

Clinical case of late diagnosed diabetes in ketoacidotic coma iii in teenager: lessons to be learned

Abstract

Patient Z, 13years old, entered the ICU of the Republican Specialized Scientific-Practical Medical Centre of Endocrinology (RSSPMCE) with preliminary diagnosis: Diabetes mellitus newly diagnosed. Diabetic ketoacidotic coma. Concomitant: Left-sided pneumonia. Anamnesis: according to her mother, during the preceding 1.5 months, girl complained of dry mouth, thirst, frequent urination, and weight loss. With complaints of nausea and vomiting, in a precomatose state of consciousness 15.01.14 the patient was hospitalized to the infectious hospital, where diabetes was diagnosed; insulin therapy, infusion therapy was started according to the recommendations of endocrinologists consultants, but the patient's condition progressively worsened, and at 17.01.2014 patient was transferred to the intensive care unit of RSSPMCE.

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Objectively

The general condition at admission is extremely severe. Consciousness at the level of coma II. Skin was dry, pale in color. Body temperature with elevations up to 38.6°C. The tongue was dry, densely coated with white coating. Cussmaul's breath, BR 36 per minute, the smell of ketones in the exhaled air. Pulse 128 per min. BP 100/60 mm Hg. Diuresis: polyuria. Glycemia at admission was 30.4mmol/L, ketones in urine: +++++. CBC, urine, biochemical analysis of blood had not any special features. The central vein was catheterized. Nasogastric tube and Foley's bladder catheter were placed. Intensive therapy was initiated immediately: infusion therapy started with saline, small doses of regular insulin intravenously, correction of electrolyte abnormalities: potassium within infusion solutions, heparin therapy, anti bacterial therapy using wide spectrum antibiotics. In the dynamics of the phenomenon of increasing respiratory failure and falling hemodynamics, the patient was intubated, pulmonary ventilation was started in the SIMV mode, inotropic support with dopamine, norepinephrine, and dobutamine started. On this background on the 3rd day the patient had cardiac arrest, after resuscitation heart activity was restored in 10 minutes. Consciousness at the level of coma III with periods of psychomotor agitation and tonic-and-clonic convulsions. In the lungs: left-sided upper-lobe large-focal pneumonia, *Pseudomonas Aeruginosae* was found in the sputum. Intensive therapy: infusion therapy (saline, glucose 5% and 10% solution), insulin therapy, potassium solutions, heparin, antibacterial therapy according to sensitivity.

Rehydration is crucial in treatment of diabetic ketoacidosis, according to international guidelines,¹⁻³ it should be started with 0.9% sodium chloride to reduce glycemia below 14mmol/l with further infusion of 5-10% glucose solution (preferably 10%) 10-15-20mL/kg/hour. The question of taking diuresis into account remains discussible. Thus, ISPAD separately notes that when calculating the volume of rehydration solutions, the volume of diuresis should not be taken into account.³ We are convinced - and our practice confirms this - that counting the volume of diuresis is extremely important in calculating the volume of rehydration solutions. And, if necessary, the speed of infusion therapy should be increased.

In the analyzed case, the patient was admitted with extremely severe dehydration, disturbed microcirculation, including violation of renal blood flow, which was reflected in diuresis inadequate to glycemia. Infusion therapy was carried out under strict control of CVP and diuresis, however, CVP was negative during the first two days despite the fact that the volume of administered liquids was 11 liters on the second day, and the daily urine output was 9L. Nevertheless, in terms of body weight, the speed of infusion therapy was 15mL/kg/h for the first 24hours, 11.4mL/kg/h during the following 24hours, followed by a decrease to 7-8mL/kg/hour at the 3rd and 4th days, 6mL/kg/hour at the day 5. With the elimination of hypovolemia, the volume of intravenously administered solutions decreased, on the fourth day the introduction of fluids through the nasogastric tube was started (with normalization of absorption through GI tract), and on the 5th day feeding was started. The decrease in glycemia during the first day was down to 13mmol/L, on day 2 - to 7.8mmol/L. Ketonuria was eliminated by the 2nd day.

Insulin therapy

According to international recommendations,¹⁻³ insulin therapy in DKA is performed in a low dose regime at a rate of 0.1U/kg/h with an increase in the rate of infusion if necessary up to 0.2U/kg/h. Intramuscular or subcutaneous injection of insulin is ineffective due to impaired absorption (microcirculation disturbance). Intramuscular insulin administration can be used when intravenous insulin therapy is not possible. After stabilization of the level of glycemia, normalization of acid-base balance, restoration of consciousness and stabilization of blood pressure, transfer to subcutaneous injections of insulin is recommended (rapid and long acting insulins). In the analyzed case, the need for insulin during the first day was 0.2U/kg/hour during the first 6hours, but as this dose was insufficient, it has been raised to 0.38U/kg/hr during the 2nd day, 0.1U/kg/hour for the first 12hours of the 3rd day, with gradual decrease during the next day to 0.07-0.06-0.04 U/kg/hour (insulin dose is adjusted to the infusions of glucose solutions). Long acting insulin was added on the 6th day of treatment. At the time of discharge, the daily dose of insulin was 13units (Figure 1). It should be noted that at present, our ICU adheres to the tactics of early initiation of long acting insulin administration. In this case,

the earlier introduction of long acting insulin was impossible due to inadequate microcirculation. There is a clear dependence of the need for insulin on concomitant diseases: at the time of entry–pneumonia, on the 16th day- the development of a right-sided, and on the 19-20th day–bilateral acute purulent otitis media.

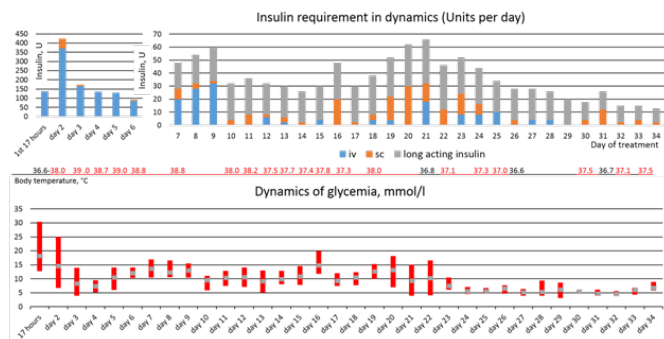


Figure 1 Insulin requirement, body temperature, and glycemia in dynamics of treatment.

Restoration of electrolytes disturbances

Taking into consideration the high risk of rapid development of hypokalemia, infusion of potassium solutions is started simultaneously with the onset of insulin therapy in DKA. If the level of plasma potassium is unknown, infusion of potassium solutions begins no later than 2 hours after the onset of insulin therapy under the control of ECG and diuresis. Potassium solutions were administered according to international recommendations,³ however, in the first 2 days there was a laboratory confirmed hypokalemia: blood serum potassium was 3.0 mmol/L. Sanation bronchoscopy was performed repeatedly starting with the day of transfer to artificial ventilation, then daily until the transfer to independent breathing. The dynamics of the neurostatus is represented in (Figure 2).

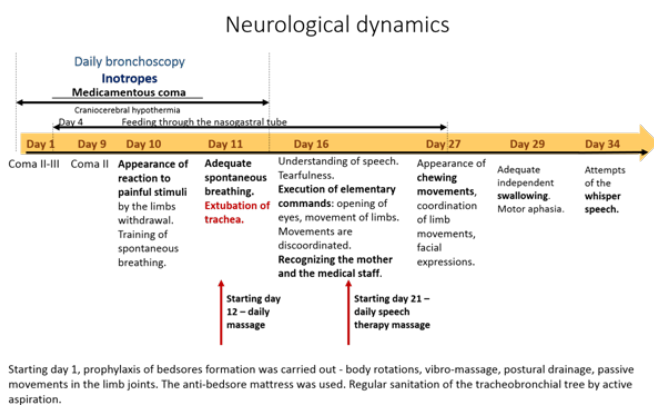


Figure 2 Dynamics of neurological status during the treatment period.

/According to the literature, the causes of death in diabetic ketoacidosis are: cerebral edema (up to 90%), DIC-syndrome (up to 82%), cardiovascular failure (35-39%), acute renal failure (12-13%), intercurrent infection (88%).⁴ The reasons for the development of a critical life-threatening condition in this patient:

- Late admission
- Late Diagnostics
- The presence of severe competing somatic diseases, which in themselves carry an immediate threat to life - left-sided large-focal pneumonia, bilateral acute purulent otitis media.

iv. Excessively high insulin requirements associated with puberty.

The data on the incidence of ketoacidosis in the literature are based on different calculations: according to Ellemann K,⁵ Henriksen O,⁶ DKA in Denmark occurs at a frequency of 12.9-120/100000 of the general population. According to Dedov II et al.² in Russia the prevalence of DKA is 5-20 per 1000 patients with diabetes. The EURODIAB study⁸ conducted in 2006 showed that 8.6% of patients with type 1 diabetes develop DKA. Given the growing prevalence of diabetes, particular attention should be paid to data on the diagnosis of diabetes in a state of ketoacidosis. Thus, according to the data of the Russian Federation,⁷ up to 15.3% of diabetic ketoacidotic comas occur in newly diagnosed diabetes. In the US, this figure is 19.9%.⁸ The EURODIAB study revealed the dependence of DM manifestation in the state of ketoacidosis on the prevalence of diabetes in the country: in countries with low prevalence of the disease, up to 67% of type 1 diabetes was diagnosed in DKA, whereas in countries with high prevalence DM was revealed in DKA only in 26%. This fact shows the alertness of not only local doctors, but also of the entire population. In 2012, 30% of patients admitted to our ICU with DKA were patients with newly diagnosed disease, 10% of them were admitted in comatose state. We found the same tendency also in 2013-2016: 25-33% of hospitalized patients had newly diagnosed diabetes (34-39% of hospitalized children), about 10% of patients with diabetes were in comatose state on admission. The mortality rate for DKA is 4-4.7%,^{3,6} depends on age: 15% among patients over 70 years and 2% among patients younger than 70 years. According to the Russian Federation,^{2,9} the death rate in DKA is 5-15%, exceeding 16% for coma. According to Indian researchers,¹⁰ 2.5 to 9% of DKA is fatal. There are 202-299 patients hospitalized per year with DKA to our ICU. Of these, there were two fatal cases in 2012 of a child N. of 3 years and in 2015 of a child M. aged 8 years within the first 24 hours from the moment of hospitalization. Both children had newly diagnosed diabetes, were hospitalized in coma III, with brain swelling. Child N. had bilateral pneumonia (virological study showed parainfluenza 3 type +++). Autopsy showed severe degeneration of parenchymal organs. Child M had disseminated tuberculosis, myocarditis and multiple organ failure by the moment of admission. So, in both cases, the cause of death was late admission, late treatment and severe concomitant diseases.

To conclude, in order to improve timely diagnostics and adequate therapy, it is necessary to have an urgent emergency determination of glycemia for all patients entering hospitals with complaints of thirst, dry mouth, nausea, vomiting, dyspnea, and/or tachycardia. It is essential (and is already being done) to organize active postgraduate training of all doctors of intensive care and resuscitation departments in tactics of DKA management. Unified standards of DKA management should be and are at the stage of implementation all over the Republic.

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Conflict of Interest

The author declares no conflict of interest.

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