Truth behind hypokalemia

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Scenario 1: full of doubt in emergency room

On that day, I was on duty in emergency room. At about 11:00 am, I heard someone cried for help in the hall, "Please help my kid..." The sound came nearer. I ran out of the emergency room immediately. It was a girl at the age of 15. She was helped by her family to the room. The nurses had already prepared the bed for the patient.

Within a few minutes, I had got prepared for fighting. The patient from An'hui was traveling in Shanghai. Half day ago, she had sudden nausea and vomiting associated with weakness of both lower limbs likely because she did not get adapted to climate or diet in a new place. She did not have abdominal pain and diarrhea. But she was not able to walk. Her family immediately sent her to the emergency room of our hospital.

Physical examination was immediately performed, revealing T37 ℃, BP 120/80 mmHg, clear consciousness, poor mental state, no cyanosis of mouth and lip, coarse breath sounds of both lungs, no dry and wet rales heard. HR was 90 bpm. Heart rhythm was regular. No pathological murmur was heard. The abdomen was flat and soft without tenderness and rebound tenderness. The liver and spleen were not palpitated under ribs. No swelling was found in both lower limbs. The muscle strength of both lower limbs was at level III. Babinski's sign was negative.

The patient had history of nausea and vomiting. The decrease in muscle strength of both lower limbs made me think about hypokalemia. However, many factors could trigger hypokalemia. Which one was it? Relevant examinations including blood biochemistry and ECG were completed. Soon, the bedside ECG indicated sinus rhythm, HR 70 bpm and evident U wave. It was no doubt to be hypokalemia. Blood biochemistry also demonstrated this. I looked at the test report and found that the level of serum potassium was only 1.9 mmoL/L. It was severe hypokalemia. This made me feel as if treading on thin ice.

Before onset, the patient had nausea and vomiting. But the short-term history would not lower serum potassium too much. Additionally, what was the explanation for abnormal liver function? Was there association between hypokalemia and abnormal liver function? Four hours later, the levels of electrolytes were reviewed and serum potassium level was elevated to 2.4 mmoL/L.

My sixth sense told me it was not a simple case. If hypokalemia was not corrected and underlying factor was not identified, the treatment would be confused, just like flowers in fog and moon in water. The patient would develop arrhythmia and even die at any time. Hence, I immediately contacted the endocrinology department to make diagnosis.

Scenario 2: truth came out in department of endocrinology

In Department of Endocrinology, the patient received adequate supplementation of potassium and was closely monitored for electrolyte level. Still, the serum potassium increased slowly. The blood gas analysis suggested metabolic acidosis with respiratory alkalosis. The standard urine test indicated pH 7.0 in normal range. For the patient with metabolic acidosis, the pH of urine should be lower than 5.3. Apparently, the patient had paradoxical alkaline urine. Was it renal tubular acidosis (RTA)? This finding made me excited.

The current clinical data indicated hyperchloremic metabolic acidosis with normal AG, hypokalemia and paradoxical alkaline urine. This should be type I RTA. Its pathogenetic mechanism involves the following steps: H^+ pump (H^+-ATPase) in renal tubular epithelial cell is exhausted, resulting in disorder of active secretion of H^+; cell permeability of renal tubular epithelial cell is abnormal, leading to passive diffusion of intraluminal H+ into fluid

surrounding the tube.

It is well known that the body eliminates potassium mainly dependent on kidney. The elimination of potassium by kidney includes three parts: filtration via glomerulus; reabsorption of potassium by proximal convoluted tubule and medullary loop; regulation of potassium excretion by distal convoluted tubule and collecting tube. For frequently changed uptake of potassium, the body is mainly dependent on distal convoluted tubule and collecting tube to regulate secretion and reabsorption of potassium and hence to maintain potassium balance.

What caused RTA? In this confusing situation, I asked further about the medical history of the patient, and learned that the patient was often dry in her mouth and drank a lot of water each time she ate steamed bread. This new clue seemed to uncover a new world for me. Was it Sicca syndrome behind these symptoms? I completed examination to test markers of autoimmunity and rheumatism-related immunology. Three days later, the results indicated positive anti-SSA, anti-SSB and anti-nuclear antibody.

Next, I asked to conduct consultation by the rheumatism immunity department. Labial salivary gland biopsy and ECT of salivary gland were performed for the patient. It was considered as Sicca syndrome complicated with RTA and hypokalemia based on pathological examination. The riddle was answered. The case was resolved, which made me relaxed. After positive potassium supplementation and etiological treatment, the patient was recovering and discharged.

Inspiration from practice

(I) For such sicca syndrome complicated with hypokalemia caused by RTA, potassium citrate is best drug to supplement potassium;

(II) National reports indicate 30–50% cases are complicated with renal damage, among which, 35% have involvement of distal renal tubule, causing type I RTA, characterized by periodic hypokalemia paralysis, renal calcification, kidney calculus and nephrogenic diabetes insipidus. Moreover, live damage occurs in about 25% of cases with Sicca syndrome who may or may not have relevant clinical symptoms or different manifestations of liver function impairment. Some patients are complicated with autoimmune liver disease, particularly frequent for primary biliary cirrhosis;

(III) It is tricky to treat this case. In such case, clinician must seek clue step by step and link clues together to detect the truth. Sicca syndrome is a connective tissue disease characterized by high lymphocytes infiltration that can invades exocrine glands such as lacrimal gland and salivary gland. Ammonium chloride stress test identifies about 50% of patients with clinical sub-type RTA prone to hypokalemia. It is not easy to find out the cause of hypokalemia. So clinician should practice their skills to tell the truth.

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Footnote:

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