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Mohd Zailani Abd Shariff[1], Ahmad Hariz Ahmad Jaafar[1], Chan Pei Fong[1]

[1] Department of Emergency and Trauma, Hospital Raja Permaisuri Bainun, Ipoh

Introduction

Renal tubular acidosis (RTA) is a condition in which kidneys are unable to remove acids from the blood into the urine as they should. Distal renal tubular acidosis (dRTA), also known as type 1 RTA is caused by impaired net excretion of acids by the distal tubules. It is characterized by non-anion gap metabolic acidosis and severe hypokalemia as illustrated in this case.

Case report

We report a case of a 36 years old Malay lady with underlying dRTA causing her to have multiple admissions for symptomatic hypokalemia. She presented with generalized body weakness and vomiting for 1 day. Physical exam revealed a lethargic lady with normal mental status and quadriplegia. She was intubated for respiratory distress within two hours of admission. Electrocardiogram (ECG) showed generalized ST depression, prominent U wave and pseudo-prolonged QTc (refer to Figure 1). She was immediately started on intravenous potassium chloride (KCL) 3 gram in 500 ml normal saline over 3 hours and intravenous magnesium sulphate 2 gram over 20 minutes.

Despite multiple corrections of potassium via central venous access, her serum potassium level remained persistently low initially (refer to Table 1). She received a total potassium correction 11gram over 24 hours stay at the emergency department. She also developed hypernatremia from large volume of normal saline used during the potassium correction.

She was managed for sepsis at the intensive care unit and underwent hemodialysis for acute kidney injury. Her potassium level gradually improved with Shohl's solution, sodium bicarbonate (NaHCo3), oral potassium supplements and spironolactone. She recovered and was discharged well after 11 days.

	26/7/21			27/7/21		28/7/21				6/8/21
	1300H	1800 H	0000 H	700H	1200 H	0000H	0600 H	1200 H	1800 H	2200 H	1200 H
Potassium (mmol/L)	1.4	1.3	1.9	1.7	1.5	1.9	2.0	1.9	2.2	3.8	3.8
Sodium	127	129	133		152		160	162	159	160	136
Chloride	106	108	106		132		133	136	132	135	120
Urea	4.0	4.3	4.6		5.3		7.1	9.8	11.2	13	9
Creatinine	64	60	63		117		184			423	273
Mg	1.04										
Po4	0.92										
Ca	2.15										
PH	7.17	7.09	7.12	7.08	7.2	7.0	7.2	7.1		7.1	7.3
PCo2	29	45	42	45	41	60	45	43		33	28
Po2	93	120	140	96	137	121	105	157		188	116
HCo3	10	13.6	13.4	13.1	16	13.9	17.6	15.3		11.6	17

Table 1. Serial blood investigations. Blue highlighted is result in Emergency Department, Yellow highlighted is result upon discharge.

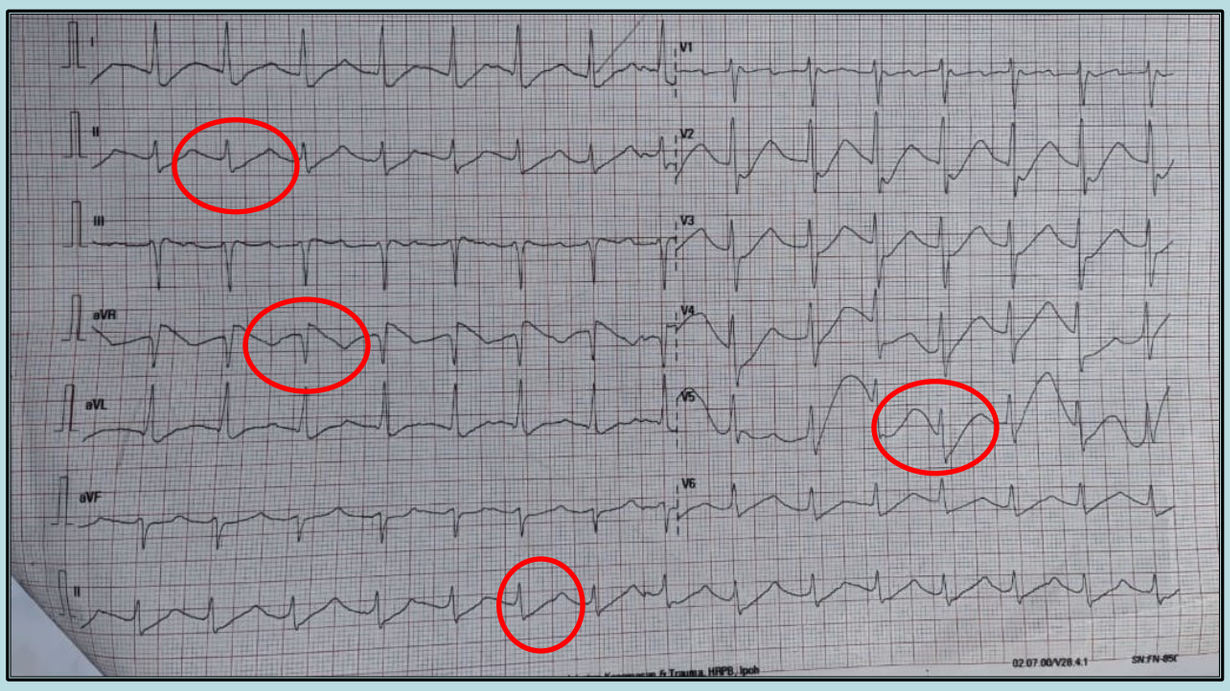

Figure 1.ECG (red circle) showed Pseudo prolonged QT due to flattened T-wave and presence of U wave

Discussion

1) Managing severe hypokalemia in a busy emergency department is a big challenge and requires fast decision making. The above case showed that a patient can rapidly deteriorate due to respiratory muscle weakness.

2) Symptomatic persistent hypokalemia requires rapid replenishment of potassium and continuous cardiac monitoring for any complications due to over or under correction.

3) Potassium correction should be limited to 10 mEq/hour via peripheral venous line and 40 mEq/hour if given through central venous line. Higher dose of potassium is painful if given via peripherally and may cause cardiac arrhythmias if given too fast.

4) Magnesium sulphate was given in this case to stabilize cardiac membrane potential and decrease cell excitability that can aggravate the adverse effects of hypokalemia in target tissues.

5) In dRTA patient, correction of potassium requires additional medication to prevent renal loss of potassium in comparison to other diseases. Sodium bicarbonate and Shohl's solution (citrate) were administrated in this case to improve metabolic acidosis and alkalinize the urine. Spironolactone was used for its potassium sparing effect.

6) Hypernatremia developed in this case due to large volume of normal saline used for potassium correction. Alternative solution such as balanced solution could been used for potassium correction.

Conclusion

1) Early recognition and prompt action is needed in patients with dRTA which can present with life-threatening hyperkalemia.
2) Faster correction rate and higher dosage of potassium via central venous access are needed to prevent cardiac and respiratory complications.
3) Treatment with sodium bicarbonate and citrate solution can help to improve metabolic acidosis and prevent potassium loss.

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DECLARATION OF CONFLICT
The authors whose names are listed certify that they have NO affiliations with or involvement in any organization or entity with any financial or nonfinancial interest.

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