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Case Report

Severe hyperkalemia with normal electrocardiogram

Sanjay Sharma, Harish Gupta, Meena Ghosh, Anantanarayan Padmanabhan

Abstract

We report a case of severe hyperkalemia and normal electrocardiogram in an ex-preterm infant with renal failure due to bilateral obstruction of pelvicalyceal system caused by renal fungal mycetoma. In spite of maximal serum potassium of 11.3 mmol/L the ECG did not show any changes associated with hyperkalemia.

Key words: Electrocardiogram, hyperkalemia, renal failure

Introduction

The ECG is generally considered to be a sensitive indicator of hyperkalemia and physicians often use ECG to confirm the presence of severe hyperkalemia and as a guide to the choice of therapeutic options. However there are occasional case reports of severe hyperkalemia associated with minimal ECG changes and even rare instances of absolutely normal ECG in the presence of severe hyperkalemia.

Case Report

A 55-day-old infant (post conceptional age 37 weeks) was admitted with one day history of poor activity, poor feeding and decreased urination, a week after her discharge from the special care baby unit. All investigations prior to discharge were normal. She was admitted to rule out late onset sepsis / metabolic problem. Her weight was 1.93 kg, vitals were normal and systemic examination was unremarkable except lethargy. She was born at 29 weeks gestation with a birth wt of 1.09 kg. She was ventilated for two days for hyaline membrane disease, had umbilical lines inserted and received parenteral nutrition for six days. She received two weeks of antibiotics for presumed sepsis along with five days of reventilation. Weight at discharge was 1.87 kg and all investigations prior to discharge were normal.

At the time of readmission her investigations revealed, Hb-11.8, WBC-31.4 K/µL (Lymphocytes-6.7 K/µL, a shift to left) Platelets-834 k/µL, Hct-37.4%, RBS- 5.6 mmol/L, Urea- 16 mmol/L, creatinine- 606 µmol/L, serum sodium-123 mmol/L, potassium -11.3 mmol/L, chloride-96.8 mmol/L, serum calcium 2.5 mmol/L. ABG on room air -pH 7.358, Po2 75.6 mm Hg, Pco2 33.7 mm Hg, HCO3 18 mmol/L, Base excess - 6.8, Anion gap-18.3

The blood samples were not hemolysed and were obtained from an easy free-flowing venous sample [Figure 1].

ECG showed that the HR- was 148 /min, regular, PR interval was 0.12 sec (normal for age and HR), no ST

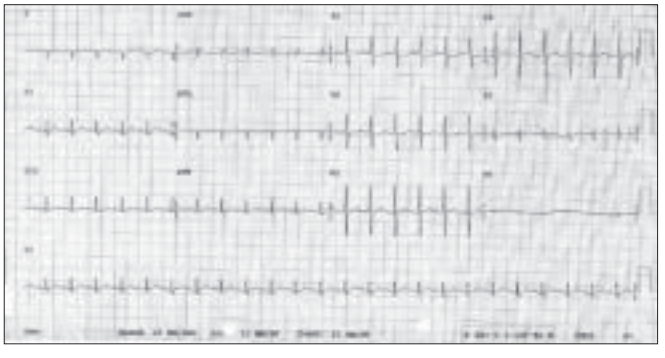

Figure 1: 12 lead ECG showing a normal tracing

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segment changes were seen, QRS duration was normal and QTc interval was also normal. No tall, tented T waves were seen.

The patient was treated with intravenous fluids, IV antibiotics, calcium gluconate, sodium bicarbonate, insulin glucose drip and nebulised salbutamol. Despite repeated cycles of medical management of hyperkalemia, the lowest serum potassium attained was 8.8 mmol/L. The patient was transferred to a tertiary care hospital the next day for dialysis therapy after consultation with a pediatric nephrologist. Work-up at tertiary care revealed the cause of renal failure and hyperkalemia as disseminated candida sepsis with candida endophthalmitis and bilateral renal tract obstruction due to Candida Bezoars.

Discussion

Potassium is the major intracellular cation (150 mmol/L), the extracellular concentration being about 4 mmol/L. This creates a large concentration gradient across the cell membrane which is important for maintaining the resting membrane potential. Thus potassium is critical for the excitability of nerve and muscle cells and for the contractibility of cardiac, skeletal and smooth muscles.

Major consequences of hyperkalemia result from its neuromuscular effects. The most prominent effects of hyperkalemia are on the myocardium.[1] Decrease in resting membrane potential decreases myocardial cell conduction velocity and increases the rate of depolarization. The decrease in conduction velocity leads to increase in the PR interval and the width of QRS complex on ECG. The increased repolarisation rate leads to increased height of the T wave. A slow conduction velocity especially in the presence of peaked T waves increases the chance that ventricular fibrillation may develop, leading to death. Mild hyperkalemia (Serum potassium 5.5-- 6 mmol/L) is associated with T wave changes, known as tenting. More severe hyperkalemia is associated with delayed conduction through His Purkinje system and the ventricular myocardium, resulting in an increase in PR interval and longer QRS duration, followed by progressive flattening and eventual absence of P waves. Under extreme conditions, QRS complex widens sufficiently so that it merges with the T wave, resulting in sine wave pattern.[2] The correlation between absolute levels of potassium and ECG changes is not precise.

Thomson found that oral administration of potassium increased the height of T waves in 15 out of 124 subjects but all individuals who developed serum potassium >6.6 mmol/L exhibited peaked T waves.[3] Tarail found that patients with renal insufficiency did not consistently have changes typical of hyperkalemia until serum potassium concentration exceeded 7.6 mmol/L.[4] It has been postulated that levels of potassium greater than 8 mmol/L are almost always associated with classical ECG manifestations.

Atypical or nonspecific ECG changes described in profound hyperkalemia include arrhythmias, depression or elevation of ST segment, decrease in height of R wave with development of deep S waves, QRS axis shift to the right or left, bundle branch blocks and sino-atrial exit blocks.[5]

Hyperkalemia in children is commonly fictitious. Fictitious hyperkalemia or pseudohyperkalemia is typically caused by hemolysis during venipuncture (by either excessive vacuum of the blood draw or by a syringe needle that is of too fine a gauge, with heel or finger prick sampling as well as traumatic venipuncture commonly performed on children). Excessive tourniquet time, too tight tourniquet or fist clenching during phlebotomy (which presumably leads to efflux of potassium from the muscle cells into the bloodstream) are other important cause of fictitious hyperkalemia. Drawing blood samples from a vein or line into which potassium is being infused can also cause pseudohyperkalemia. Pseudohyperkalemia should be avoided by taking necessary precautions during blood sampling.

Our case is interesting in that there were no ECG changes in spite of very high potassium levels; reports of such cases are rarely seen.[6] Several explanations have been forwarded for lack of ECG changes in severe hyperkalemia. LVH and intraventricular conduction defects mask ECG manifestations of hyperkalemia.[7] Acidosis, hypoxia, hyponatremia and hypocalcemia increase myocardial sensitivity to hyperkalemia whereas hypernatremia or hypercalcemia may minimize the effect of hyperkalemia on the heart.[7,8]

Finally the rate of rise of serum potassium may also influence the development of ECG changes.

Rapid increases in serum potassium may potentiate the cardio-toxic effects of hyperkalemia, whereas if hyperkalemia develops slowly cardiac manifestations may be attenuated.[9] In our case serum potassium levels were consistently very high (8.8 -11.3 mmol/L) despite intensive medical management, yet surprisingly repeated ECG tracing did not show any ECG evidence of hyperkalemia. There was no associated hypernatremia or hypercalcemia that could explain absent ECG changes. The rise in potassium levels and renal impairment had occurred over a period of a week as the child had been discharged well within a week prior to the present admission - may be this could explain the absence of ECG changes.

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