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CLINICAL QUIZ

Mysterious hyperkalemia and cardiac arrest in a newborn infant undergoing continuous veno-venous hemofiltration dialysis: question

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Case summary

A 16-days-old girl was transferred to our pediatric intensive care unit (PICU) because of neurological and respiratory deterioration. She had been born after an uneventful pregnancy to Druse parents who were first cousins. She was being fed on breast milk. On day 7 she refused feeding, her cry weakened, and she was hospitalized in another hospital. On admission she was afebrile and had generalized hypotonicity, head lag, areflexia, drooling and ptosis. Findings from sepsis workup and liver and kidney function tests were normal. Her level of consciousness continued to deteriorate, she developed extreme weakness, bradypnea and recurrent apnea and was transferred to our PICU. On admission she required intubation and mechanical ventilation because of extreme bradypnea. On the next day, metabolic workup revealed very high urine and blood levels of leucine, isoleucine and valine, which led to the diagnosis of maple syrup urine disease (MSUD).

Continuous veno-venous hemofiltration dialysis (CVVHD) was started at 1824 hours using the Prisma system (Hospal, France) with an M10 filter (total volume 3.4 ml), infant tubing and the following settings: blood flow 20 ml/min; replacement fluid flow 100 ml/h; dialysate flow 100 ml/h and no net removal. A 5 l Hemosol BO kit (Hospal) was used, containing a standard, potassium (K^+)-free electrolyte solution and additional sodium bicarbonate.

At 2200 hours a decrease in serum K^+ to 3.04 mEq/l was noted. Therefore, at 2215 hours, 20 mEq potassium chloride (KCl) were added to the 5 l Hemosol replacement bag (which had been connected into the system only several minutes earlier), in order to create a solution with K^+ concentration of 4 mEq/l.

At 2222 hours blood was sampled and stored for later determination of amino acid levels. At 2226 hours ventricular premature beats (VPBs) were seen on the monitor, changing rapidly to alternating periods of other erratic dysrhythmias, pulseless ventricular tachycardia (VT), ventricular fibrillation (VF), pulseless electrical activity (PEA) and asystole. The patient was immediately disconnected from the ventilator and ventilated with a bag-valve device, and cardiopulmonary resuscitation was started. Patency and correct position of the endotracheal tube were confirmed, and there were no signs of pneumothorax.

Blood sampled through the arterial line at 2233 hours showed partial oxygen pressure pO_2 of 616 torr, partial carbon dioxide pressure (pCO_2) of 10 torr, pH of 7.75 and K^+ of 9.60 mEq/l. An additional blood specimen, drawn at 2239 hours to confirm this extreme K^+ level, showed a K^+ level of 12.6 mEq/l. CVVHD was discontinued immediately, the Prisma filter was rinsed with 500 ml normal saline

The answer to this question can be found at http://dx.doi.org/10.1007/s00467-008-0806-1.

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solution, and a new replacement bag, containing no potassium, was connected. Analysis of blood sampled at 2249 hours showed a K^+ level of 6.81 mEq/l. CVVHD was commenced at 2250 hours.

Concurrently, cardiopulmonary resuscitation (CPR), defibrillation attempts, and administration of epinephrine, lidocaine, sodium bicarbonate and calcium chloride were continued. There were several brief periods of spontaneous circulation, interchanging with periods of pulseless arrhythmias. At 2250 hours the infant had a sinus rhythm lasting approximately 2 min, followed again by VPBs, runs of VT, PEA and asystole. Analysis of blood sampled at 2258 hours revealed a K^+ level of 13.35 mEq/l, the highest level recorded during the entire event. At 2304 hours, following the administration of an additional dose of sodium bicarbonate and one dose of amiodarone, the infant regained sinus rhythm and CPR was discontinued. The patient's K^+ level at this time was 9.96 mEq/l. CVVHD was continued, and potassium levels normalized within the next hour, with no further intervention.

CVVHD was continued for an additional 20 h, until serum levels of leucine, isoleucine and valine had normalized. Subsequently, the infant had an uneventful recovery. Follow-up examination in the metabolic disorders outpatient clinic revealed no neurological or developmental abnormalities.

Questions

1. How should this event be investigated?
2. What were the cause and mechanism of the acute hyperkalemia?
3. How could the second increase in serum K^+—after the filter had been rinsed and a K^+-free replacement bag had been connected—be explained?