Singapore Med J 2007; 48(11) : e293

Life-threatening hyperkalaemia developing following excessive ingestion of orange juice in a patient with baseline normal renal function

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ABSTRACT

Hyperkalaemia is a less-recognised life-threatening cause of paralysis. We describe a 51-year-old African-American man, who suffered from muscle weakness progressing to ascending symmetric paralysis, and inability to masticate. Physical examination revealed flaccid paralysis with areflexia of the four limbs. Computed tomography of the brain and cervical spine did not demonstrate any organic lesions. Laboratory investigations revealed serum potassium 9.0 mEq/L (not haemolysed), blood urea nitrogen 34 mg/dL, and serum creatinine 2.0 mg/dL. Electrocardiography showed typical features of hyperkalaemia. After emergent treatment for hyperkalaemia was initiated, serum potassium was rapidly-normalised to 5 mEq/L and all neuromuscular symptoms reversed within one hour. Upon reviewing his food and medication history, he admitted drinking 2.5 litres of orange juice (which contains about 450 mg of potassium in 1,000 ml) per day for the past three weeks to quench his thirst. Hyperkalaemia should be borne in mind in the differential diagnosis of acute paralysis. Hidden sources of potassium intake, such as orange juice, should not be overlooked, even in patients with baseline normal renal function.

Keywords: baseline normal renal function, orange juice, hyperkalaemia, paralysis, potassium intake

Singapore Med J 2007;48(11):e293–e295

INTRODUCTION

Some fruit juices have a very high potassium content. Several cases of fruit juice-induced hyperkalaemia have been reported in the literature. We present a highly unusual case of a 51-year-old African-American man, with baseline normal renal function, who developed life-threatening hyperkalaemia (potassium 9.0 mEq/L) secondary to excessive orange juice consumption of about 2.5 L/day, leading to quadriparesis. Emergent treatment of hyperkalaemia saved his life. Hyperkalaemia of this extent, precipitated by a large amount of orange juice consumption and leading to quadriparesis, is extremely unusual.

CASE REPORT

A 51-year-old African-American man with a past medical history of hypertension and human immunodeficiency virus (HIV) infection diagnosed three years ago, with CD4 of 400 three weeks ago, presented to the emergency department with complaints of generalised weakness, tiredness, muscular weakness progressing to ascending symmetrical paralysis, and difficulty in mastication for the past two days. Home medications include lamivudine, abacavir, atazanavir, norvir and amlodipine. Social and family history was unremarkable. Vital signs were stable, physical examination revealed flaccid paralysis with areflexia of the four limbs. Computed tomography of the brain and cervical spine did not demonstrate any organic lesions.

Laboratory results showed markedly-elevated potassium 9.0 mEq/L (not haemolysed), blood urea nitrogen 34 mg/dL, serum creatinine 2.0 mg/dL, sodium 135 mEq/L, chloride 108 mEq/L, bicarbonate 22 mEq/L and creatinine phosphokinase 50 IU/L. The rest of the laboratory results included haemoglobin 14.4 g/dL, haematocrit 43.1%, white blood cells 10/mm^3 and platelets 263/uL. Urinalysis showed a pH of 5.5 and a trace of proteins. Liver function tests were with in normal limits. Electrocardiography (ECG) showed typical features of hyperkalaemia with tall upright T waves in all leads, PR interval of 212 and QRSD increased to 184. Emergent treatment for hyperkalaemia was instituted, which rapidly normalised serum potassium to 5.0 mEq/L and reversed all neuromuscular symptoms within a few hours. Patient blood urea nitrogen and serum creatinine also returned to baseline

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Table I. Common potassium-containing fruits and juices.

Fruit/fruit juice*	Unit (g)	K^+ concentration (mmol/1,000 g)	K^+ content (mg)
Banana*	126	89	451
Tomato juice#	227	59	533
Peach*	98	48	190
Orange juice#	227	48	436
Grapefruit juice	227	42	378
Apple juice#	22	33	295
Grape juice#	227	37	334
Honeydew melon*	1,340	65	3,500
Watermelon*	5,040	21	4,140

* 1 medium-sized fruit

\# equivalent of 8 oz

levels within the next 24 hours. Calculated trans-tubular potassium gradient (TTKG) was more than seven, consistent with normal aldosterone function. Renal ultrasonography was also unremarkable.

Upon reviewing his food and medication history, he admitted drinking approximately 2.5 L of orange juice every day (containing about 436 mg potassium in 1,000 ml juice) to quench his thirst for the past few weeks. It has been postulated that he developed dehydration as a result of hot and humid weather, which led to acute renal failure brought about by the patient's over-consumption of orange juice (rich in potassium) in attempts to quench his thirst. The patient was discharged after two days, with instructions not to rely only on orange juice to satisfy his thirst.

DISCUSSION

Potassium is the most common cation in the body. The ratio of the intracellular-to-extracellular potassium concentration is the primary determinant of the resting membrane potential (Em). Alterations in the Em disrupt the normal function of neural, cardiac, and muscular tissues. Normal serum potassium ranges from 3.5 to 5.2 mmol/L. The molecular weight of potassium is 39.1, so a daily potassium intake of 80 mmol is roughly equivalent to 3.1 g of potassium. Potassium is rapidly and completely absorbed by the small intestine. Net gastrointestinal (GI) absorption (intake minus GI losses) is approximately 90%.[1] Lower GI secretions have high concentrations of potassium, 80–90 mmol/L, but due to the limited amount of stool (80–120 g/d), daily GI excretion of potassium is only 10 mEq.[2,3] Renal excretion of potassium can range from 5 to 500 mEq/d.[4,5] Though 500 mmol of potassium is filtered by the glomerulus each day, essentially all of it is resorbed in the proximal tubule and loop of Henle. Any potassium that is ultimately excreted in the urine must be secreted by the tubules. The ability of the kidney to excrete potassium is flexible and adaptable. If dietary ingestion of potassium is increased over a number of days, the kidney increases daily potassium excretion to match. Because of this, dietary loads of potassium usually do not result in hyperkalaemia unless they are sudden, or paired with a defect in renal potassium handling. Likewise, conditions associated with the movement of intracellular potassium to the extracellular space are associated with only transient hyperkalaemia because either the kidneys excrete or the cells reuptake the excess potassium. Decrease in the ability of the kidney to excrete potassium, increases its susceptibility to hyperkalaemia from increased potassium intake or transcellular shifts distal nephron.[6] Hyperkalaemia has been reported to follow the use of potassium chloride salt substitutes, even in the presence of normal renal function.[7] One teaspoon of potassium chloride contains 50–65 mEq of potassium. Enteral nutrition supplements may be rich sources of potassium. Ensure Plus at 100 ml/hr provides 130 mEq of potassium per day.

Changes in the extracellular concentration can have dramatic effects on the resting membrane potential and the cell's ability to depolarise. As extracellular potassium rises, the normally negative Em increases toward zero; this allows easier depolarisation (i.e., increased excitability). Hyperkalaemia shortens the refractory period following depolarisation by facilitating faster potassium uptake. In the myocardium, inactivated sodium channels slow conduction velocity, and high serum potassium speeds repolarisation. On ECG, hyperkalaemia causes widened QRS complexes (slowed conduction velocity) and shortened ST intervals with tented T waves (rapid repolarisation). The slowed conduction associated with rapid repolarisation predisposes the myocardium to ventricular fibrillation. Ascending paralysis mimicking

Guillain-Barré syndrome has been documented with a serum potassium level of 7 mmol/L. In a review of all published cases of hyperkalaemic paralysis (excluding hereditary periodic paralysis), the average potassium level was 9 mmol/L. Electromyograms showed the paralysis to be due to abnormal nerve depolarisation rather than muscle pathology.[8]

Hyperkalaemic paralysis resulting from a plethora of causes can be simply divided into two groups, namely: familial hyperkalaemic periodic paralysis (HYPP) and secondary hyperkalaemic paralysis. The HYPP characterised by a suddenly-increased shift of cellular potassium into extracellular fluid is inherited as autosomal dominant, mainly due to inactivation mutations in the SCN4A gene encoding the tetrodotoxin-sensitive sodium channel of skeletal muscle.[9] The secondary hyperkalaemic paralysis can be caused by any aetiology of profound hyperkalaemia, such as excess potassium intake (salt substitutes, drugs or foods containing potassium) or potassium production (rhabdomyolysis, haemolysis), potassium redistribution (mannitol, succinylcholine, digoxin, and β-blockers) and reduced renal potassium excretion (Addison's disease, hypoaldosteronism, obstructive uropathy, NSAIDs, cyclosporine, angiotensin-converting enzyme inhibitors, and potassium-sparing agents).[10,11]

This patient suffered from the typical ascending paralysis. There were no other plausible causes for his paralysis except for extreme hyperkalaemia. Hyperkalaemia-induced paralysis was diagnosed from the fact that correction of hyperkalaemia rapidly terminated the paralysis without any sequela. He did not have the family history of paralysis to suggest familial HYPP or the secondary causes of hyperkalaemia, except the extremely high content of potassium input from the orange juice. Other important causes of acute paralysis, like Guillain-Barré Syndrome and even hypokalaemic paralysis (either primary or secondary), should also be considered in the differential diagnosis as they are also quite common and can have similar clinical findings. Because cardiac muscle is much more sensitive to hyperkalaemia, patients with hyperkalaemic paralysis usually show cardiac manifestations and typical ECG changes, like peak T-waves, prolonged P-R interval, loss of the P-wave amplitude, widening QRS complex, or "sine wave" pattern. Nevertheless, some patients with severe hyperkalaemia can manifest paralysis even without any cardiac features.[12]

The major morbidity and mortality of unrecognised hyperkalaemic paralysis include respiratory failure, life-threatening arrhythmias, and death. Immediate correction of hyperkalaemia not only terminates the paralysis but also reduces the potential risks related to it.[13] Both hypokalaemia and hyperkalaemia are common in HIV-infected patients. Hyperkalaemia and hyponatraemia may also be a manifestation of mineralocorticoid deficiency due to adrenal insufficiency or the syndrome of hyporeninemic hypoaldosteronism.[14] A systemic abnormality in potassium equilibrium, which favours the development of hyperkalaemia by a mechanism unrelated to renal potassium excretion, has also been identified in HIV-infected individuals.[15]

In the diagnosis of hyperkalaemia, urine chemistries have a limited role since they are primarily useful for differentiating decreased renal excretion from increased potassium loads. Increased potassium loads, whether endogenous or exogenous, rarely are an occult cause of persistent hyperkalaemia, and so the urinary chemistries nearly always point to inappropriate renal handling of potassium.[6] This case signifies the importance of history and knowledge of potassium contents of various foods consumed by patients in particular clinical scenarios .

REFERENCES

1. Agarwal R, Afzalpurkar R, Fordtran JS. Pathophysiology of potassium absorption and secretion by the human intestine. Gastroenterology 1994; 107:548-71.
2. Cummings JH, Bingham SA, Heaton KW, Eastwood MA. Fecal weight, colon cancer risk, and dietary intake of nonstarch polysaccharides (dietary fiber). Gastroenterology 1992; 103:1783-9.
3. Deitrick JE, Whedon GD, Shorr E. Effects of immobilization upon various metabolic and physiologic functions in normal men. Am J Med 1948; 4:3-36.
4. Squires RD, Huth EJ. Experimental potassium depletion in normal human subjects. I. Relation of ionic intakes to the renal conservation of potassium. J Clin Invest 1959; 38:1134-48.
5. Rabelink TJ, Koomans HA, Hené RJ, Dorhout Mees EJ. Early and late adjustment to potassium loading in humans. Kidney Int 1990; 38:942-7.
6. Malnic G, Klose RM, Giebisch G. Micropuncture study of distal tubular potassium and sodium transport in rat nephron. Am J Physiol 1966; 211:529-47.
7. Schim van der Loeff HJ, Strack van Schijndel RJ, Thijs LG. Cardiac arrest due to oral potassium intake. Intensive Care Med 1988; 15:58-9.
8. Evers S, Engelien A, Karsch V, Hund M. Secondary hyperkalaemic paralysis. J Neurol Neurosurg Psychiatry 1998; 64:249-52.
9. Ptácek LJ, George AL Jr, Griggs RC, et al. Identification of a mutation in the gene causing hyperkalemic periodic paralysis. Cell 1991; 67: 1021-7.
10. Pollen RH, Williams RH. Hyperkalemic neuromyopathy in Addison's disease. N Engl J Med 1960; 263:273-8.
11. Maury E, Lemant J, Dussaule JC, Penicaud Vedrine, Offenstadt G. A reversible paralysis. Lancet 2002; 360:1660.
12. Martinez-Vea A, Bardaji A, Garcia C, Oliver JA. Severe hyperkalemia with minimal electrocardiographic manifestations: a report of seven cases. J Electrocardiol 1999; 32:45-9.
13. Livingstone IR, Cumming WJ. Hyperkalaemic paralysis resembling Guillain-Barre syndrome. Lancet 1979; 2:963-4.
14. Marks JB. Endocrine manifestations of human immunodeficiency virus (HIV) infection. Am J Med Sci 1991; 302:110-7.
15. Kalin MF, Poretsky L, Seres DS, Zumoff. Hyporeninemic hypoaldosteronism associated with acquired immune deficiency syndrome. Am J Med 1987; 82:1035-8.