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# TOPICAL ISSUES OF DIABETIC KETOACIDOTIC COMA

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## ABSTRACT

Diabetes is a serious chronic disease that develops when the pancreas does not produce enough insulin (this is a hormone that regulates blood sugar or glucose) or when the body cannot effectively use the insulin it has produced. Diabetic ketoacidotic coma is a formidable complication of diabetes mellitus, a consequence of absolute or relative insulin insufficiency and a sharp decrease in glucose utilization by body tissues. In many cases, it develops in patients with type 1 diabetes mellitus, which is characterized by a severe labile course. The active cleavage and incorporation of free fatty acids into the metabolism leads to the formation of ketone bodies. Their synthesis increases to 1000 mmol per day, which significantly exceeds the ability of the kidneys to remove ketones in the urine. [5].

**Keywords:** etiology, pathogenesis, laboratory diagnostics, treatment.

## RELEVANCE

Comatose states are frequent and extremely dangerous complications that lead to death. At the same time, it is necessary to be able to provide qualified medical care, on which the prognosis for the patient's life depends. Among diabetic patients, ketoacidotic ones are registered with the highest frequency. The mortality rate is significantly higher and is 10-20% of cases. The proportion of deaths due to high blood glucose is higher in low- and middle-income countries than in high-income countries. The aggravating risk factors are old age, hypoxia, alcohol intake, overweight, obesity, pronounced decrease in kidney function.

Materials and methods of research. In the databases Umedp, Cyberleninka, MSD directory by keywords, a search was conducted among English and Russian-language works published in the period from 2013 to the present time.

Results of the study: Comatose states are a serious complication of diabetes mellitus. The provoking factors that contribute to the development of ketoacidosis are: incorrect calculation of the dose or its uneven distribution during the day; violation of the technique of insulin administration, temporary cessation of insulin therapy, 4 changing the insulin preparation without first determining the sensitivity of the patient to the new drug. These stressful exogenous factors most often cause a comatose state in patients with untimely recognized

diabetes. [1].

The mechanism of development of diabetic ketoacidotic coma is based on absolute insulin deficiency in combination with increased production of counterinsular hormones (catecholamines, glucagon, cortisol). As a result, there is a significant increase in glucose production by the liver and a violation of its utilization by body tissues, an increase in hyperglycemia in the blood and a violation of the osmolarity of the extracellular space. Lack of insulin in combination with a relative excess of counterinsular hormones leads to the release of free fatty acids in the circulation (lipolysis processes) and their unrestrained oxidation in the liver to ketone bodies (acetoacetate, acetone). As a result, hyperketonemia develops, and later metabolic acidosis. As a result of pronounced glucosuria, osmotic diuresis, dehydration, loss of sodium, potassium and other electrolytes develops. [2].

In laboratory diagnostics, the main parameters are: glucose, ketone bodies, electrolytes.

Glucose.

1. Plasma glucose level is usually > 16.7 mmol/L, but can range from almost normal to very high. Very high glucose levels are characteristic of hyperosmolar coma.

2. The degree of hyperglycemia depends on the degree of decrease in the volume of extracellular fluid.A rapid drop in extracellular fluid volume leads to a decrease in renal blood flow and a decrease in glucose excretion.

3. Osmotic diuresis caused by hyperglycemia is accompanied by large losses of fluid and electrolytes, dehydration and hyperosmolarity of plasma. At normal glucose levels, its contribution to plasma osmolality is small. However, with the degree of hyperglycemia that is usually observed in diabetic ketoacidosis, glucose largely determines the increase in plasma osmolality (usually up to 340 mosmol/kg). In hyperosmolar coma, plasma osmolality is much higher (up to 450 mosmol/kg).

Ketone bodies. The total concentration of acetone, beta-hydroxybutyric acid and acetoacetic acid in serum exceeds 3 mmol / l, and sometimes reaches 30 mmol / l (the norm is up to 0.15 mmol / l).

1. The level of acetone (formed by non-enzymatic decarboxylation of acetoacetic acid) in serum is elevated and usually 3-4 times higher than the level of acetoacetic acid. Unlike other ketone bodies, acetone does not play a role in the development of acidosis.

2. The ratio of beta-hydroxybutyric acid and acetoacetic acid in mild diabetic ketoacidosis is 3:1, and in severe diabetic ketoacidosis reaches 15:1.

3. When measuring the level of ketone bodies in serum and urine using test strips, it must be remembered that sodium nitroprusside reacts with acetoacetic acid, does not react with beta-hydroxybutyric acid and reacts weakly with acetone. Therefore, low concentrations of ketone bodies obtained using these test strips do not mean the absence of diabetic ketoacidosis.

4. As diabetic ketoacidosis is eliminated, beta-hydroxybutyric acid is converted into acetoacetic acid. Therefore, the concentration of ketone bodies measured using test strips increases. However, this does not mean that diabetic ketoacidosis is increasing.

Metabolic acidosis is characterized by a concentration of bicarbonate in serum < 15 meq/l and arterial blood pH < 7.35. In severe diabetic ketoacidosis, pH < 7.0. Acidosis is mainly caused by the accumulation of beta-hydroxybutyric and acetoacetic acids in plasma. Insufficient blood supply to tissues causes lactic acidosis. Hyperchloremic acidosis can develop against the

background of infusion therapy and persist for some time after the elimination of diabetic ketoacidosis due to the introduction of excess chloride.

Electrolytes.

1. The concentration of sodium in the serum may be reduced, normal or increased. Hyperglycemia is necessarily accompanied by the transition of water from the intracellular space to the extracellular. This redistribution of water, despite plasma hyperosmolarity and dehydration, may be the cause of apparent hyponatremia. Hypertriglyceridemia also contributes to the apparent decrease in sodium concentration.

2. Serum potassium levels can also be low, normal or high. The potassium level depends both on the release of this cation from the cells due to acidosis, and on the degree of decrease in the volume of extracellular fluid. Therefore, normal or high serum potassium levels do not reflect the existing potassium deficiency (this deficiency is due to osmotic diuresis). An initially low concentration of potassium indicates a significant loss of potassium and requires rapid replenishment.

3. Serum phosphate levels may be normal, but, as in the case of potassium, this does not reflect a real phosphorus deficiency. This deficiency always occurs against the background of increased catabolism, since phosphate passes from the intracellular space to the extracellular and is lost in urine during osmotic diuresis

Other laboratory indicators

1. AMC is usually in the range of 20-30 mg%, which reflects a moderate decrease in the volume of extracellular fluid.

2. Leukocytosis in diabetic ketoacidosis (15,000–20,000 μl-1) is not necessarily caused by infection or inflammation.

3. Serum amylase levels are sometimes elevated. The reason is unknown. Amylase can enter the blood from the pancreas (but this does not indicate pancreatitis) or from the salivary glands.

4. Sometimes the level of AlAT and AsAT increases, but the diagnostic significance of this indicator has not been established.

5. In diabetic ketoacidosis, pseudodisfunction of the thyroid gland is observed. [3].

Treatment of hyperglycemic coma is based on the same principles and includes:

- Infusion therapy,
- Insulin therapy,
- Correction of electrolyte disorders,
- Treatment of conditions leading to hyperglycemia.

When conducting infusion therapy for patients with diabetic coma, it is necessary to determine its composition, volume and rate of infusion of solutions. In order to avoid errors and iatrogenic complications (such as pulmonary edema, cerebral edema, acute circulatory insufficiency) during rehydration treatment, monitoring of the main parameters reflecting the degree of volemia of the body (hourly diuresis, blood concentration indices), as well as osmolarity and electrolyte balance of plasma is mandatory. Isotonic crystal-like solutions should be used as the main component of the infusion program. Since 0.9% sodium chloride solution causes hyperchloremic acidosis, preference should be given to polyionic isotonic solutions with a buffer. Infusion therapy is carried out at a rate of 500 ml / h; its volume in the first 12 hours can reach 5 liters. An important criterion for the adequacy of infusion treatment is hourly diuresis, the

volume of which should not be below 40 ml / h. After lowering the blood glucose level to 14 mmol / l, intensive infusion therapy is discontinued and rehydration is carried out by ingestion of liquid and administration of 150-200 ml / h of 5% glucose solution.

Then insulin therapy is started at the same time. Initially, a bolus is injected intravenously – 10-20 units of simple insulin (0.2-0.3 units / kg) (some authors recommend following the rule when selecting an insulin dose: bolus dose of insulin = glycemic level (mmol / l) / 2). Then a constant intravenous infusion of insulin is carried out at a dose of 0.1 U / h. If after 3-4 hours the glycemia does not decrease by 30%, the dose is doubled; if after another 4-5 hours the glycemia does not decrease by 50%, the dose of insulin is doubled again. On the other hand, it is impractical to reduce blood glucose levels faster than 5-6 mmol / h, and in the first 10 hours – below 14 mmol / l. After reaching a glycemic level below 14 mmol / l, insulin is administered subcutaneously at a dose of 0.05-0.1 U / kg every 3 hours. Treatment with insulin requires adequate correction of hypokalemia, consisting in the addition of 40 mmol of potassium (3 g of potassium chloride) to each liter of transfused solutions. In order to avoid cardiac complications, the infusion rate of potassium-containing solutions should not exceed 20 mmol of potassium per hour. [4].

## CONCLUSIONS

Diabetes is a chronic progressive disease characterized by an increase in blood glucose. With unsatisfactory control of diabetes, health and life-threatening complications arise. Acute complications make a significant contribution to mortality, increase costs and reduce the quality of life of people living with diabetes. Excessively high blood glucose levels can be life-threatening if they cause the development of a diabetic coma. Hyperglycemic coma requires emergency medical care, including rapid correct differential diagnosis and immediate response measures. Otherwise, the threat to life increases dramatically; the mortality associated with hyperglycemic comas is currently estimated at 10-20% and significantly depends on age (among elderly patients, this indicator is much higher).

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