

‘Doctor, they said I have high blood potassium’



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Hyperkalemia is a potentially lethal clinical condition. Potassium is mainly an intracellular ion, 98% of which resides inside the cells and this is essential in the maintenance of the transmembrane electrical potential of the cell membrane. High blood potassium interferes with the functioning of the myocardial muscles and may eventually leads to cardiac arrest. The normal concentration of plasma potassium is around 4 mmol/L while the intracellular potassium is around 120 mmol/L. Conditions like acidosis cause the movement of potassium from the cells into the extracellular space and thus causing hyperkalemia. The commonest cause of hyperkalemia includes renal impairment and acidosis. Other causes include the administration of drugs like angiotensin receptor blockers and cell damage with release of the intracellular contents in conditions like rhabdomyolysis or tumour lysis during chemotherapy.

Because of the potential serious consequence of hyperkalemia, doctors are naturally concerned when a patient’s serum potassium is above normal. A common scenario is that a patient had blood taken in a laboratory. On the next day, the nurse called the patient and informed him about high blood potassium and asked him to come back for ‘high blood potassium medicine’. The medicine is usually in the form of Resonium which is an ion exchange-resin. When taken orally, it will adsorb the potassium in the bowel in exchange for sodium (Resonium A) or calcium (Resonium C). However, it takes a long time to work and it is not palatable. A high serum level may occur in different situations and each may need different treatment.

Patient 1

A patient was followed up in a public hospital for stage 4 Chronic Kidney Disease (CKD). Her estimated glomerular filtration rate was 20 ml/minute. She had blood taken on a follow up visit and on the next day, she was called by a nurse there that she had high blood potassium and was asked to attend for high blood potassium medicine (Fig. 1).



Figure 1. The ion-exchange resin Resonium

She was very worried because she could not talk to the doctor directly for more information and she went for a ‘second opinion’. Thanks to the ‘ppi-ep^r’ [the ‘electronic patient record’ sharing] scheme, her blood test result was retrieved online. Her serum sodium was 127 mmol/l and potassium 5.4 mmol/L (Fig. 2). There was no data on her acid-base status. Her serum potassium was around 5 all along.

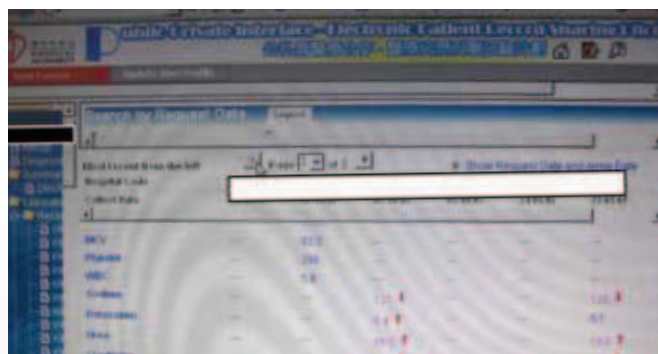


Figure 2. The lab results

She was reassured that the increase was only mild and there was no need to worry. She did not take the Resonium, and repeated blood test showed normal serum potassium.

Mild increase in serum potassium in a CKD patient may be due to drugs such as angiotensin receptor blockers (ARB) or angiotensin converting enzyme blockers. Review of the drugs and advice on low potassium diet would probably be more effective.

Patient 2

A 67 year-old female was suffering from hypertension. She was given an ARB in the form of losartan 100 mg om but the blood pressure was not well controlled. She was given a diuretic to augment the therapeutic effect of losartan. Blood tests showed Na⁺ 135 mmol/L, K⁺ 6.5 mmol/L, Cl⁻ 100 mmol/L and HCO₃⁻ 30 mmol/L. The serum creatinine was 85 µmol/L and the MDRD eGFR was 61 ml/minute. She was asymptomatic.

ARB is a good medication for hypertension, especially in patients with diabetes mellitus for the renal protective effect. One of the side effects is hyperkalemia. Thiazide diuretics can also reduce blood pressure but it may lead to hypokalemia. An ARB and a thiazide diuretic may be a suitable combination as their effects on the blood potassium tends to 'neutralize' each other. This patient was given amiloride hydrochloride (Moduretic), which is a 'potassium sparing' diuretic and can also cause hyperkalemia. This resultant effect of this drug combination in causing hyperkalemia can be marked, especially in patients with renal impairment. In this case, the amiloride was changed to a thiazide diuretics and the hyperkalemia was corrected.



Figure 3. The patient's drug combination and the lab report

Patient 3

A patient was receiving maintenance hemodialysis for end-stage renal failure and was followed up in a public renal specialty clinic. One day he had routine test done and was later called back urgently to take medicine for the hyperkalemia. His serum potassium was 6.5 mmol/L while his 'usual' potassium was 5 mmol/L. Upon arrival at the clinic, he had blood tested again. The repeated serum potassium was 5.4 mmol/L. He was asked to

continue the Resonium anyway, but the patient did not comply. He did not suffer from any mishaps.

143	141	138	137-144	mmol/L
5.0	6.5 *	5.4 *	3.5-5.0	mmol/L
17.6 *	22.4 *	24.1 *	3.1 - 7.85	mmol/L
907 *	952 *	954 *	65 - 1095	µmol/L
76	83 *		66 - 895	g/L
46	49	46	35 - 52	g/L
30	34			g/L
6	7		< 195	µmol/L
62	74	73	43 - 1055	U/L
10	13		< 675	U/L
2.38	2.42	2.26	2.15 - 2.55	mmol/L
2.28	2.28	2.16		mmol/L
1.82 *	2.39 *	2.11 *	0.72 - 1.395	mmol/L
		5.4		mmol/L

Figure 4. The laboratory report

The serum potassium may be falsely high due to hemolysis as a result of incorrect blood taking or storing the blood sample for too long. It is likely that the high blood potassium in this case was due to some technical errors as the level became normal on repeated test. Repeating potassium estimation in the lab takes time. In a private clinic with no urgent laboratory support, it might be useful to run the ECG for evidence of hyperkalemia because it gives an instant and reliable clue to the situation.

Patient 4

A patient with stage 4 CKD was admitted to hospital because of dyspnea. He had been taking some non-steroidal anti-inflammatory agents (NSAID) for a few days for bone pain. On admission, he was oliguric and his blood urea was 45 mmol/L. X-ray chest revealed acute pulmonary edema. His serum potassium was 8.1 mmol/L and the serum bicarbonate level was 10 mmol/L. The ECG showed loss of the P wave with wide QRS complexes.

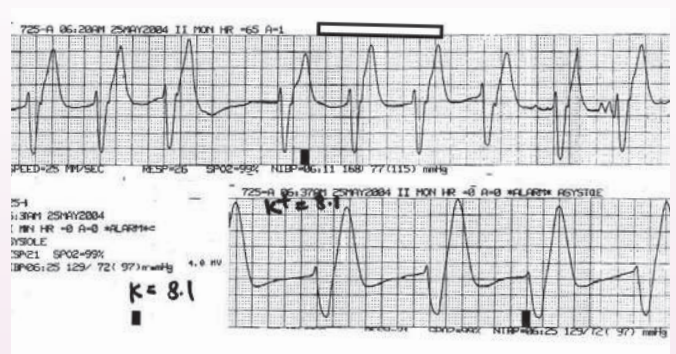


Figure 5. ECG at the start of dialysis. Note the absence of P wave and the wide QRS complexes in the lower tracing.

The patient has an acute on chronic renal failure due to NSAID administration. An acute decrease in renal function in already compromised kidneys can give rise to dangerously high serum potassium. The high serum potassium of 8.1 mmol/L and the wide QRS complexes indicated that it was a medical emergency. He was put on hemodialysis immediately. In the middle of the dialysis, the ECG monitor tracing showed the reappearance of the P wave and the QRS complexes became narrow.



Figure 6. ECG in the middle of the dialysis, note the P wave and the narrow QRS complex in the middle of the tracing.

At the end of the dialysis, the ECG was normal-looking and the serum potassium was 5.1 mmol/L. His condition was much improved due to the removal of the uremic toxins, correction of the potassium level and acidosis together with removal of excessive fluid.

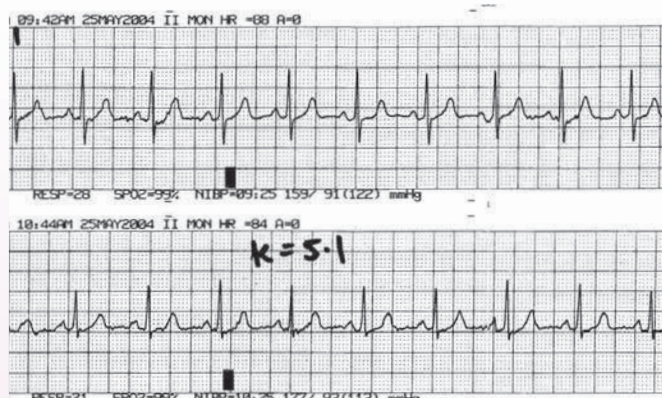


Figure 7. ECG became normal at the end of dialysis.

The above case showed that high serum potassium is a medical emergency and the best treatment is removal by hemodialysis. It can remove urea, potassium and

excessive fluid from the body. Modern dialysis uses bicarbonate in the dialysate bath. During dialysis, bicarbonate moves from the dialysate to the blood with partial correction of the acidosis. The removal of potassium by hemodialysis is rapid but since potassium is mainly an intracellular ion, after the cessation of dialysis, more intracellular potassium may move out of the cells, thus causing a 'rebound' in serum potassium on the next day (Fig. 8).



Figure 8. Hemodialysis will correct hyperkalemia, acidosis and fluid overload, while removing blood urea at the same time.

Peritoneal dialysis can also remove potassium and toxins but the efficacy is lower. The rate of potassium removal may not be adequate in patient with hypercatabolic syndrome.

While waiting for hemodialysis to be set up, one can use some measures to tide the patient over, such as the injection of calcium and insulin-glucose administration. Resonium has no place because it takes too long to act.

Conclusion

Hyperkalemia has different causes and comes with different grades of severity. It may due to technical error in blood sampling. In mild cases, management of the underlying cause is essential. It is life threatening in severe cases and immediate active treatment is required. A rapid rise in serum level is much more dangerous than a chronically elevated level. Dialysis should be considered if it is anticipated that the level will continue to rise. The ECG is a useful tool in assessment of the hyperkalemia. In severe cases, hemodialysis is effective and life-saving.