

HYPERGLYCEMIC COMA - CAUSES, SYMPTOMS, DIAGNOSIS AND TREATMENT**Narzilloeva Malika Shuhrat qizi**

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Abstract. *Hyperglycemic coma is a type of diabetic coma in which there is a sharp increase in glucose levels without ketoacidosis. The condition occurs with prolonged dehydration, excess carbohydrate intake, and relative insulin deficiency.*

Keywords: *insulin deficiency, dehydration, excessive glucose intake, precoma period, hallucinations*

ГИПЕРГЛИКЕМИЧЕСКАЯ КОМА - ПРИЧИНЫ, СИМПТОМЫ, ДИАГНОСТИКА И ЛЕЧЕНИЕ

Аннотация. *Гипергликемическая кома - тип диабетической комы, при котором наблюдается резкое повышение уровня глюкозы без кетоацидоза. Состояние возникает при длительном обезвоживании, избыточном потреблении углеводов и относительном дефиците инсулина.*

Ключевые слова: *дефицит инсулина, обезвоживание, избыточное потребление глюкозы, прекоматозный период, галлюцинации.*

Hyperglycemic (diabetic hyperosmolar) coma accounts for 5-10% of all types of diabetic coma and is characterized by high mortality. It is more common in patients over 50 years of age.

The condition requires intensive and emergency medical care, which is carried out in the intensive care unit. Since the prevalence of comatose states in diabetes remains consistently high, the task of endocrinologists is to improve the hypoglycemic therapy regimen and increase patient compliance.

The development of hyperglycemic coma is associated with severe insulin deficiency and its tissue effects. In practical endocrinology, the condition is most often encountered in patients with type 2 diabetes mellitus who have a long history of the disease and severe metabolic disorders. Risk factors include old age and concomitant pathologies of the urinary system. Direct causes of diabetic hyperglycemia:

Dehydration. Pathological fluid loss is most often observed with diarrhea and vomiting, which occur against the background of acute gastroenteritis, food poisoning, acute pancreatitis.

Dehydration develops with extensive burns, massive bleeding, and irrational use of diuretics.

Climate factors. Dehydration occurs with increased sweating in hot climates. People who work in the open sun or in hot workshops and do not monitor the balance of fluid consumed are at risk.

Excessive glucose intake. Hyperglycemic conditions may occur with the simultaneous consumption of large amounts of simple carbohydrates. Coma is possible with intravenous administration of hypertonic glucose solutions.

Relative insulin deficiency. A comatose state is formed when there is a discrepancy between the insulin level and the tissues' need for this hormone. Such situations develop against the background of an exacerbation of a chronic disease, injuries, and surgical operations.

Symptoms of hyperglycemic coma Diabetic coma develops over 1-2 weeks and is characterized by a prolonged precoma period. Initial symptoms include increased thirst, dry mouth, weakness, and drowsiness. The symptoms are similar to the usual signs of diabetes, so patients do not pay due attention to them and do not consult a doctor. Then there is abundant and frequent urination. Due to dehydration, the skin loses elasticity, becomes dry and flabby.

Hyperglycemic precoma is characterized by pronounced neurological symptoms. Patients experience time and space orientation disorders, severe coordination disorders, and true hallucinations. Brain damage is manifested by convulsive syndrome, nystagmus, and impaired perception and reproduction of speech. Local paresis and paralysis occur occasionally.

If the patient does not receive treatment in time, precoma progresses to the stage of hyperglycemic coma. Neurological symptoms include loss of consciousness, generalized seizures, and pathological reflexes. A third of patients develop high fever. On external examination, sharpened facial features, dry skin, lips, and tongue are noted. Shortness of breath, muffled heart sounds, and decreased blood pressure are detected.

To establish a diagnosis, it is necessary to conduct a physical examination, identify typical neurological signs, and clarify the presence of diabetes in the anamnesis. Diagnostics are carried out in parallel with the provision of first medical aid. To confirm hyperglycemic coma, the following research methods are prescribed:

Biochemical blood test. Pathognomonic sign of diabetic hyperosmolar coma is plasma glucose level over 30 mmol/l. Osmolarity index, which is calculated taking into account electrolyte metabolism parameters, is over 330 mosmol/l.

Urinalysis. Biochemical examination shows a high level of glucosuria and the absence of ketone bodies in the urine - an important sign for differential diagnosis with ketoacidotic coma in diabetes mellitus.

ECG. Cardiography demonstrates conduction disturbances, signs of myocardial ischemia and other consequences of electrolyte exchange pathologies. According to indications, a comprehensive examination of the patient is supplemented by ultrasound of the heart.

CT of the brain. Multiple neurological symptoms require exclusion of organic diseases of the brain. In complicated hyperglycemic coma, CT images show signs of cerebral edema.

Differential diagnostics are carried out with other types of diabetic coma: ketoacidotic; lactic acidotic; hypoglycemic.

Treatment of hyperglycemic coma The condition requires emergency care, which is aimed at combating dehydration, eliminating insulin deficiency and restoring water and electrolyte balance. Treatment is carried out according to the basic principles of correction of emergency conditions. When selecting medications, current biochemical blood parameters and their dynamics are taken into account. The main components of hyperosmolar coma therapy:

Infusion therapy. In critical hypernatremia, treatment begins with a hypoosmolar glucose solution; in other cases, a hypotonic or isotonic sodium chloride solution is used. The infusion rate is calculated taking into account the central venous pressure and the volume of diuresis.

Insulin therapy. Hormone replacement therapy is performed with small doses of short-acting insulin. Blood glucose levels are reduced by no more than 5.5 mmol/l per hour to avoid rebound hypoglycemia.

Anticoagulants. For preventive purposes, heparin preparations are prescribed, which stabilize blood clotting indices and prevent thromboembolic complications. After reducing glycemia to 10-12 mmol/l and normalizing the acid-base balance of the blood, patients are transferred to subcutaneous administration of insulin. According to indications, thrombosis prevention is carried out with oral antiplatelet agents. When consciousness and the ability to swallow are fully restored, one can switch to fractional enteral nutrition with a moderate amount of proteins and limited carbohydrates.

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