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Case Report
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A Case of Successful Rescue of Cardiac Arrest Caused by Diabetic Ketoacidosis Combined with Hypokalemia

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Abstract

A 19-year-old male patient was hospitalized with coma with extremely high blood sugar levels. Low blood potassium, shock, acidosis and cardiac arrest in the course of disease, the rescue is successful but there are limb extremity dysfunctions. The lessons are that we should be alert to those comatose emergency patients with hyperglycemia, which could make them lethal in the short term. So we should need to fight against the time to heal them, not to delay the prime time of treatment for inspection. In addition, the peripheral neuropathy is a rare, possibly associated with multiple factors.

Keywords: Diabetic ketoacidosis; Hypokalemia; Cardiac arrest; Rescue

Case Presentation

A 19 year-old man presented as an acute patient to the emergency department with coma and a 2-days history of oliguria, fatigue, being slow in reaction. The blood sugar test showed: "high". And we noted that the patient was overweight. His parents stated that he had no past medical history of note. On admission, his pulse was 107 times per minute, his blood pressure was 77 over 44 mmHg and his temperature was 37.2°C. And both sides pupils were round and about 0.4mm, direct and indirect light reflex weaken. Bladder region was not filling. Blood glucose on arterial blood gas analysis was 114.9 $mmol.L^{-1}$ (normal range 3.9–6.1 $mmol.L^{-1}$). The patient was found to have metabolic acidosis on arterial blood gas (pH 6.94, lactate 3.6 $mmol.L^{-1}$, bicarbonate 3.2 $mmol.L^{-1}$). Plasma ketone testing was positive. Sodium was 118 $mmol.L^{-1}$, potassium 1.9 $mmol.L^{-1}$ and creatinine 261μmol.L^{-1}. WBC 23.9 × 10^9, N % 85%, CRP 13 $mg.L^{-1}$. Further, the Head CT test showed no special sign. A presumptive diagnosis of diabetic ketoacidosis (DKA) and coma of unknown origin were made.

Immediately, he was transferred to the intensive care unit. 10 Unites insulin were given via subcutaneous injection, followed a continuous 4 $units.h^{-1}$ intravenous, meanwhile, rapid infusion of 0.9% saline through both peripheral vein and femoral vein were given, there were 8375ml fluid infused in 16 hours [1]. Also we started potassium supplement [2] at the time of initial volume expansion, in addition to potassium supplement via IV fluid infusion, potassium chloride boluses were in speed of $1g.h^{-1}$ by intravenous injection pump, combined with nasal feeding in $1g.h^{-1}$ simultaneously. Some bicarbonate was used to correct acidosis [2]. Then about 3 hours later, the patient began ventricular tachycardia, soon cardiac arrest followed and respiratory arrested. Cardiopulmonary resuscitation and endotracheal intubation were immediately carried out. Within 5 minutes, the patient recovered to sinus rhythm. Then ice cap, mannitol (125ml) [3] and ganglioside were used to protect brain cells. At six o'clock in the morning on the second day of admission, the patient got a high fever of 45.5°C, imipenem (1.0g q8h) were given for anti-infection and some blood samples were taken for cultivating. And dopamine boluses were in speed of 15 $\mu g.kg^{-1}.min^{-1}$ by intravenous injection pump. At 12:00, patient turned conscious, but was slow in reaction. On the third day of admission, we stopped the usage of dopamine and the blood pressure could maintain within normal range. On the fifth day of admission, vital signs of the patient restored normal, the tube placed in his airway was removed, and hyperbaric oxygen therapy was begun. On the 10^{th} day of admission, we found he could not lift wrists and feet, nor bend his fingers. On the 15^{th} day of admission, he transferred to general ward. On the 22^{th} day of admission, he went to the other hospital for specific treatments. About 2 months later, through telephone follow-up, we learnt that his disability of lifting wrists and feet had not improved.

Discussion

The prominent clinical manifestations of the patient were extreme hyperglycemia, hypokalemia,

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hypertonic status, severe dehydration, acidosis, hemodynamic instability, acute kidney injury, disturbance of consciousness with the Glasgow coma scale of 3, sofa scale of 8 and APCHE II scale of 34, which is extremely high mortality rate in primary hospitals (no CRRT [4], no ECMO). There was a profound lesson in our ICU in 2014: A female patient with 22 years old, in a coma for two days and night emergency blood sugar check "high" when she was sent to our emergency room, it delayed precious rescue time for checking (such as cranial CT, consultation of department of gynaecology), her breath and heartbeat stopped suddenly when she was sent to the ICU and died eventually.

The lessons are that we should be alert to those comatose emergency patients, paying attention to their blood sugar levels, such as hypertonic conditions, dehydration, and acidosis and electrolyte disorder caused by extremely high blood sugar levels, which could make them lethal in the short term. We should need to fight against the time to heal them, not to delay the prime time of treatment for inspection.

The patient's pedal, prolapse of the wrist, evoked potentials and electromyography (other hospital) suggest “peripheral neuropathy” [5], considering atypical neuropathy “acute focal neuropathy”. It is relatively rare, and is not directly related to long-term hyperglycemia. In addition, cerebral edema, acidosis, hyperosmosis, and low blood potassium caused by cardiac arrest could be the hypotheses that cause nerve damage.

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