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DIABETIC KETOACIDOTIC COMA.

Negmatova Gulnoza Shukhratovna
Head of Endocrinology Department.
Samarkand State Medical University
Togaeva Gulnora Siddikovna
Scientific leader: assistant.
Samarkand State Medical University
Alamova Shahzoda Baxodirovna
Student of Samarkand State Medical University

Annotation: *Diabetic ketoacidosis most often occurs in patients with type 1 diabetes; it develops when insulin levels are too low to meet basic metabolic needs. In a minority of patients with type 1 diabetes, DKA becomes the first manifestation of the disease. Insulin deficiency can be absolute (for example, when the next injection of exogenous insulin is missed) or relative (when the usual doses of insulin do not cover the metabolic needs that increase with stress).*

Keywords: *diabetes, insulin deficiency, glycerol, diabetic ketoacidosis.*

In severe stressful situations, DKA can also develop in patients with type 2 diabetes mellitus. Type 2 diabetic ketoacidosis is a variant of type 2 diabetes that sometimes develops in obese patients, most commonly in individuals of African (including African American or Afro-Caribbean) ancestry. Patients with diabetic ketoacidosis (also referred to as Flatbush diabetes) may have significant deterioration in beta-cell function with hyperglycemia, and are therefore more prone to developing DKA in the event of significant hyperglycemia. SGLT-2 inhibitors have been associated with the development of diabetic ketoacidosis in both types 1 and 2 diabetes mellitus. In pregnant women and patients taking SGLT2 inhibitors, DKA may also occur at lower blood glucose levels than other causes of DKA.

In insulin deficiency, the body obtains energy from lipid and amino acid metabolism instead of glucose metabolism. Uncontrolled lipolysis results in elevated serum levels of glycerol and free fatty acids; the level of alanine also increases due to the catabolism of muscle tissue. Glycerol and alanine serve as substrates for hepatic gluconeogenesis, which is stimulated by excess glucagon associated with insulin deficiency. Acute physiological stressors (eg, infections, myocardial infarction) in patients with type 1 diabetes can cause acidosis, mild glucose elevation, dehydration, and severe potassium loss.

Rarely, in about 1% of cases, acute cerebral edema develops (mainly in children and less often in adolescents and adult patients), but this complication is fatal. Diagnosis is established with arterial blood pH < 7.30, anion gap > 12, and the presence of serum

ketone bodies against the background of hyperglycemia. Acidosis is usually corrected with intravenous fluids and insulin; bicarbonate is administered only with severe acidosis (pH < 7), which persists after 1 hour of therapy. Insulin is not given until serum potassium reaches ≥ 3.3 mEq/L (≥ 3.3 mmol/L).

At the same time, glucagon stimulates the conversion of free fatty acids into ketone bodies in mitochondria. Normally, insulin blocks ketogenesis by inhibiting the transport of FFA derivatives into mitochondria, but in the absence of insulin, ketone bodies are formed. The main ones are acetoacetic and beta-hydroxybutyric acids - strong organic acids, which determine metabolic acidosis. From acetoacetic acid, acetone is formed, which accumulates in the serum and is slowly excreted through the lungs.

Hyperglycemia due to insulin deficiency is accompanied by osmotic diuresis, leading to significant losses of water and electrolytes in the urine. Excretion of ketone bodies in the urine causes additional loss of sodium and potassium. Serum sodium levels either decrease due to natriuresis or increase due to the excretion of large volumes of free water. Potassium is also lost in large amounts, sometimes > 300 mEq/24 h (> 300 mmol/24 h). Despite a significant decrease in the total amount of potassium in the body, its serum level initially remains normal or even increases due to the movement of potassium into the extracellular space characteristic of acidosis. Treatment usually results in a further decrease in serum potassium, as insulin stimulates its movement back into the cells. Life-threatening hypokalemia can develop if serum potassium levels are not monitored and replenished.

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