

Case Report

Non-dialytic management of acute life-threatening hyperkalaemia in an elderly patient with chronic kidney disease

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Introduction

Hyperkalaemia is a potentially fatal, yet treatable, electrolyte abnormality, defined as a serum potassium level greater than 5.5mEq/L. A level exceeding 7mEq/L is considered severe hyperkalaemia [1]. Hyperkalaemia is uncommon in patients without renal disease. The prevalence of severe hyperkalaemia in pre-dialysis chronic kidney disease patients is 31.5 % [2,3]. The mortality rate in severe hyperkalaemia is around 30% [4]. Rapid and effective treatment should be provided to reverse this fatal condition.

We report a case of severe hyperkalaemia ($K^+=10\text{mEq/L}$) in an elderly patient with multiple co-morbidities and moderate chronic kidney disease, successfully managed without haemodialysis.

Case presentation

An 88-year-old man with diabetes mellitus, hypertension, chronic kidney disease and bronchial asthma was admitted to hospital with breathlessness and generalised weakness of 2 days duration. He was taking frusemide, metformin, gliclazide, losartan, rosuvastatin, finasteride, salmeterol-fluticasone inhaler, salbutamol inhaler and deriphyllin on a regular basis.

On examination, he was dyspnoeic, lethargic, dehydrated and drowsy with a Glasgow coma scale (GCS) of 11. Pulse rate was 135 bpm, blood pressure 130/80 mmHg, respiratory rate 32/min and there were bilateral diffuse rhonchi. Oxygen saturation on air was 97%.

Laboratory results revealed a serum potassium of 10mEq/L, sodium 136mEq/L and creatinine 2.4 mg/dL. Urine analysis showed proteinuria (300mg/dL), field full of pus cells and 10 RBCs/field. The blood counts showed WBC $13220/\text{mm}^3$, haemoglobin 13.29g/dL and platelets $308 \times 10^9/\text{L}$. The C reactive protein (CRP) was 103. Chest radiograph was normal. An electrocardiogram showed tall T waves and marginally widened QRS complexes (Figure 1).

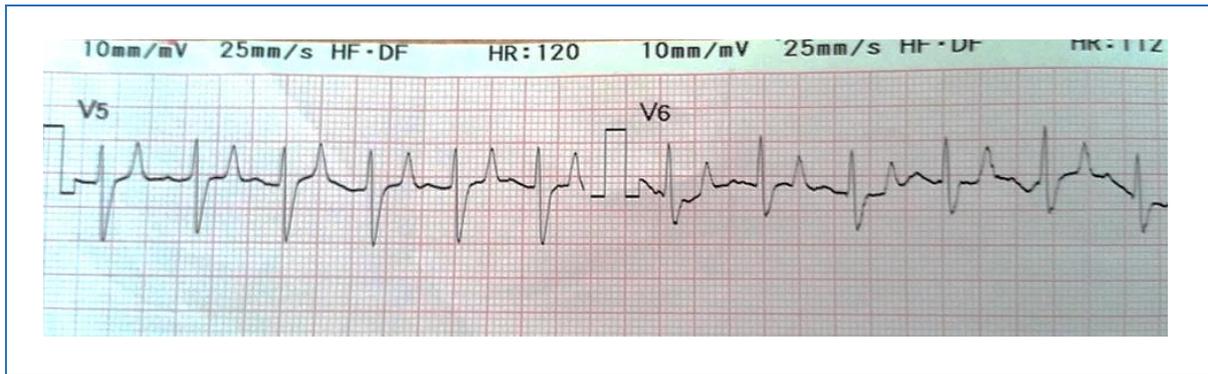


Figure 1: ECG showing features of hyperkalaemia

Hyperkalaemia was treated with intravenous (IV) fluids, 10ml of intravenous 10% calcium gluconate, 10 units of intravenous insulin in 50ml of 50% dextrose 8 hourly, salbutamol nebulisation, intravenous frusemide 20mg three times daily and calcium resonium 15g three times daily. Losartan was stopped and he was started on a low potassium diet via a nasogastric tube. The reduction of serum potassium level with treatment is shown Figure 2. During this 28-hour period his urine output remained very good (1.2ml/kg/hr) and he was clinically and haemodynamically stable.

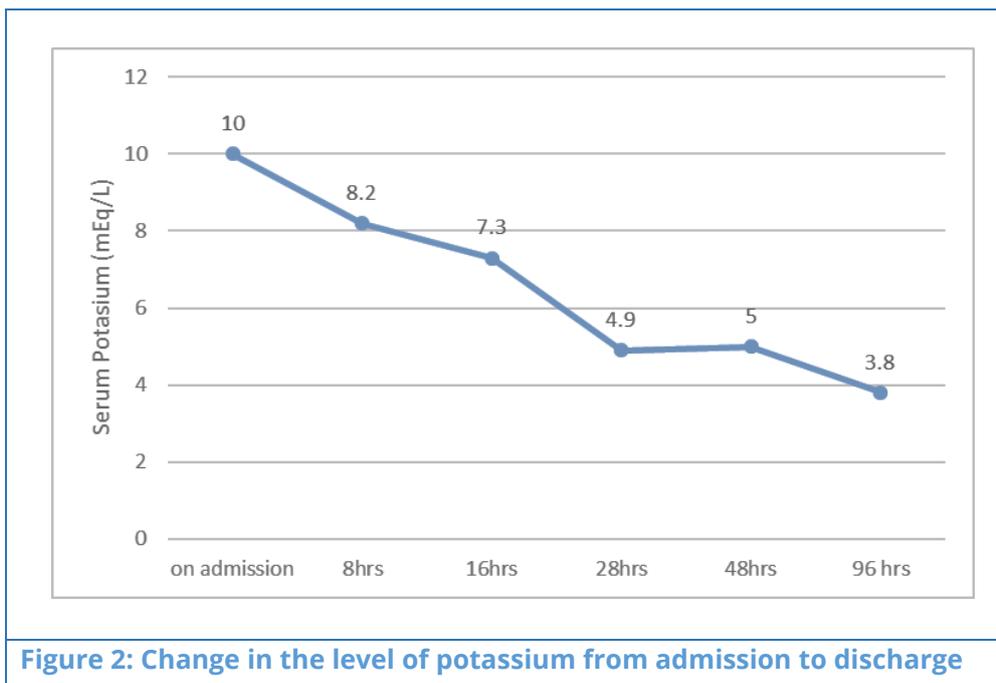


Figure 2: Change in the level of potassium from admission to discharge

The potential urine infection, suggested by the urine analysis, was treated with IV cefotaxime 1g twice daily. The patient's overall condition gradually improved and he was discharged 3 days after admission. His pre-discharge potassium and serum creatinine were 3.8mEq/L and 1.6mg/dL respectively.

Discussion

This case describes an 88-year-old elderly patient with multiple co-morbidities presenting with severe hyperkalaemia and minimal electrocardiographic changes, managed without haemodialysis. There were three factors identified as contributing to hyperkalaemia in

this patient. Firstly, he was already diagnosed with chronic kidney disease and the urine infection may have caused an acute reversible deterioration of kidney function. Secondly, he was taking losartan for a long period. Finally, he was consuming significant quantities of bananas which contain high levels of potassium (one banana contains 18.5 mEq) [5]. In hyperkalaemia, classic electrocardiographic changes range from peaked T waves in mild cases ($K^+=5-6.5\text{mEq/L}$) to diminished P waves and prolonged QRS waves in moderate cases ($K^+=6.5-8.5\text{mEq/L}$). In severe cases ($K^+>7\text{mEq/L}$), progressive widening of QRS complexes and sine waves can be seen. Although our patient had severe hyperkalaemia with a potassium level of 10mEq/L , his electrocardiographic changes did not reflect the severity of hyperkalaemia. This phenomenon has been observed in several cases where there has been a poor correlation between severity of hyperkalaemia and degree of ECG changes, and severe life-threatening hyperkalaemia can occur without typical ECG changes [6].

Haemodialysis was not considered in this patient as he was haemodynamically stable and maintained good urine output enabling renal elimination of potassium. Further the haemodialysis services were not readily available in the hospital and the patient would have had to be transferred to another hospital for such facilities.

In the literature, there are at least 10 reported cases with severe hyperkalaemia and renal dysfunction, managed without haemodialysis [6]. When compared to these reports, this patient was much older and had more co-morbidities, with a very high serum potassium level of 10mEq/L . It has been observed that in patients with hyperkalaemia with normal renal function, rate of potassium excretion by haemodialysis is similar to those who were managed without haemodialysis [6].

Conclusion

Severe hyperkalaemia is a life-threatening condition. Cardiac arrhythmia is often an unpredictable cause of death. ECG is an unreliable indicator regarding the severity of hyperkalaemia. Rapid reduction of serum potassium levels with calcium gluconate, insulin dextrose, frusemide and resins is safe and effective. It has been mentioned that medical management alone is sufficient in those with normal renal function while it should be considered as an essential intervention in those with renal dysfunction [6].

However, we feel that in patients with renal failure with a good urine output, severe hyperkalaemia can be effectively treated without haemodialysis: as shown in our case. This is an important therapeutic observation based on which medical management could be initiated especially in centres where facilities for urgent haemodialysis is not always readily available.

Abbreviations

ECG: Electrocardiogram, WBC: White Cell Count, RBC: Red Blood Cells

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