Life threatening hyperkalemia treated with prolonged continuous insulin infusion

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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 12.3 mmol/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 waved 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 reduced level of activity. On clinical examination, signs of severe dehydration that included tachycardia, dry mucous 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 metabolic acidosis: Ph:7.23, HCO3: 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 1 mmol/kg, insulin 0.1 IU/kg with Dextrose 10% 5ml/kg and calcium polystyrene sulfonate (potassium binding resin). After 1 hour, his potassium level dropped to 8 mmol/l.

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 mucous 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, HCO3: 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).

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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 μmol/l (normal: 30–55). Despite good hydration, normalization of renal function test (serum creatinine: 39 μmol/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. 1. ECG shows peaked tented T wave which indicate hyperkalemia.

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 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.

References

[1] Attia Najya A, Marzouk Yousef I. Pseudohypoaldosteronism in a neonate presenting as life-threatening hyperkalaemia. Arch Dis Child 2002;86(3):219–21.
[2] Dillon MJ, Leonard JV, Buckler JM, et al. Pseudohypoaldosteronism. Arch Dis Child 1996;74(2):149–50.
[3] Harel Ziv, Kamel Kamel S. Optimal dose and method of administration of intravenous insulin in the management emergency hyperkalaemia: a systematic review. PLoS One 2016;11(5):e0154963.
[4] Parham Walter A, Mehdirad Ali A, Biermann Kurt M, Fredman Carey S. Hyperkalemia revisited. Tex Heart Inst J 2006;33(1):40–7.
[5] Li Tingting, Vijayan Anitha. Insulin for the treatment of hyperkalaemia: a double-edged sword? Clin Kidney J 2014;7:239–41.
[6] Lawrence S. Weisberg, Management of severe hyperkalaemia. Crit Care Med 2008;36(12):3246–51.
[7] Kamel KS, Wei C. Controversial issues in the treatment of hyperkalaemia. Nephrol Dial Transplant 2003;18(11):2215–8.
[8] Elliott MJ, Ronksley PE, Clase CM, Ahmed SR, Hemmelmaj BR. Management of patients with acute hyperkalaemia. CMAJ Can Med Assoc J 2010;182(15):631–5.
[9] Allon M, Copney K. Albuterol and insulin for treatment of hyperkalaemia in hemodialysis patients. Kidney Int 1990;38(5):869–72.
[10] Mushagai MA, Masood M. Treatment of hyperkalaemia with salbutamol and insulin. Pakistan J Med Sci 2006;22(2):176–9.
[11] Lens XM, Montoliu J, Cases A, Campistol JM, Revert L. Treatment of hyperkalaemia in renal failure: salbutamol v. insulin. Nephrol Dialysis Transplant Off publ Eur Dial Transpl Soc—Eur Ren Ass Soc 1989;4(3):228–32.
[12] Just D, Rumboldt Z. Should glucose be administered before, with, or after insulin, in the management of hyperkalaemia? Ren Fail 1993;15(1):73–6.
[13] Chothia MY, Halperin ML, Rensberg MA, Hassan MS, Davids MR. Bolus administration of intravenous glucose in the treatment of hyperkalaemia: a randomized controlled trial. Nephron Physiol 2014;129(1):1–8.
[14] Ngugi NN, McLigeyo SO, Kayima JK. Treatment of hyperkalaemia by altering the transcellular gradient in patients with renal failure: effect of various therapeutic approaches. East Afr Med J 1997;74(8):503–9.
[15] Mahajan SK, Mangla M, Kishore K. Comparison of aminophylline and insulin-dextrose infusions in acute therapy of hyperkalaemia in end-stage renal dis ease patients. J Assoc Phys India 2001;49:1082–5.
[16] Duraran M, Ates K, Erurtuk S, Duman N, Karatan O, Erbay B, et al. Comparison of aminophylline and insulin infusions in treatment of hyperkalaemia in patients with end-stage renal disease. Nephron 1996;73(1):105.
[17] Kim HJ. Combined effect of bicarbonate and insulin in acute therapy of hyperkalaemia in end-stage renal disease patients. Nephron 1996;72(3):476–82.
[18] Blumberg A, Weidmann P, Shaw S, Gnadinger M. Effect of various therapeutic approaches on plasma potassium and major regulating factors in terminal renal failure. Am J Med 1988;85(4):507–12.
[19] Allon M, Shanklin N. Effect of bicarbonate administration on plasma potassium in dialysis patients: interactions with insulin and albuterol. Am J Kidney Dis: Off J Natl Kidney Found 1996;28(4):308–14.
[20] Daly Kayleen, Farrington Elizabeth. Hyperkalemia and hyperkalaemia in infants and children: pathophysiology and treatment. J Pediatr Health Care 2013;27(6):486–96.
[21] Aheer P, Crowe AV. The management of hyperkalaemia in the emergency department. J Accid Emerg Med 2000;17:188–91.