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Life threatening hyperkalemia treated with prolonged continuous insulin infusion

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ABSTRACT

Hyperkalemia is a life threatening electrolyte imbalance that may be fatal if not treated appropriately. There are multiple medications used to treat hyperkalemia to lower it to a safe level. We report a case of a 4-month old infant with Pseudohypoaldosteronism who had cardiac arrest secondary to severe hyperkalemia of 12.3mmol/l. It was refractory to anti hyperkalemic medications that necessitated the transfer of the patient to a tertiary hospital for dialysis. The potassium level has dropped gradually to a normal level with continuous insulin infusion and dextrose for almost 12 hours that waned the need of the dialysis. This case highlights the effectiveness of prolonged continuous insulin infusion in treating life-threatening hyperkalemia especially in hospitals where there are no dialysis services available or until the dialysis is initiated.

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1. Introduction

Hyperkalemia is a life-threatening electrolyte imbalance that can be fatal if not detected and treated promptly. Pseudohypoaldosteronism (PHA) is a rare hereditary disorder caused by resistance to the aldosterone action [1]. It is characterized by hyperkalemia, hyponatremia, metabolic acidosis, and high plasma aldosterone and renin concentrations [2].

Patients with PHA can develop severe and lethal hyperkalemia.

There are multiple therapeutic strategies to treat hyperkalemia that aim to protect the heart from arrhythmias, eliminate potassium from the body and shift potassium into the cells [3].

We report a case with PHA who presented to a secondary hospital with vomiting, diarrhea and low activity and had cardiac arrest secondary to severe hyperkalemia of 12.3 mmol/l. His hyperkalemia was refractory to conventional management that necessitated transferring him to a tertiary hospital for dialysis. His potassium level dropped down with continuous insulin infusion and dextrose that waned the need of dialysis.

There is no previous report to our knowledge of using continuous insulin infusion and dextrose for more than 60 minutes to

treat acute hyperkalemia [3]. Insulin infusion and dextrose was continuously given to the patient for 12 hours and potassium level has dropped gradually to a normal level.

2. Patient case presentation

A 4-month old infant known to have PHA presented to a secondary hospital with history of vomiting, diarrhea and reduced level of activity. On clinical examination, signs of severe dehydration that includes tachycardia, dry mucus membrane, sunken eyes, delayed capillary refill time, mottled skins and crying without tears were found. He was given normal saline boluses and started on intravenous fluid for severe dehydration while waiting blood investigation results. Suddenly, he developed cardiac arrest with asystole rhythm and started on cardiopulmonary resuscitation. Laboratory result showed hyperkalemia K^+ : 12.3 mmol/l, hyponatremia Na: 119 mmol/l and metabolic acidosis: Ph: 7.23, HCO_3^- : 14, lactate: 2.8. During resuscitation he received adrenaline bolus, calcium gluconate, sodium bicarbonate, insulin and dextrose and was intubated. Spontaneous circulation returned after 10 minutes of resuscitation. ECG after ROSC showed peaked tented T wave (Fig. 1).

Repeated investigations after resuscitation showed potassium of 8 mmol/l. He was given additional doses of calcium gluconate as cardiac cell stabilizer, hyperkalemic medications including nebulized salbutamol, sodium bicarbonate 1mmol/kg, insulin 0.1IU/kg with Dextrose 10% 5ml/kg and calcium polystyrene sulfonate (potassium binding resin). After 1 hour, his potassium level dropped

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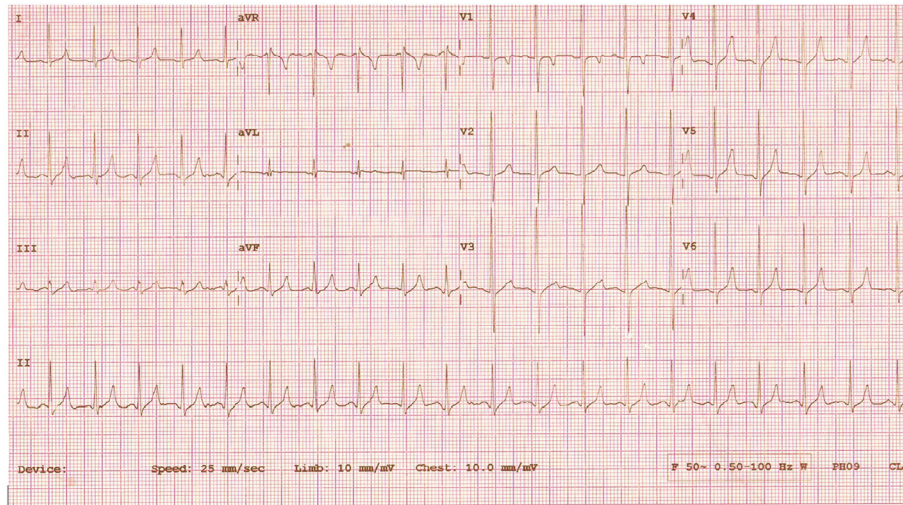


Fig. 1. ECG shows peaked tented T wave which indicate hyperkalemia.

down to 7.3 mmol/l and was given additional doses of nebulized salbutamol, insulin & dextrose and sodium bicarbonate. He had prerenal azotemia secondary to dehydration. His initial urea reading was 10 mmol/l (normal: 1.8–6) and serum creatinine was 62 $\mu\text{mol/l}$ (normal: 30–55). Despite good hydration, normalization of renal function test (serum creatinine: 39 $\mu\text{mol/l}$) and frequent doses of anti-hyperkalemic medications, his potassium level climbed again to 9.45 mmol/l. Therefore, he was referred urgently to our tertiary hospital for dialysis as there was no dialysis services in the secondary hospital. As the transfer from other hospital, arrangement and starting dialysis took at least 3 hours and the patient had refractory hyperkalemia, we advised the treating physician to start continuous insulin infusion and dextrose with frequent monitoring of blood glucose level. He was started on insulin infusion of 0.1 IU/kg/hr and dextrose 10% + 0.9% NaCl as maintenance fluid therapy. He arrived to our pediatric intensive care unit after 4 hours of starting continuous insulin infusion. Additional normal saline

boluses were given. His potassium level was 7.8 mmol/l and his blood glucose level was 2.2 mmol/l so started on side drip Dextrose 25% at 2 ml/kg/hr in addition to his maintenance intravenous fluid of D10% + 0.9% NaCl to avoid hypoglycemia. Pediatric surgeon was consulted for peritoneal dialysis drain insertion and parent were consented. Potassium level before peritoneal drain insertion dropped to 7.1 mmol/l and the patient was hemodynamically stable and ECG was normal. We decided to hold dialysis and to continue insulin infusion and dextrose with close monitoring of potassium level and blood glucose. His potassium level continued to drop gradually and no episodes of hypoglycemia was recorded (Fig. 2). Insulin and dextrose infusion were discontinued when potassium level dropped to less than 4 mmol/l. No rebound of hyperkalemia was noticed. The total duration of continuous insulin infusion was 12 hours. Sodium polystyrene sulfonate 1 gram/kg/dose was given 6 hrly for body potassium elimination. The child was extubated and shifted to pediatric ward under endocrinologist care.

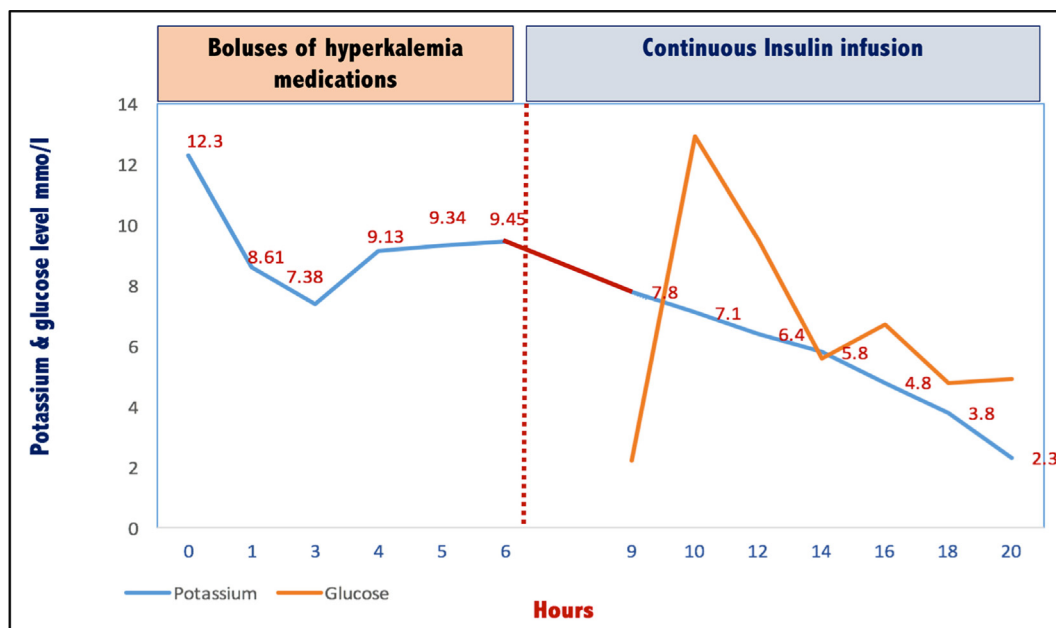


Fig. 2. Left side of the graph showed initial drop of potassium level then rising up despite boluses of hyperkalemia medications. Right side of the graph showed continuous drop of potassium to normal level after initiation of continuous insulin infusion (blue line). Brown line shows glucose level during insulin infusion.

3. Discussion

Hyperkalemia is a life-threatening condition that requires early recognition and aggressive treatment [1]. There are multiple therapeutic agents used to treat hyperkalemia alone or in combination for its prompt management [4]. Although hyperkalemia is one of the deadliest electrolyte abnormalities, it is also one of the most treatable [5]. The aims of severe hyperkalemia managements are to antagonize its electrophysiologic effects on myocardium (membrane stabilization), redistribute potassium from extracellular to intracellular compartment and removal of potassium from the body [4,5]. Membrane stabilization is achieved by infusion of calcium but it does not affect the serum concentration of potassium [6]. Distribution of potassium between the intracellular and the extracellular fluid compartments is regulated by physiologic factors such as insulin and catecholamines which stimulate the activity of the $\text{Na}^+ - \text{K}^+ \text{ATPase}$ [6]. The most frequent and the first-line medication used in emergency setting to shift potassium into cells is intravenous short acting insulin which lowers the serum potassium level by 1 mEq/L (mmol/L) within 10–20 minutes [5–9]. It is administered with glucose as it can induce severe hypoglycemia. The optimal dose, route of administration and duration of insulin in the management of emergency hyperkalemia is controversial [3,8]. It was administered as a bolus in a number of trials [10–14]. In other studies, it was given as an infusion over 15 minutes [15], over 30 minutes [16,17] and over 60 minutes [18–20]. The hypokalemic effect of insulin can be seen within 20 minutes, peaks between 30 and 60 minutes, and may last for up to 6 hours [21]. In our case, insulin was infused over 12 hours continuously till serum potassium dropped to normal level (Fig. 2). We did not find any clinical trial where insulin was infused over more than 60 minutes. Our patient had cardiac arrest secondary to a very high potassium level of 12.3 mmol/l and responded initially to potassium lowering agents in the first 3 hours of management. In the subsequent hours it started to rise again despite assurance that he was not receiving any exogenous potassium and although he received fluid boluses and multiple doses of nebulized salbutamol, insulin and sodium bicarbonate. He was given also potassium binding resin that has slow onset and delayed action by its binding of K in the intestinal lumen [22]. If hyperkalemia is refractory to the medical management, dialysis is the definitive and most effective hypokalaemic measure. The patient was in a hospital where there were no pediatric dialysis services available and the closest dialysis center was around 1.5 hour away. The transfer of the patient to a tertiary center and the initiation of the dialysis was estimated to be at least 3 hours. Since the patient had refractory life threatening hyperkalemia and already had cardiac arrest secondary to a very high level of potassium and till dialysis is initiated, he was started on continuous insulin infusion with a close monitoring of blood glucose level. There was one asymptomatic drop of blood glucose (2.2 mmol/l) after three hours of initiation of insulin infusion that was overcome by increasing intravenous dextrose concentration after which no hypoglycemia was documented. Potassium level was impressively dropping down with continuous insulin infusion that changed the plan of initiation of dialysis. It should not be the standard of care but in a setting where there is no dialysis or the dialysis cannot be initiated early due to a technical issue or patient instability, continuous insulin infusion may help in lowering potassium level and can be a lifesaving in life threatening hyperkalemia while monitoring blood glucose levels very closely.

4. Conclusion

Hyperkalemia is a life-threatening electrolyte imbalance that

needs prompt and aggressive management. This case highlights the effectiveness of prolonged continuous insulin infusion in treating hyperkalemia especially in a setting where there are no dialysis services available or till the dialysis is initiated.

Conflicts of interest

We have no conflicts of interest to disclose.

Ethical statement

I testify on behalf of all co-authors that our case report with title, Life threatening hyperkalemia treated with prolonged continuous insulin infusion, has not been published in whole or in part elsewhere and not currently being considered for publication in another journal. All authors have been personally and actively involved in substantive work leading to the manuscript, and will hold themselves jointly and individually responsible for its content.

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