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Thrombophilia's in Patients with Diabetes Mellitus

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Abstract. A clinical case of severe manifestation of type 2 diabetes mellitus in a teenage boy with obesity and the development of severe ketoacidosis as part of multiple organ failure syndrome is described. Timely elimination of diabetic ketoacidosis, hemodialysis and complex therapy in a multidisciplinary hospital made it possible to improve the teenager's condition and avoid death.
Keywords: diabetes mellitus type 2, method, treatment, obesity, manifestation, ketoacidosis.

INTRODUCTION

Diabetes mellitus (DM) is a metabolic disease of various etiologies, which is characterized by chronic hyperglycemia resulting from impaired secretion or action of insulin, or both factors simultaneously [1, 2]. It is the most common of all endocrine diseases, and the number of patients with diabetes is increasing worldwide. In recent years, there has been a significant increase in the prevalence of diabetes mellitus among children and adolescents [2, 3]. Until now, type 1 diabetes, characterized by an acute onset, severe hyperglycemia and ketoacidosis, has been considered a feature of diabetes in children and adolescents [1, 2]. With the development of modern diagnostic methods, other types of diabetes in children and adolescents have begun to be identified: neonatal, type 2 diabetes, MODY diabetes, which is of great importance in the choice of therapeutic tactics and the prevention of complications. Meanwhile, data have emerged on cases of atypical diabetes with features of types 1 and 2 [4, 5]. In the available domestic literature, we have not found a description of this type of diabetes in an obese adolescent.

MATERIALS AND METHODS

A 16-year-old patient was admitted for emergency reasons to the intensive care unit of the regional children's clinical hospital of the Sverdlovsk region (ODKB) on January 25, 2019 in extremely serious condition with manifest diabetes mellitus in ketoacidosis. From January 15 to January 19, 2019, thirst, polyuria, severe weakness, and weight loss appeared against the background of a suspected acute viral infection. They did not seek medical help. The child's condition worsened - from January 21, drowsiness and refusal to eat appeared. And only on January 23, having discovered severe swelling of the child's face, slurred speech and impaired consciousness, the parents called an ambulance. The boy was hospitalized in the intensive care unit (RAO) of the Children's Hospital of N-Tagil at his place of residence, where he was from January 23 to 25 with a diagnosis of newly diagnosed type 1 diabetes mellitus. Severe ketoacidosis.

Insulin therapy was carried out at a rate of 0.1 U/kg body weight per hour using a microjet and infusion of glucose-saline solutions at a rate of 100.0 ml/hour.

RESULTS AND DISCUSSION

The examination revealed signs of severe metabolic and water-electrolyte disorders: hyperglycemia up to 40.0 mmol/l; Blood pH – 6.97; increase in ALT activity to 127 IU; urea level up to 12.4 mmol/l and creatinine from 177.0 to 344.0 μmol/l against the background of an increase in CRP from 6.0 to 66.0 mg/l, leukocytosis (27.7 * 109), neutrophilia.

The child's condition during the therapy is showing negative dynamics. Hemodynamics were stable only with vasopressor support. Blood test: moderate leukocytosis 14.0*109, glycemia 40.0 mmol/l; Blood pH – 7.10; BE – 21.9 mmol/l; ketonuria - 1.5 mmol/l; hypoproteinemia (59.0 g/l); a moderate increase in ALT to 49.0 IU and alkaline phosphatase (143.0 IU) and a significant increase in urea levels (17.1 mmol/l); creatinine (448.0 mmol/l), CRP (120.7 mg/l) and an increase in D-dimer values in the coagulogram to 6.55 mcg/ml (normal - up to 0.5 mg/l). The procalcitonin (PCT) test was also high (6.68 ng/ml). The R-gram of the chest organs shows no pathology. An ultrasound examination of the abdominal organs and retroperitoneal space

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revealed diffuse changes in the liver parenchyma (fatty hepatosis) and pancreas, and free fluid in the abdominal cavity. Doppler ultrasound of renal vessels

On January 25, 2019, a decrease in blood flow velocity indicators and peripheral resistance indicators (PPR) was detected at the level of the parenchyma of both kidneys (possibly the activation of the mechanism of arteriovenous shunting of blood); increased PPS at the level of the hilum on the right.

In treatment: continued infusion of solutions and insulin, inotropic support with adrenaline 0.2 mcg/kg per minute; antibacterial therapy (sultasin, meronem). However, in the dynamics from January 25 to 27, the child's levels of creatinine (up to 510.0 μmol/l) and urea (up to 22.4 mmol/l), CRP (up to 224.2 mg/l), AST (up to 55 IU), α - amylase (up to 510.0 IU) and D-dimers up to 6.91 μg/ml. Consulted several times with a nephrologist - diagnosis: acute kidney injury (prerenal acute renal failure) as part of multiple organ failure syndrome. The need for hemodialysis was substantiated (5 sessions were performed).

At RAO, he was observed by an endocrinologist for the purpose of correcting insulin therapy. He was consulted by a neurologist and diagnosed with severe acute toxic-metabolic encephalopathy.

On the 14th day from the onset of the disease, taking into account the improvement in the child's general condition, the restoration of diuresis, the normalization of CBS indicators, creatinine and urea levels, the patient was transferred to the endocrinology department of the Children's Clinical Hospital in order to clarify the diagnosis and continue therapy.

In the endocrinology department, nosebleeds were repeatedly noted against the background of an increase in A/D to 150/90 mm. Hg Art. and pain in the epigastric region.

Anamnesis of life. Heredity is aggravated by obesity, cardiovascular pathology, and bronchial asthma (in the mother). Child from 2 pregnancies, 2 births. Early toxicosis of pregnancy occurring against the background of obesity. The birth was urgent, spontaneous, body weight 3100 g, length 49 cm, Quetelet index – 63.3%. He was observed by a neurologist for up to a year for residual symptoms of intrauterine hypoxia. Previous diseases - ARVI, chicken pox (2004). Since the age of 5, he has been registered with a pulmonologist and an allergist-immunologist with a diagnosis of bronchial asthma, mild. He received periodic basic inhalation therapy. Since the fall of 2019, episodes of elevated blood pressure up to 140/100 mm have been observed. Hg Art. and nosebleeds. Enalapril was used situationally.

Physical examination and examination data. Height 172 cm, body weight 96 kg, BMI 32.8 kg/m2 (SDS BMI+2.96). The constitution is hypersthenic. On the skin of the chest, abdomen, thighs and popliteal fossae there are pale and bright pink stretch marks, follicular hyperkeratosis in the shoulder area, acanthosis pigmentosa on the neck, axillary and groin areas. The subcutaneous fat layer is developed evenly and excessively, the type of fat deposition is abdominal. The thyroid gland is not enlarged. Blood pressure 140/90 mmHg, heart rate 88 beats/min. The liver is palpated 2.0 cm below the costal margin and the edge of the spleen. Tanner-4, gonad volume d=l=14 ml.

Consulted with an otolaryngologist, diagnosis: recurrent nosebleeds. Consulted with a pulmonologist: based on the medical history, mild bronchial asthma in remission was diagnosed. He was consulted by a cardiologist, and based on Holter monitoring of ECG and blood pressure, a diagnosis was made: Labile arterial hypertension.

After FGDS, he was again consulted by a gastroenterologist - diagnosis: Steatohepatitis, low degree of biochemical activity. Pancreatic steatosis. GERD: erosive esophagitis. Duodeno - gastric reflux.

He was consulted by a hematologist, according to whose prescription the child received injections of Clexane (1.0 ml once a day subcutaneously) until the coagulogram parameters normalized. A study was conducted to determine genetic polymorphisms associated with folate cycle disorders - the following were found: 1. heterozygous state of the AC polymorphism 1298 A>C; 2. homozygous state GG polymorphism 66 A>G methionine synthase reductase; determination of genetic polymorphisms associated with the risk of developing thrombophilia) - the following were found: 1. heterozygous state GA polymorphism G455A of β-fibrinogen, 2. heterozygous state CT polymorphism 807 C>T Phe224Phe alpha-2 integrin, 3. homozygous state 4G4G polymorphism -675 5G >4G plasminogen activator inhibitor.

CONCLUSION

The presented clinical example demonstrates a severe, atypical manifestation of type 2 diabetes mellitus for an obese patient and indicates the possibility of developing type 2 diabetes in adolescents with obesity in ketosis and ketoacidosis.

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