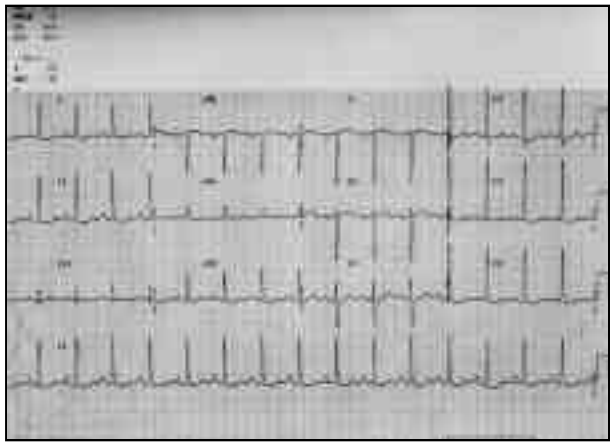

Fig 1: ECG of the patient obtained on admission when the serum potassium concentration was 9.6 mmol/l. Note that the T waves are not peaked, QRS complexes are of normal duration and that the Pwaves are also present. There are minor ST and T wave changes in I, II aVL, V_3-V_6.

potassium concentration confirmed the initial value.

Despite the severe hyperkalemia, the ECG (Fig. 1) showed normal sinus rhythm, normal axis, PR = 0.19 ms, QRS = 0.07 ms with normal P and QRS morphology and non-specific minor ST and T wave changes.

Management: The patient was admitted to the ICU for close monitoring and management. She was given intravenous fluids, calcium gluconate, glucose-insulin infusion and sodium bicarbonate.

The repeated S. potassium level continued to remain high (SK > 9.6 mmol/l); so another cycle of calcium gluconate, glucose-insulin infusion and sodium bicarbonate was administered with no effect; when the repeated S. potassium level was 10.3 mmol/l, an emergency dialysis was ordered. Dialysis was performed for three hours without complications and after one session of dialysis, the patient recovered quickly and the S. potassium became normal (SK= 4.8 mmol/l). The blood urea and S. creatinine also returned to their usual baseline values (blood urea =11 mmol/l, S. creatinine = 140 mmol/l). There were no enzyme changes suggestive of recent myocardial infarction. When the S. potassium was 4.8 mmol/l, a repeat ECG was unchanged from the one taken on admission.

DISCUSSION

Hyperkalemia in this case obviously developed due to progressive deterioration in renal function and was probably further accentuated by the potassium sparing diuretics, digoxin and valsartan therapy.

In hyperkalemia of such severity, typical ECG changes have been documented[10-11]. In two human studies, volunteers who ingested large quantities of potassium salts developed the expected ECG abnormalities[12-13]. In another human study, Thomson[14] found that oral administration of potassium chloride or potassium citrate increased the height of the T waves in 15 out of 24 subjects but all individuals who developed S. potassium > 6.5 mmol/l exhibited peaked T waves. When Dreifus and Pick[15] correlated ECG and electrolyte abnormalities, they found that while only half of the patients with S. potassium ≥ 5.6 mmol/l had associated ECG changes, all had such changes when the S. potassium exceeded 6.7 mmol/l. In contrast, Tarail[16] found that patients with renal insufficiency did not consistently have ECG changes typical of hyperkalemia until the S. potassium concentration exceeded 7.6 mmol/l.

Laboratory errors could possibly have explained one of the high potassium values. However, persistent elevated values with a consistent time pattern were obtained, with no indication of hemolysis in the plasma samples. Hyperkalemia, although less pronounced, without ECG changes have been reported in diabetics on amiloride, Addison patients and in those on triametrene[17].

There is no explanation for the lack of ECG changes correlated with the severe hyperkalemia. Perhaps, if the S. potassium rises sufficiently slowly, the myocardial transmembrane potential is maintained by other compensatory changes. In this regard, there is some evidence that the rate of change in S. potassium concentration rather than the final value achieved is more important in producing cardio-toxicity[18]. This may explain the observation that patients with chronic renal failure appears to tolerate higher levels of S. potassium than patients without chronic renal failure and this may be related to the lack of ECG changes as seen in our case.

Increase in S. calcium concentration may minimize the effect of hyperkalemia on the heart[2-4]. In our case, the S. calcium level was normal. However since the ionized calcium level was not measured and the patient was acidemic this possibility cannot be excluded. Two additional explanations can also be proposed. Increased S. sodium concentration can also abolish the ECG effects of an elevated S. potassium level[2-4]. Therefore, the elevated S. sodium level might have counteracted the ECG changes of hyperkalemia; however this is not the case in our patient as the S. sodium level was normal. Finally, patients may have had ECG changes suggestive of myocardial damage which might have obscured changes typical of hyperkalemia. Whatever the explanation, this case underscores the lack of correlation between ECG changes and profound hyperkalemia.

CONCLUSION

An ECG without typical findings of hyperkalemia does not exclude the presence of significant hyperkalemia and the ECG may not reliably monitor changes in the S. potassium concentration.

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