SYMPTOMATIC CEREBRAL INFARCTION: A RARE COMPLICATION OF DIABETIC KETOACIDOSIS

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INTRODUCTION: Diabetic ketoacidosis (DKA), which may be mortal, is accompanied by intracerebral complications from 0.3 % to 1 %. Cerebrovascular events account for 10 % of intracerebral complications.

Our aim is to present a case of symptomatic cerebral infarction as a serious complication of DKA and draw attention to the management of DKA treatment.

CASE REPORT: A previously healthy 3.5-year-old male patient was referred to our endocrinology department with DKA for further evaluation and treatment. Before coming to our center, insulin and sodium bicarbonate bolus had been given before adequate hydration.

On examination, he was extremely dehydrated, had dried lips and tongue with a loss of skin turgor, and had a fluid deficit of 10 %. The score in the Glasgow Coma Scale was determined as 7. She was lethargic and tachypneic with Kussmaul's breathing. His respiratory rate was 36 breaths per minute, pulse was 136 per minute and axillary temperature was 36.7°C. The activity of the deep tendon reflexes was normal and the Babinski sign was negative bilaterally. Laboratory investigations showed serious acidosis (pH: 7.04; HCO3: 3.8 mmol/L; pCO2: 11 mm Hg), hyperglycemia

(plasma glucose: 194 mg/dL) and ketonuria, Serum sodium concentration was found to be 139 mmol/L and potassium 3.1 mmol/L. Level of HbA1c was 10.9 % (normal range 4.8%–6.0%).

The patient was monitored in the pediatric intensive care unit. The fluid treatment plan was made for 36 hours. Rehydration with 0.9 % saline was started (10 mL/kg/h for the first 1 h), followed by continuous intravenous infusion of a

5% glucose solution containing 100 mEq/L sodium chloride, 40 mEq/L 7.5% potassium chloride. Insulin infusion was started with 0.05 U/kg/h. Capillary blood glucose was monitored hourly; electrolytes, urea, and blood gases were repeated with an interval of 2 h.

His DKA was resolved about 24 h after the admission. When the findings of the examination were completely normal, subcutaneous insulin therapy was started with an adequate caloric diet for diabetic people.

On the second day, left-sided hemiparesis and increased deep tendon reflexes and positive Babinski reflex on the left side were detected. Magnetic resonance imaging (MRI) and MR-angiography (MRA) of the brain showed right sided fronto-parieto-occipital infarction. After 3 months of follow-up, the neurological findings of the patient who was on physical therapy program were improved.

CONCLUSIONS: Finally, a well rehydration strategy in the first hours of therapy is crucial in reducing the neurological complications. Since it may be mortal, particularly high-risk and severe cases of DKA should be treated with child endocrinology specialists and, if possible, treated under the conditions of the pediatric intensive care unit.

Topic: Diabetes and insulin

References:

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