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Education "North Ossetian State Medical Academy" of the Ministry
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(FGBOU VO SOGMA MRussian Health Ministry)

Department of Internal Medicine №2

DIABETES

METHODOLOGICAL MATERIALS

main professional educational program of higher education - specialty program in the specialty31.05.01 General Medicine

Vladikavkaz

Methodological materials are intended for teaching 5th year students (9th semester) of the medical faculty of the Federal State Budgetary Educational Institution of Higher Education SOGMA of the Ministry of Health of the Russian Federation in the discipline "Endocrinology"

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DIABETES SUGARA disease caused by absolute or relative insulin deficiency, which manifests itself only with a combination of endogenous (genetic) and exogenous (external) factors, accompanied by a violation of the metabolism of proteins, fats, carbohydrates and gradual damage to all organs and systems.

Epidemiology.

In 1992, 90 million patients were registered worldwide. The prevalence of the diseasenor is 2.0-4.0%. The number of known patients is approximately 1/3 of the actual number. According to the WHO, a widespread increase in the incidence of diabetes mellitus (DM) is predicted for the next decade. In 10 years, the number of patients is expected to double.

Almost 80% of patients have catype 1 diabetes mellitus, 10-15% suffer from type 2 diabetes mellitus. In 5-10% of cases, diabetes is caused by various diseases (symptomatic). The growth of type 2 diabetes mellitus is noted after 50 years of age. The peaks of type 1 diabetes mellitus are at the age of 3-5 and 11-14 years. In 1991-1994 there was an increase in the incidence of children under 1 year of age. About 60% of all patients are over 50 years old. The ratio of men to women is approximately 1: 2.

Etiology and pathogenesis...

Genetic predisposing factors...

The realization of hereditary predisposition does not obey Mendel's law. Genetic factors for type 1 diabetes and type 2 diabetes are controversial. Type 2 diabetes is characterized by inheritance according to an autosomal dominant trait, there is a significant dependence of the manifestation of diabetes mellitus on the action of environmental factors and age, high concordance (about 100%). Type 1 diabetes is associated with the HLA haplotype; HLA antigens located on the 6th autosomal chromosome B8, B15, DR3, DR4, DW3, DW4 are considered diabetogenic. HLA antigens B7, DW2, DR.2 - antidiabetic "protective". The probability of manifestation is determined by the set of HLA alleles. The B8 antigen determines the predisposition to autoimmune diabetes mellitus and other endocrine autoimmune diseases (Hashimoto's thyroiditis, primary adrenal cortex insufficiency). Carriage of the B15 antigen contributes to the development of virus-induced diabetes mellitus (after mumps, rubella and other viral diseases). The concordance of type 1 diabetes is significantly lower than type 2 diabetes. So, in the presence of a disease in the father, the probability of manifestation is about 6%, and if the mother is sick with diabetes, the probability of the disease in the child is about 2%.

Environmental and biological predisposing factors.

For type 1 LED.

1. Autoimmune diseases, especially endocrine diseases, autoimmune thyroiditis, chronic adrenal cortex insufficiency.
2. The presence of antibodies to mucoid cells of the stomach, to antigens of the thyroid gland, to cells of the adrenal cortex, to cells of the pancreatic islets.
3. Viral infections of a seasonal nature, carriage of the Coxsackie B4 virus, mumps,

measles rubella, infectious mononucleosis, infectious hepatitis and other, mainly viral, infections that cause damage to beta cells and their inflammation - insulitis.

***For type 2 LED**:*

1. Obesity. With excess body weight, insulin sensitivity decreases and the risk of disease increases by 6-10 times.
2. Unbalanced nutrition (high-calorie diet, lack of coarse fiber dietary fiber). Malnutrition leads to obesity and diseases of the gastrointestinal tract, which contributes to the development of diabetes mellitus.
3. Sedentary lifestyle, physical inactivity. Predisposes to diabetes mellitus by decreasing glucose utilization by tissues.
4. Psycho-emotional overload, stressful situations. Promote an increase in the secretion of diabetogenic hormones (catecholamines, glucocorticoids, endorphins).
5. Chronic gastritis, cholecystitis are accompanied by a violation of the secretion of gastrointestinal hormones that regulate the level of insulin and glucose.
6. Ischemic heart disease and arterial hypertension contribute to the development of diabetes mellitus by increasing the content of contrainsular hormones and decreasing insulin sensitivity.
7. The use of drugs. Diuretic drugs, especially thiazide drugs, antihypertensive drugs (clonidine, hemiton) containing adrenaline (novodrin, izadrin), cytostatic drugs, glucocorticoids (methotrexate, mercaptopurine, dexamethasone, prednisolone) have a diabetogenic effect.

Pathogenesis

With a decrease in insulin secretion, a decrease in its activity or a violation of reception by cells of insulin-dependent tissues (muscle, fat, liver), all types of metabolism are disrupted.

Violation of carbohydrate metabolism.

1. The intensity of glucose entering the cell decreases, the blood glucose content increases and glucose uptake pathways that are independent of insulin are activated: a polyol (sorbitol) shunt, during which glucose is reduced to sorbitol and then oxidized to fructose. However, oxidation to fructose is limited by the insulin-dependent enzyme sorbitol dehydrogenase. When the polyol shunt is activated, sorbitol accumulates in the tissues, which contributes to the development of cataracts, neuropathy, and microangiopathy.
2. The glucuronate pathway of carbohydrate metabolism is accompanied by the formation of glycosaminoglycans, which form the basis of arthropathies in diabetes mellitus.
3. Intensive synthesis of glycoproteins contributes to the progression of angiopathies.
4. Glycosylation of proteins is accompanied by an increase in the content of glycosylated hemoglobin.

The listed ways of extrainsulin assimilation of glucose do not provide the main function of carbohydrates - energy. As a result, the paradox of metabolism develops -the blood is saturated with glucose, and the cells are hungry for energy. The pathways of endogenous formation of glucose from glycogen and protein (gluconeogenesis) are activated, however, this glucose is not absorbed by cells due to lack of insulin. Pentose-phosphate shunt and aerobic glycolysis are inhibited, persistent hyperglycemia, energy deficiency and cell hypoxia occur. The concentration of glycosylated hemoglobin, which is not an oxygen carrier, increases, which aggravates hypoxia.

Violation of protein metabolism.

The biosynthesis of energy proteins-ribonucleotides decreases, which leads to a decrease in macroergs in skeletal muscles and myocardium, constituting a component of muscle weakness. The formation of cyclic nucleotides, which mediate intracellular hormonal reactions, decreases. The biosynthesis of transport, ribosomal and messenger RNA in the liver, muscles, kidneys, and adipose tissue is suppressed. Reduces the biosynthesis of DNA, including in the nucleus of cells. Protein breakdown is activated. In general, there is a violation of protein metabolism with a predominance of catabolic processes.

Violation of lipid metabolism.

The breakdown of lipids is activated, and the processes of peroxidation are enhanced, which is accompanied by the accumulation of toxic substrates that damage blood vessels. The synthesis of lipids from food components is impaired. In connection with the inhibition of the Krebs cycle, intermediate metabolic components accumulate in the blood - lipido-acetoacetic and beta-hydroxybutyric acids, which contribute to the development of ketoacidosis.

CLASSIFICATION (WHO, 1999)

A. Clinical classes Diabetes mellitus...

one. Type 1 diabetes mellitus (destruction of beta cells, usually leading to absolute insulin deficiency).

BUT. Autoimmune,

B. Idiopathic.

2. Diabetes mellitus type 2 (from predominant insulin resistance with relative insulin deficiency to predominant secretory defect with or without insulin deficiency).

3. Other specific types of diabetes:

A. Genetic defects in beta cell function.
B. Genetic defects in the action of insulin.
C. Diseases of the exocrine pancreas.
D. Endocrinopathy.
D. Diabetes induced by drugs or chemicals.
E. Infections.
G. Unusual forms of immune-mediated diabetes.
H. Other genetic syndromes sometimes associated with diabetes.

4. Gestational diabetes mellitus.

Type 1 diabetes mellitus begins mainly in childhood and adolescence, is characterized by a progressive course, a tendency to ketoacidosis. Patients need constant insulin treatment.

Other types of diabetes mellitus account for 5-8% of the total number of patients with diabetes mellitus. This includes the serviceteas of diabetes mellitus against the background of chronic pancreatitis or pancreatic tumor; diabetes mellitus in endocrine diseases (Itsenko-Cushing's disease,pheochromocytoma, acromegaly, glucagonoma). Drug-related diabetes occurs in people who take antihypertensive drugs, diuretics, corticosteroids, and cytostatics for a long time.

Development-promoting chemicals diabetes include alloxan, streptozotocin. Insulin receptor abnormalities are observed in lipoatrophic diabetes mellitus. Genetic syndromes, accompanied by diabetes mellitus, are as follows: Alstrem, Cockane, Lawrence-Moon-Biedl, Klinefelter, Prader-Willi, Shereshevsky-Turner syndrome, Friedreich's ataxia, mucoviscidosis. Mixed conditions leading to diabetes mellitus identified in the classification include a combination of various causes (for example: diseases of a hormonal

nature and impaired insulin reception).

Gestational diabetes different from other types of diabetes. This includes women in whom clinical diabetes mellitus or IGT is established duringduring pregnancy.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The diagnosis of clinical diabetes mellitus is based on the following symptoms: general weakness, polydipthis, polyuria, weight loss, itching of the skin, suppuration of traumatic injuries, lesions of the peripheral nervous system. In the presence of these signs, it is necessary to study fasting blood glucose and 2 hours after eating at least 2 times.

The diagnosis is considered reliable if nato glycemiaat repeated studies more than 6.0 mmol / l (glucose oxidase method), and after a meal — 10 mmol / l or more. Additional criteria - the content of glycosylated hemoglobin is higher than 10%.

Diagnosis of impaired glucose tolerance:

1) fasting glycemia less than 6.0 mmol / l, glycemia after meals - no more than 10.0 mmol / l;

2) glycemia after taking 75.0 g of glucose, after 1 hour - 11.0 mmol / l or more, and after 2 hours - 7.8-11.0 mmol / l.

The test should be carried out against the background of a normal motor and food regime. The amount of carbohydrates in the daily diet should be more than 150.0 g. A break from eating before glucose load is at least 10 hours. After taking blood on an empty stomach, the subject drinks 75.0 g within 5 minutesglucose dissolved in 250 ml of water. Repeated blood sampling is done after 1 and 2 hours. With mass research - only after 2 hours. During the test, smoking and eating are not allowed (Table 1).

Tab. one. **Diagnostic values of glucose tolerance test results**

Conditions research	Glucose concentration, mmol / l			
	Whole blood		Plasma	
	Venous	Capillary	Venous	Capillary
Fasting diabetes mellitus	**6,7**	**6.0**	**7.8**	**7.8**
2 hours after glucose load	**10.0**	**11.1**	**11.1**	**12.3**
Impaired glucose tolerance, fasting	**6.0**	**6.0**	**7.8**	**7.8**
2 hours after glucose load	**6.7-10.0**	**7.8-11.0**	**7.8-11.1**	**8.9-12.1**

In case of doubtful results, the test can be repeated according to the described method or while taking prednisolone (glucose prednisolone test). Prednisolone 15 mg is prescribed 2 and 8 hours before glucose loading.

Diagnosis of diabetes mellitus in children.

The clinical stage is diagnosed by typical signs - thirst, polyuria, often with nocturia, weight loss, the smell of acetone from mouth. The laboratory criteria are the same as for adults.

A glucose tolerance test is performed only in the absence of clinical symptoms,

according to individual indications (overweight, diabetes mellitus in relatives, the presence of diabetogenic HLA antigens). The amount of glucose for the sample is determined at the rate of 1.75 g per 1 kg of body weight. The diagnostic criteria for impaired glucose tolerance remain the same as for adults. Determination of antibodies to the islets of Langerhans is the most accurate method of early diagnosis.

Diagnosis of diabetes in pregnant women.

A glucose tolerance test can be performed starting in the first trimester of pregnancy. Repeat research in each trimester. The evaluation criteria do not differ from the usual ones. Violation of carbohydrate tolerance is observed in 15-18% of pregnant women.

Screening for diabetes mellitus.

purpose - diagnostics early stages, prevention of complications, preservation of working capacity.

Methodology:

The first stage is a questionnaire survey and clinical examination, including questions about risk factors and clinical signs of the disease.

The second stage is the study of fasting glucose and clinical examination.

The third stage is the study of a glucose tolerance test.

Fourth - determination of the content of glycosylated hemoglobin, IRI, C-peptide in the blood.

The results of the first stage - questioning - are assessed on a scale of points, which makes it possible to distinguish a group with suspicion of a clinical stage of the disease, with a probability of impaired glucose tolerance (based on a combination of risk factors), and a group that has no grounds for further examination (without risk factors).

Assessment of the second stage of screening is carried out according to the level of fasting glucose and the results of a clinical examination.

On the basis of the first two stages, persons with clinical diabetes mellitus are selected for the appointment of an adequateNoah therapy. With fasting glycemia below 6 mmol / L and the presence of risk factors, proceed to the third stage - the determination of the glucose tolerance test. As a result, a group with impaired tolerance is distinguished, subject to dispensary observation and rehabilitation, as well as a group with dubious results, such as glucose tolerance. Persons with a low (less than 3.5 mmol / l) level of glycemia are especially distinguished, since they have the likelihood of the initial stage of diabetes mellitus with hyperinsulinism. Such persons need to determine the level of IRI, glycosylated hemoglobin, C-peptide.

CLINICAL PICTURE…

In the clinic of diabetes mellitus, the following groups of symptoms are distinguished:

1. Symptoms mainly due to metabolic disorders of proteins, fats and carbohydrates.
2. Symptom complex of damage to the cardiovascular system.
3. Signs characterizing the damage to the nervous system.

Early signs: general weakness, thirst, more often - weight loss with increased appetite, itching of the skin.

Stage of advanced clinical symptoms characterIt is characterized by a symptom complex of damage to all organs and systems.

Skin and subcutaneous tissue - dryness, peeling, maceration, cracks, xanthosis of the palmar surface of the hands and soles. Rubeosis on the cheekbones, chin, overbrow arches. Pigmented spots on the shins (“spotted shins”). Lipoid necrobiosis, furunculosis, eczema,

psoriasis. Hypotrophy of subcutaneous adipose tissue or its pronounced density, especially at the sites of insulin injection. After the introduction of insulin, areas of atrophy of the subcutaneous tissue ("lipoatrophic syndrome") can also be observed. Post-injection hypertrophic syndrome in the form of infiltrates can be due to injections of various drugs.

Musculoskeletal system - Dupu contractureitrena. Osteoarthropathy (cubic foot), deformation of the interphalangeal joints of the fingers and toes, osteoporosis.

Respiratory system - dryness and atrophy of the mucous membrane of the upper respiratory tract. Tendency to bronchitis, pneumonia and tuberculosis.

The cardiovascular system - damage to the heart and blood vessels occupies an essential place in the clinic of diabetes mellitus and determines the prognosis for this disease.

Digestive organs - marked from the side of the oral cavitythere is atrophy of the papillae of the tongue, a tendency to gingivitis, periodontal disease, stomatitis. The defeat of the stomach is characterized by inhibition of acid-forming and enzymatic functions, atrophy of the mucous membrane and glandular apparatus. Changes in the small intestine consist in a decrease in enzymatic and hormone-forming functions. Disorders of the large intestine are characterized by a tendency to atony, decreased motor function. At the same time, with the development of autonomic neuropathy with a violation of the autonomic innervation of the intestines, persistent diarrhea is observed in patients, which is not eliminated by the intake of enzymatic drugs and astringents. Liver damage is characterized by the development of fatty degeneration against the background of depletion of glycogen reserves, impaired lipid and protein metabolism. A certain place in the pathogenesis of liver damage is occupied by a violation of the outflow of bile due to dyskinesia of the biliary tract. The gallbladder is often enlarged, distended, sensitive to palpation. There is a tendency to stagnation of bile, the formation of stones, the development of an inflammatory process in the gallbladder.

Angiopathies refer to late complications of diabetes, since clinical signs of vascular disorders appear several years after the onset of the disease. However, their pathogenetic basis begins with the onset of hyperglycemia, that is, simultaneously with the development of diabetes itself.

A. S. Efimov proposed the following classification of angiopathy:

Microangiopathy: nephroangiopathy, retinopathy, microangiopathy of the lower extremities, generalized (damage to the vessels of the skin, internal organs).

Macroangiopathy: aorta and coronary vessels, cerebral vessels, peripheral vessels, generalized.

Universal angiopathy: combination of micro- and macroangiopathy.

Angiopathies include specific vascular lesions caused by the action of the following factors:

1. Violations of hemorheology (activation of thromboxane, suppression of prostacyclin, aggregation of platelets and erythrocytes, a decrease in plasminogen, an increase in von Willebrand factor).
2. Metabolic disorders (glycosylation of proteins and low-density lipids, activation of lipid peroxidation).
3. Autoimmune processes (suppression of T-suppressors, deficiency of the T3-component, inhibition of the phagocytic reaction).
4. Tissue and circulatory hypoxia.
5. Hyperproduction of growth hormone, catecholamines, corticosteroids.
6. Release of kinins and prostaglandins.

Microangiopathy:

Nephroangiopathy - the most common cause of death in patients with diabetes mellitus under 40 years of age. It is characterized by damage to the capillaries of the glomerulus, leads to nodular or diffuse glomerulosclerosis, and may be accompanied by damage to the arterioles and renal arteries. In the clinical course, 4 stages are distinguished:

1. Asymptomatic - there is no urinary syndrome, blood pressure is not elevated, the diagnostic criterion is acceleration of renal blood flow, microalbuminuria in stressful situations.

2. Prenephrotic - unstable urinary syndrome, but persistent microalbuminuria, blood pressure is not increased.

Diagnostic criteria - increased glomerular filtration, microalbuminuria, discrepancy between glycemia and glycosuria with a predominance of the latter due to increased glomerular filtration.

3. Nephrotic - stable urinary syndrome, persistent albuminuria, increased blood pressure, periodic edema of the subcutaneous tissue.

Diagnostic criteria are albuminuria, microhematuria, cylindruria, decreased renal blood flow and glomerular filtration. Concentration function of the kidneys is not impaired. At this stage, there is a mismatch between glycemia and glycosuria with a predominance of hyperglycemia and inadequate amount of sugar in the urinedue to a decrease in glomerular filtration.

four. Nephrosclerotic - severe urinary syndrome, stable arterial hypertension, persistent edema, oliguria, hypoisostenuria, impaired renal concentration function (chronic renal failure).

Laboratory diagnostic criteria. Azotemia, anemia, proteinuria, a sharp decrease in glomerular f i l t ration. False compensation for diabetes mellitus, Zubroda syndrome: normoglycemia and aglycosuria due to the absence of enzymes that inactivate insulin.

Kidney damage is often accompanied by pyelonephritis. Predisposing factors: decompensation of diabetes mellitus, glomerulosclerosis, bladder atony, decreased resistance to infection. Pyelonephritis often has a latent course.

Retinopathy: *damage to the organs of vision.* The most common causes of blindness are diabetic cataracts and retinopathy. However, the defeat affects all parts of the organs of vision.

Eyelid changes: frequent inflammatory diseases (barley, blepharitis, eczema). Vascular and degenerative changes (microaneurysms, xanthomas). Recurrent barley, blepharitis, conjunctivitis in 4-6% of cases may be the only signs of diabetesor impaired glucose tolerance.

Changes in the conjunctiva: manifested by damage to the microvessels of the limbal region (aneurysmal angiopathy of the limbus) or dystrophic changes (small yellowish-white blotches or large loose amorphous foci).

Changes in the cornea: mainly of a dystrophic nature (keratopathy, corneal degeneration, deposition of pigment on the endothelium).

Changes in the iris: iris rubeosis, edema and swelling of the pigment epithelium, iris subatrophy, sluggish iridocyclitis.

Lens changes: true diabetic cataract is characterized by flocculent subcapsular opacities, symmetry. Found in more than50% of patients with prolonged decompensation.

Changes in the vitreous body: violation of the transparency of the vitreous body as a

result of vascular (hemorrhageeffusion), inflammatory (exudation) or degenerative processes (destruction of the vitreous body), the deposition of cholesterol - "golden rain".

Changes in the retina: based on hemostasis, ischemia, loss of pericytes, regulating vascular tone, aneurysmal dilatation, hemorrhage, doucheradio vessels. First of all, the centralpart of the retina, central vision deteriorates, becomes difficult to read.

Stage classification (WHO):

1 stage: non-proliferative diabetic retinopathy - enlargement and tortuosity of venules, microaneurysms (venulopathy).

2 stage: preproliferative diabetic retinopathy - punctate hemorrhages, exudates, white-yellow clouding foci. There may be a central retinal vein thrombosis.

3 stage: proliferative retinopathy - neovascularization and proliferation of connective tissue in the retina. Retinal detachment, secondary glaucoma.

For diabetic-sclerotic retinopathy, in addition to aleIn the case of simple diabetic retinopathy, there are sclerosis of the retinal arteries, a symptom of "silver wire", streak-like hemorrhages in the retina.

In diabetic hypertensive retinopathy, in addition to the changes inherent in diabetes, there are also changes characteristic of hypertension: narrowing of the arteries, increased reflex on them, Salus symptom, arteriolar hemorrhages.

In diabetic renal retinopathy - pronounced pericapillary edema of the retina, a "star" figure in the macular region, a large number of cotton-wool foci. Retinal angiosclerosis is absent.

With sharp fluctuations in glycemia, a violation of refraction and accommodation occurs. Clinical equivalents are hyperopia or myopia.

Macroangiopathy...

At the heart of macroangiopathy is atherosclerosis, characterized by pronounced activity and rapid progression, circulatory location of plaques, and systemic lesions. Macroangiopathology in diabetes mellitus has some morphological differences from ordinary atherosclerosis - the circulatory arrangement of plaques, systemicity, a tendency to complicated lesions with caseous decay, the formation of aneurysms.

1. Aorta - clinical signs of aortic atherosclerosis - dizziness, increased pulse pressure, pain in the abdomen is possible. Diagnostic criteria — X-ray, angiography.

2. Vessels of the brain. Clinical manifestations - headaches, dizziness, memory impairment, decreased intelligencenatural working capacity. The criterion for additional diagnostics is rheoencephalography.

Macroangiopathy of the lower extremities.

Stage 1: preclinical.

There are no subjective and objective symptoms.

Research methods and diagnostic criteria - thermal imaging, determination of tissue blood flow velocity, histochemical examination of a biopsy specimen of the skin and muscles of the lower leg, thromboelastogram, coagulogram.

A thermal imaging study reveals a mosaic pattern of the thermal imaging image in the foot area: acceleration of tissue blood flow in the lower leg area, increased platelet aggregation and thickening of the basement membranes of the vessels of the microvasculature, deposition of glycoproteins in the vessel walls, irregularity of the caliber of blood vessels -

in the study of biopsy and thromboelastography.

Stage 2: functional (initial clinical manifestations).

Complaints pain in the feet, fatigue when walking. On examination - uneven color of the skin of the feet due to paresis of the capillaries with small hemorrhages, a decrease in skin temperature in the area of the feet. Research methods and diagnostic criteria. In a thermal imaging study - a decrease in the intensity of the pattern in the foot area. The rate of tissue blood flow is slowed down. With histochemistrystudy - along with the narrowing of the lumen and aneurysmal dilatation of the vessels of the microcirculatory bed, there are areas of vascular obliteration caused by the aggregation and adhesion of blood cells and the infiltration of the walls with glycoproteins.

Stage 3: organic (pronounced clinical manifestations).

SubThe objective signs are a feeling of chilliness and weakness in the legs, pain mostly at rest. On physical examination - muscle hypotrophy of the foot and lower leg, cyanosis of the fingers, uneven color of the skin of the lower leg (“spottedshin "), hyperkeratosis. The pulsation of the great vessels is sufficient.

In a thermal imaging study - “foot amputation” syndrome. The rate of tissue blood flow is dramatically slowed down. Increased coagulation properties of blood, hyperaggregation of formed elements.

Histochemical examination indicates a violation of the intravascular, vascular and perivascular link of microcirculation (obliteration, narrowing of the lumen, infiltration by mast cells).

4 Stage: development of trophic ulcers or gangrene.

Clinical features: pain in the lower legs when walking, intermittent claudication syndrome. On examination - marbled colorskin, alopecia in the lower legs, hypothermia of the feet. Weakening and absence of pulsation of the great vessels of the lower extremities. Possible trophic ulcers in the toes, sharply painful with the development of gangrene (dry or wet).

Additional diagnostic criteria: violation of the main blood circulation in rheovasography, calcification of the great vessels in radiography, narrowing lumen - with angiography.

"Diabetic foot" - syndrome caused by MIcroangiopathy, distal neuropathy, osteoarthropathy and osteoporosis.

Clinical manifestations: change in the shape of the foot ("cubic foot"), trophic disorders, edema, pain syndrome. When an infection is attached - gangreon, osteomyelitis.

Clinical forms of heart damage:

1. Myocardiopathy.
2. Coronary artery disease.

Myocardiopathy is based on microangiopathy, autonomic neuropathy, decreased biosynthesis of ribonucleotides (energy substrates of protein metabolism), myocardial hypoxia.

Clinical symptoms. Subjective signs: pain in the apex of the heart, not associated with physical activity, resistant to nitrites, prolonged, aching or stabbing, often nocturnal. Palpitations, interruptions in the region of the heart at rest. When objective examination - tachycardia at rest, extrasystole, deafness of heart sounds, in the later stages - signs of failure of the right ventricle (heaviness in the right hypochondrium, pressure in the epigastrium after eating, the predominance of nocturnal diuresis over daytime, oliguria, peripheral edema). ECG signs of cardiopathy -

sinus tachycardia, sinus arrhythmia, ventricular premature beats, decreased voltage of the main teeth, Macruso.

Coronary artery disease. It occurs equally often in men and women, clinical manifestations develop about 10 years earlier than in people without diabetes. The course is active, progressive, with a tendency to myocardial infarction. In the presence of diabetic autonomic neuropathy, there may be a painless variant of the course of ischemic heart disease with the development of atypical myocardial infarction.

Clinical signs differ in type 1 diabetes and type 2 diabetes. In patients with type 1 diabetes, IHD is often asymptomatic, the first sign may be left ventricular heart failure as an equivalent of focal dystrophy.fia or acute myocardial infarction.

The second variant of the course of ischemic heart disease is atypical pain syndrome in the region of the heart, without characteristic localization and irradiation, but controlled by nitrites.

The third variant of the course of ischemic heart disease is heart rhythm disturbances such as paroxysmal atrial fibrillation or polytopic extrasystole with the development of circulatory failure. Focal asymptomatic myocardial infarction is possible, the diagnosis of which is established by chance, with an ECG study.

In patients with type 2 diabetes, the clinical picture of ischemic heart disease is typical with pain syndrome of an angina pectoris nature. Possiblealso rhythm disturbances and the development of circulatory failure in the left ventricular type. Often there are conduction disturbances of varying degrees, which are established on the ECG. In the case of the development of a complete transverse blockade, the clinical symptoms correspond to the Morgagni-Adams-Stokes syndrome.

Myocar infarctionYes, with diabetes, it is often accompanied by gastric bleeding (equivalent to DIC) or the development of an aneurysm of the left ventricle of the heart.

Neuropapia:

one. Damage to the peripheral nervous system.

2. Damage to the central nervous system.

Clinical forms of lesions of the peripheral nervous system:

1. Radiculopathy. The pathogenesis is based on demyelination of axial cylinders in the posterior roots and columns of the spinal cord. Clinical symptoms - impaired deep muscle sensitivity, disappearance of tendon reflexes, ataxia, instability in the Romberg position. Ostrye pains, irregularity of the pupils (diabetic pseudotabes).

2. *Mononeuropathy.* At the heart of pathogenesis is defeat individual pairs of cranial nerves. Clinical signs are diplopia, ophthalmoplegia, or others, depending on the location of the lesion.

Polyneuropathy - the most common form of damage to the peripheral nervous system, at the heart of the pathogenesis is the defeat of terminal nerve endings due to a violation of the metabolism of B vitamins, the activation of the polyol shunt - the accumulation of sorbitol and cell hydration. Clinical signs - paresthesia, changes in pain sensitivity: first hyperesthesia, then hypoesthesia. Possible trophic disorders with the formation of deep ulcers in the plantar side of the foot.

Amyotrophy - violation of pain sensitivity in the area of the anterior surface of the thigh and the extensors of the feet.

Autonomic neuropathy. Its pathogenesis is based on damage to the autonomic nervous system at the level of symptomatic ganglia. Clinical symptoms: orthostatic hypotension, nocturnal diarrhea, bladder atony, biliary dyskinesia, stomach atony, hyperhidrosis.

Damage to the central nervous system.

Encephalopathy. The pathogenesis of acute encephalopathy is based on cerebral hypoxia caused by a hypoglycemic state. Clinical signs - soporous condition, epileptiform

convulsive syndrome, inappropriate behavior.

Chronic encephalopathy is based on long-term decompensation of diabetes mellitus, alternation of ketoacidosis and hypoglycemia. Clinical manifestations - irritability, conflict, deterioration in the ability to work associated with intellectual stress, a decrease in criticism.

The defeat of the ENT organs. The pathogenesis is based on microangiopathy, autonomic neuropathy, impaired glucose and protein metabolism. Clinic - the mucous membrane of the nose and pharynx is dry, hyperemic, with a smoothed relief due to desquamation of the epithelium against the background of localacidotic shift. The mucous membrane of the epiglottis, vocal cords is hyperemic, with a crimson tint ("crimson larynx"). Palatine tonsils - hyperemic, often atrophic, in the lacunae - serous-purulent exudate, "false tonsillitis". In smears from the contents of the lacunae - degenerative forms of leukocytes, deflated epithelium.

Outer ear - itching, sulfur plugs, lipid deposits in the fibrous layer of the tympanic membrane or atrophy of its central part.

Middle ear - protracted, oligosymptomatic otitis media with a tendency to perforation of the tympanic membrane.

Inner ear - vestibular disorders, hearing loss.

Dysfunction of the endocrine glands in patients with diabetes mellitus due to various reasons: with type 1 diabetes, there may be thyrotoxicosis due to diffuse toxic goiter. Some patients develop autoimmune thyroiditis, chronic adrenal cortex insufficiency.

Sex glands - in men with prolonged decompensationcation, there is a tendency to impotence due to impaired androgen biosynthesis, microangiopathy of testicular vessels, and autonomic neuropathy. In women, secondary amenorrhea occurs against the background of diabetes decompensation. Dysfunction of the sex glands can be transient. Diabetes compensation promotes sexual recovery.

Features of type 1 LED and type 2 LED.

The basis for distinguishing between the main subclasses of diabetes mellitus is the dependence of the life of a diabetic patient on insulin or the absence of such dependence (Table 2).

Table 2. **Clinical features of type 1 diabetes and type 2 diabetes.**

Clinical signs	**Type 1 SD**	**SD type 2**
one	**2**	**3**
Prevalence among patients with diabetes	**10 - 15%**	**70 - 80%**
Age at onset	**More often up to 25 years**	**Usually over 35**
Onset of the disease	**Acute with typical symptoms**	**Slow, gradual, no clear signs**
Body mass	**Decreased, normal**	**Increased by 80%**
Clinical symptoms	**Clearly expressed**	**Erased, sometimes asymptomatic**
Flow	**Labile**	**Relatively stable**
Tendency to acidosis	**Expressed**	**Usually absent**

Vascular complications	**Pronounced, predominant lesions of small vessels**	**Pronounced, but predominant lesions of large vessels**
The level of ketone bodies in the blood	**Often elevated**	**Usually within normal limits**
Analysis of urine	**The presence of glucose and often acetone**	**Usually the presence of glucose**
Seasonality of the onset of the disease	**Often autumn-winter**	**Absent**
Plasma insulin and C-peptide	**Insulinopenia and decreased C-peptide**	**Normal or hyperinsulinemia**
Pancreas condition	**A decrease in the number of beta cells, their degranulation, a decrease or absence of insulin in them, the islet consists of A-, D- and PP-cells**	**The number of islets and the percentage of B-, A-, D- and PP-cells within the age norm**
Lymphocytes and other inflammatory cells in the islet	**Present in the first weeks of illness**	**Usually absent**
Islet antibodies of the pancreas	**Detected in the first weeks of the disease**	**Usually absent**
Genetic markers	**Combination with HLA-B8, B15, DR3, DR4, DRW3, DRW4.**	**The genes of the system do not differ from the healthy population**
Concordance in monozygotic twins	**Less than 50%**	**More than 90%**
The incidence of diabetes in 1st degree relatives	**Less than 10%**	**More than 20%**
Treatment	**Diet, insulin**	**Diet, oral hypoglycemic drugs (less often insulin)**
Late complications	**Predominantly microangiopathy**	**Predominantly macroangiopathies**

Treatment.

Treatment methods:

1. Diet based on the principle of physiology in the composition of proteins, fats and carbohydrates with the exclusion of refined, easily digestible carbohydrates.
2. Adequate exercise
3. Sugar reducing therapy:
   but) insulin,
   b) tableted sugar reducing agents,
   in) a combination of insulin and tablet preparatov.

Treatment goals:

but) normalization of body weight,
b) achieving clinical and metabolic compensation,

in) prevention of complications,

d) preservation of the ability to work.

Diet therapy- This is a decisive condition for the successful treatment of diabetes mellitus, regardless of the etiology, pathogenesis and nature of the course of the disease. The mainThe dietary requirements for diabetic patients are as follows:

but) full physiological composition of the main food ingredients (proteins, fats and carbohydrates);

b) calculation of the daily energy value of food on the physiological body weight of the patient, taking into account physical activity;

in) exclusion of refined, easily digestible carbohydrates;

d) limiting foods containing high coli the amount of animal fats, and an increase in the content in radiet of vegetable fats and lipotropic products;

e) a sufficient content of vitamins in the daily diet;

e) daily use of coarse fiber foods;

g) a certain meal time (diet) in accordance with with the effect of the deployment of sugar-reducing drugs.

The energy value of the diet of diabetic patients is based on the following calculation: carbohydrates - 50-60%, fats - 15-20%, protein - 20-25%, which is significant does not differ from a physiological diet, thereby meeting the first requirement, that is, a complete composition of food ingredients.

As an example, we give the composition of a diet of 2,400 kcal (10,000 kJ). With normal body weight (Broca's formula: height in cm -100) the total energy value of food should be:

at rest - 20 kcal / kg; with light physical activity - 30 kcal / kg; during physical work of moderate severity - 40 kcal / kg; with hard physical labor - 45 kcal / kg.

Based on the specified need, the individual value of the diet can be calculated. For example: a patient with a height of 180 cm and a weight of 81 kg (normal body weight 80 kg), not engaged in physical work, should be assigned 2400 kcal (10,000 kJ) (at the rate of 30 kcal - 125 kJ X 80). With a moderate load - 3200 kcal (13 360 kJ) (based on 40 kcal - 167 kJ X 80), etc. If the patient's body weight does not correspond to physiological values, diet correction is necessary. For patients with obesity II-III century. it is recommended to reduce the calorie content, calculated for the ideal weight, by 20-30%. In patients with underweight, it is necessary to increase the daily caloric intake by 10-15%.

The condition for the effectiveness of diet therapy in diabetes mellitus is the exclusion of easily digestible refined carbohydrates. Sugar substitutes (sweeteners) that have a sweet taste, but do not require insulin to be absorbed, can be recommended for patients with diabetes mellitus instead of sugar. These carbohydrates include the long-known sorbitol and xylitol, which, in addition to the taste effect, have a cholekinetic, laxative effect.

Sorbitol and xylitol should be prescribed to patients taking into account their energy value: 1 g of sorbitol = 3.4 kcal (15 kJ), 1 g of xylitol = 4.0 kcal (17 kJ). The disadvantage of these substances is that they are metabolized through a sorbitol (polyol) shunt. This pathway of carbohydrate metabolism is limited by the activity of sorbitol dehydrogenase, which promotes the formation of fructose from sorbitol. With a deficiency of sorbitol dehydrogenase, sorbitol is not oxidizedinto fructose, but accumulates in tissues and, being a hydrophilic substrate, leads to tissue swelling and the progression of neuropathies, microangiopathies and the development of cataracts. Therefore, the use of these sugar substituteslimited to 15 g / day. Certain advantages have new sugar substitutes - aspartame, zukli, nutrisvit, ozomaltose, which are devoid of the listed disadvantages and do not have

strict restrictions on daily use.

In the diet of patients with diabetes mellitus, there should be a sufficient amount of vegetable oils that contain polyunsaturated fatty acids and have hypocholesterolemic effect, limiting the processes of lipid peroxidation. It is recommended to provide 50% of the body's need for fats from vegetable fats. Cottage cheese, oatmeal, soy, and sea fish also have a lipotropic effect. These foods should be included in the diet of people with diabetes.

In the daily diet of a patient with diabetes mellitusbut contain 20 to 25% protein. On average, this amounts to 1-1.5 g of protein per 1 kg of body weight. Proteins can be divided into 2 groups: "complete", that is, containing essential amino acids (these are animal proteins), and "defective", that is, not containing essential amino acids (these are plant proteins). It is recommended that the daily protein requirement be provided with foods containing animal and vegetable proteins in a 2: 1 ratio. It should be remembered that 50% of the protein ingested into the body is metabolized into carbohydrates, in connection with which it is advisable to recommend a dinner with a sufficient protein content for patients prone to nocturnal hypoglycemic reactions.

50-60% of the caloric content of the daily diet are carbohydrates. The body's need for carbohydrates is best met by including in the diet products containing slowly absorbed carbohydrates (black bread, buckwheat, pearl barley, oatmeal, vegetables). It is advisable to recommend to patients with diabetes mellitus products containing plantex or coarse-fiber carbohydrates (fiber, pectin, hemicellulose). They contribute to the normalization of the intestinal flora, increase the sensitivity of peripheral tissues to insulin, slow down the development of post-alimentary hyperglycemia, stimulate the synthesis andinsulin secretion, adsorb and remove easily digestible carbohydrates and bile acids, increase the synthesis of B vitamins.

Thus, it becomes obvious the need to include coarse fiber in the diet of diabetic patients (on average 20 g / day). A large amount of plan-tex is found in wheat and rye bran, wholemeal bread, non-peeled cereals, vegetables, berries, and nuts.

Of great importance for patients with diabetes mellitus has a diet. The distribution of the energy value of food during the day is approximately as follows: first breakfast - 30%, second - 10%, lunch - 30% afternoon tea -10 /, dinner - 20%. For patients receiving insulin replacement therapy, meal times should strictly correspond to the time of maximum insulin action. Therefore, 6 meals a day are recommended for them. Particular attention should be paid to the distribution of carbohydrate-rich foods that the patient should receive during periods of maximum drug action. You should also take into account the number of bread units at each meal (Table 3).

Tab. 3... **Replacement of grain units.**
1 grain unit (1 XE) contains

Milk, kefir, cream of any fat content	**1 cup (250 ml)**
Sweet curd	**100 g**
Cheesecakes	**1 medium (85 g)**
Ice cream	**65 g**
Bread, rolls, any, except for butter	**1 piece (25 g)**
Any raw groats **boiled**	**1 tbsp. heaping spoon (15-20 g)** **"Art. heaping spoons (50 g)**
Vermicelli, noodles, horns	**1.5 tbsp. spoons (15 g)**
Any flour	**1 tbsp. heaping spoon (15 g)**

Sugar sand **lump**	**1 tbsp. the spoon** **2.5 pieces (12 g)**
Breadcrumbs	**! Art. spoon (15 g)**
Starch	**1 tbsp. spoon (15 g)**
Rusks	**20 g**
Crackers	**3 large (20 g)**
Raw puff pastry **yeast**	**35 g** **25 g**
Pancakes	**1 large**
Pancakes	**1 medium**
Dumplings	**4 pieces**
Meat pie	**Less than ½ piece**
Mashed potatoes **fried** **dry**	**1 tbsp. slide spoon** **1.5-2 tbsp. spoons (40 g)** **25 g**
Cutlet	**1 medium**
Corn	**0.5 coarse (160 g)**
Apricot	**3 medium**
A pineapple	**1 slice (90 g)**
Orange	**1 medium coarse (170 g)**
Watermelon	**400 g with peel**
Banana	**0.5 coarse (90 g with peel)**
Cherry	**15 large (100 g)**
Pear	**1 small (90 g)**
Garnet	**1 large (200 g)**
Strawberries, blackberries, blueberries, raspberries, currants, gooseberries, lingonberries	**1 bowl (140-160g)**
Strawberry	**10 medium (160 g)**
Tangerines	**3 small (170 g)**
Peach	**1 medium (120 g)**
Blue plums **red**	**3-4 medium (100 g)** **2-3 medium (80 g)**
Apple	**1 medium (100 g)**
Orange juice	**0.5 cups (100 g)**

Patients with type 2 diabetes are also shown fractional meals (at least 4 times a day, with the exception of meals before sleep). Patients with type 2 diabetes are usually overweight. For such patients, the following ratio of proteins, fats and carbohydrates is recommended: 25%, 25%, 50%, but fats should be represented mainly by the vegetable group. The daily calorie content of the diet is limited to 1400-1500 kcal. Overweight patients are useful 1-2 times a week fasting days with food calories from 500 to 800 kcal - on such days, patients receive 50% of sugar-reducing drugs. New in the dietary management of overweight patients are food supplements that reduce the absorption of carbohydrates. As such additives, preparations containing methylcellulose or gelatin, for example Reduvak (Poland), can be used. They have a minimum calorie content and cause a feeling of fullness.

Physical exercise...

Physiotherapy (exercise therapy) is a mandatory component of the computer lexical therapy of diabetes. The positive effect of physical activity during exercise therapy is based on the fact that muscle work is carried out through the use of the energy of fats and carbohydrates, and at the same time the absorption of glucose by the muscles increases. Thus, exercise can be compared to an extra dose of insulin.

Dosed physical therapy is prescribed depending on age, fitness, cardiovascular system and the degree of diabetes compensation. In this case, it is advisable to divide patients into 2 groups:

1. Patients of young and middle age without concomitant pathology.

2. Middle-aged and elderly patients with concomitant diseases and severe angiopathies.However, it should be remembered that this division is very arbitrary, since it is based on the division of the general contingent of patients. When prescribing exercise therapy for each individual patient, it is necessary to individualize classes and make the required amendments to the general methodology.

The technique of physiotherapy exercises for patients of young and middle age without concomitant pathology on the background of diabetes compensation.

It provides for the impact of a significant overall load on the entire body, the value of which depends on the individual properties of the patient, in particular on the functional state of the cardiovascular system, the degree of fitness and the body's response to physical activity. The procedure includes exercises for the upper and lower extremities and trunk; a large place is occupied by exercises for groups of medium and large muscles. All exercises are performed rhythmically at a slow and medium pace with a significant number of repetitions (30 or more). In exercises for small muscle groups, a fast pace is acceptable. In the second half of the course of treatment, it is possible to accelerate and alternate the pace of exercises.Exercises are done with maximum amplitude. The accuracy of their implementation should be strictly monitored.

Usually, at the beginning of the course, easy-to-usedynamic relation of movement, which become more complicated gradually, as the motor skills of patients increase. An obligatory component of the procedure is exercises with pronounced effort and muscle tension, as well as games, both mobile and sedentary. Exercises with weighted objects (dumbbells, medicine balls), on apparatus (gymnastic wall, gymnastic bench), throwing and resistance exercises are widely used. Classes are conducted in a group method. Duration of classes at the beginning of the course of treatment is 15-20 minutes, at the end - 25-30 minutes.

Therapeutic gymnastics technique for diabetes mellitus with severe angiopathies and concomitant diseases of the cardiovascular system.

First of all, it is envisaged to use such a load on the body, the value of which is adequate to the state of the cardiovascular system and is determined by the reaction patient for exercise. Exercises for the main muscle groups are used, which are performed calmly, at a slow pace.

It should be remembered that with severe diabetic angiopathy, a careful regimen must be observed.Cheb physical education, because in this case, the tolerance to physical activity decreases.

Patients in a state of compensation and subcompensation of diabetes mellitus without severe complications are prescribed, in addition to therapeutic, hygienic gymnastics, walking 5-6 km a day, playing sports (tennis, badminton, volleyball).

In addition to physiotherapy exercises, hygienic gymnastics and sports, patients with diabetes mellitus must systematically perform feasible physical work.

Drug treatment:

but) insulin,
b) tableted sugar-lowering drugs,
in) phytotherapy.

Insulin therapy. According to the duration of the hypoglycemic effect, the following groups of insulin are distinguished:

one. Short-acting insulin that is injected subcutaneously, intramuscularly or intravenously and acts on average 6-8 hours.

2. Insulin of medium duration - acts on average 14-18 hours and is administered subcutaneously or intramuscularly.

3. Long-acting insulin (20-24-30 hours), which is advisable to prescribe with persistent night and morning hyperglycemia, since the maximum effect is achieved after 12-16 hours.

4. Mixed-acting insulins are preparations containing 30% intermediate-acting insulin and 70% long-acting insulin.

It is advisable to start insulin therapy with the introduction short-acting drugs. After achieving compensation and selection of an individual dose, the patient can be switched to short-acting and prolonged-acting drugs.

The indications for the administration of insulin are as follows:

1. Type 1 diabetes mellitus.
2. Diabetes mellitus type 2 with ineffective diettherapy and oral hypoglycemic drugs, as well as under the following conditions: ketoacidosis, precomatose states, progressive weight loss.
3. Pregnancy, lactation for any type of diabetes mellitus.
4. Severe polyneuropathy with pain syndrome.
5. Angiopathy with the development of trophic ulcers or gangrene.
6. Infectious and other acute diseases.
7. Surgical interventions, postoperative period.
8. Damage to the liver and kidneys with impairment of their functions.

Insulin therapy techniques.

1. Intensive therapy regimen - multiple (6- 8 times) short- acting insulin injections for newly diagnosed diabetes mellitus (dose selection), during pregnancy, with ketoacidosis.
2. Intravenous administration of short-acting insulin in coma.
3. Basis-bolus insulin therapy as a method of daily treatment.

Dose calculation. When diagnosed for the first time, the daily need for insulin is determined based on the calculation of 0.5 U per kg of actual body weight. The selection of the daily requirement is carried out in the intensive care regimen.

With the most physiological basis-bolus regimen treatment distribution of the dose of basic insulin (medium duration) and insulin for additional injections before meals (short-acting) is as follows. The patient is recommended a dose of 42 units of insulin per day. One third (14 units) will be long-acting insulin. The rest of the dose - 28 IU is distributed as follows: 10 IU - before breakfast, 10-12 IU - before lunch and 6-8 IU - before dinner.

Long-acting insulin should be administered in the evening at the same time as the short-

acting insulin injection. (medium duration drugs) or in the morning (long acting drugs).

The use of the apparatus "Artificial pancreas" ("Biostator") in clinical practice made it possible to accurately calculate the human body's need for insulin. On average, to maintain glucose homeostasis, a person needs from 20 to 40 IU of insulin per day, therefore, when prescribing insulin therapy for the first time, it is advisable to focus on these doses as optimal in the initial period. In the future, the correction is carried out according to the glycemic profile. The recommended fluctuations in glycemia during the day are from 4.0 to 9.5 mmol / l.

Generation of insulin preparations

/ generation - preparations of pork and beef insulin in arbitrary proportions with a content of non-insulin impurities up to 20%.

II generation - preparations with an impurity content of up to 0.5% (mono-peak).

III generation - completely purified from proinsulin, glucagon, C-peptide, somatostatin, pancreatic polypeptide (monocomponent).

IV generation - human insulin, obtained geneticallyin an engineering way.

When controlling the quality of a substance to obtain insulin and finished dosage forms of insulin, the following analysis methods are used:

1. EPAG - polyacrylamide gel electrophoresis to control the content of proinsulin and glucagon.
2. HPLC - high performance liquid chromatography - for identification of species, quantitative content of desamidoinsulin.
3. RIA - radioimmunoassay to determine the minimum amount of impurities: proinsulin, glucagon, somatostatin, pancreatic polypeptide.
4. Limulus-Amebocyt-Lysate test - for the presence and concentration of bacterial endotoxins.
5. All samples are compared with WHO standards. Highly purified insulins are WHO standards. There are WHO reference standards for determining the content of proinsulin and glucagon.

In the manufacture of prolonged-release drugs, stanthe shape and size of crystals and their number are given. Crystals should be of a rhomboendric structure, of the same shape, 10–40 μm in size.

Thus, before going into production, in addition to biological and physicochemical standardization, an assessment of the quality of insulin is used according to 5 parameters (EPAG, HPLC, RIA, Lizat-test, comparison with WHO standards).

The duration of action is confirmed by researchtests on rabbits using a blood glucose test.

Contact container should be made of neutral tubular glass - the first hydrolytic group, and the cork - from chlorobutyl elastomer. The neutral glass bottle guarantees thermal and hydrolytic stability, and the cork guarantees harmlessness (no pyrogenicity, no hemolytic action).

Complications of insulin therapy...

Hypoglycemia. Clinical manifestations of hypoglycemia are caused by dysfunctions of the central and autonomic nervous systems. In the development of hypoglycemic coma, the following stages can be distinguished:

Ist. - cortical - characterized by a feeling of sharp hunger, irritability, aggression;

II Art. - violation of subcortical structures with involvement of the hypothalamus in the process - characterized by pronounced autonomic reactions, that is, sweating, palpitations, trembling hands, headache, facial flushing, indomitable hunger, but

consciousness at this stage is not disturbed;

IIIArt - violation of subcortical structures with a change in consciousness, during this process either depression or aggressiveness develops, patients can commit unmotivated actions, sometimes delirium, hallucinations join;

IVst. - damage to the upper parts of the medulla oblongata - characterized by muscle hypertonicity with the development of convulsive syndrome and loss of consciousness;

Vst.- lesion of the lower parts of the medulla oblongata - characterized by deep coma, hypotension, thathicardia, the respiratory and vasomotor centers can be involved in the process, and then death occurs.

Hypoglycemic coma develops very quickly, and all stages of the process can pass in a few minutes. ResSome decrease in the level of glycemia is accompanied by the secretion of glucagon and catecholamines, which contributes to the breakdown of glycogen and a compensatory increase in the level of glycemia (post-hypoglycemic hyperglycemia). This feature can cause difficulties in determining the nature of a coma.

Treatment for hypoglycemic syndrome depends on stages of the process. So, in I and II Art. it is enough to take carbohydrate foods (honey, sugar, jam). If the patient is unconscious or inadequate during communication, an intravenous jet injection of 40% glucose solution at a dose of 60-80 ml is necessary. If the patient has not regained consciousness, it is advisable to prescribe drip intravenous administration of 5-10% glucose solution with small doses (4-6 U) of insulin (insulin is added to improve the utilization of glucose by the cells of the central nervous system), cocarboxylase, ascorbic acid. For the purpose of brain dehydration, it is advisable to prescribe osmotic diuretics.

In the absence of effect, glucocorticoids are indicated. (100 mg of hydrocortisone in 5-10% glucose solution) and adrenaline (1.0 ml of 0.1% solution), which helps to mobilize liver glycogen.

In recent years, to combat hypoglycemia, the hormone glucagon has been used, which is injected intramuscularly (2% - 1.0-2.0 ml) and has a rapid hyperglycemic effect.

All patients receiving insulin should bedomains about the signs of hypoglycemic conditions, know their causes, measures to prevent and provide self-help.

Insulin resistance. This is a condition characterized by an increase in the dose of insulin as a result of a weakening of its sugar-lowering effect in response to the necessary physiological needs of the body.

Distinguish between mild insulin resistance (insulin dose - 80-120 U / day), medium (insulin dose - up to 200 U / day) and severe - the need for insulin is more than 200 U / day. Insulin resistance can be relative or absolute. Relative insulin resistance is understood as an increase in insulin requirements associated with inadequate insulin therapy and diet. As a rule, the dose of insulin does not exceed 100 U / day. Absolute insulin resistance is due to the hyperproduction of antibodies to insulin, the absence or decrease in the sensitivity of the receptors of cells of insulin-dependent tissues to the action of insulin. Liver diseases, allergic reactions, intoxication, overweight predispose to the development of insulin resistancebody, the use of insufficiently purified insulin preparations.

One of the conditions for the prevention of insulin resistance is the elimination of food allergens, compliance diet and physical activity. Exercise is known to increase insulin sensitivity. For the treatment of insulin resistance, it is necessary to transfer the patient to an intensive insulin therapy regimen with short-acting monocomponent drugs or human insulin. For this purpose, you can use insulin dispensers or a stationary-type "Artificial

pancreas" ("Biostator") apparatus with intravenous administration of insulin in the mode of intensive insulin therapy. Hemisorption, peritoneal dialysis, the introduction of small doses of glucocorticoids together with insulin are also used to eliminate insulin resistance. There is evidence of the effect of the appointment of immunomodulators.

It should be remembered that the treatment of insulin resistance is a long process that requires perseverance and patience to achieve the desired effect and rid the patient of large non-physiological doses of insulin.

Lipodystrophy. Lipodystrophy is a relatively common complication of insulin therapy. Are manifestedlipodystrophy by atrophy of subcutaneous fat at the sites of insulin injections. Significant importance in the pathogenesis of lipodystrophy is attached to prolonged trauma to tissues and branching of peripheral nerves by mechanical, thermal and physicochemical irritations from injections. It is impossible to exclude the development of a local allergic reaction to insulin in the genesis of lipodystrophy.

For the prevention of lipodystrophy, patients must observe the following rules for administering insulin:

a) alternate the injection sites daily, according to the scheme, on the days of the week. Change the area of insulin injection at least once every 1-2 months;

b) inject insulin with a temperature not lower than room temperature, the optimal temperature of insulin should be close to body temperature; in any case, do not inject insulin immediately after taking it out of the refrigerator;

c) after treating the skin with alcohol, let the alcohol evaporate from tothe surface of the skin, since the ingress of alcohol under the skin contributes to the progression of lipodystrophy;

d) after injections, it is necessary to lightly massage the site of insulin injection.

If lipodystrophy has already developed, the fight against it should be started by changing the type of insulin. Most effectiveIn this case, monocomponent and human insulin preparations are used, which must be injected at the border of the areas of lipodystrophy and healthy tissue. In order to blockade sources of irritation and restore trophism, insulin-novocaine mixtures are topically applied. At the same time, insulin is mixed with a 0.5% solution of novocaine and the places of lipodystrophy are covered. If there is no effect, hydrocortisone, anabolic steroids, can be added to the mixture.

Insulin allergy. Insulin is a protein preparation, the introduction of which can cause an allergic reaction, local or general. An allergic reaction can develop immediately after insulin injections or one or several days after the start of insulin therapy. The most common form of allergic reactions to insulin is local skin manifestations, less often it developslesion in the form of urticaria and even less often - anaphylactic shock.

A local allergic reaction is manifested by an erythematous and hot papule, accompanied by itching of the skin. The generalized form is characterized by urticaria, severe pruritus, dyspeptic symptoms, hyperthermia, chills, angioedemakami, erosive damage to the mucous membranes. An extremely severe manifestation of insulin allergy is anaphylactic shock with the development of collapse and impaired respiratory function.

Prevention of allergic reactions consists in the use of highly purified insulin preparations. The first measure in case of local insulin allergy is to change the insulin preparation. The preference in this case followsgive less immunogenic drugs, i.e. human insulin and monocomponent. In addition, antihistamines are prescribed (suprastin, pipolfen, diphenhydramine, tavegil, fenkarol), calcium preparations, antiserotonergic drugs (peritol). In case of a general allergic reaction, it is necessary to resort to the appointment of glucocorticoid drugs, immunomodulators, hemosorption.

New ways of administering insulin. Everydayinsulin injections throughout life are accompanied by a number of inconveniences for the patient. In addition, most of the subcutaneously injected insulin acts bypassing the liver, not providing the physiological need of this organ for insulin, which in healthy people is about 50% of the total amount of hormone released from the pancreas. In this connection, research has been carried out for many years with the aim of improving insulin therapy. In Japan and Ukraine, the rectal method of insulin administration was tested, which turned out to be ineffective. In Japan, Italy and France, intranasal administration of insulin was used, which also did not give the desired effect. In the clinic of endocrinology of the Belarusian Institute for the Advanced Training of Doctorsstudies are being conducted on the administration of insulin by electrophoresis to the liver area. In recent years, there have been attempts to administer insulin per os in liposomes prepared from phospholipids subjected to ultrasonic treatment. Many researchers believe that the most promising dosage form of insulin is its liposomal form, which allows replacing injections with a more physiological oral administration of the hormone. The degree of the hypoglycemic effect of the liposomal form of insulin is influenced by the chemical composition of lipids, in particular, neutral dipalmitoylphosphalidylcholine. Turning on insulin in liposomes contributes to the prolongation of its effect.

The prospect of using liposomal form insulin in clinical practice remains open until a set of additional studies necessary to obtain approval for clinical trials of the drug is carried out.

Tableted sugar-lowering drugs

... *Since the 50s* biennium of our century, a new stage in the treatment of diabetes mellitus has begun, associated with the use of tableted sugar-reducing drugs. Currently, the clinic uses sulfonylurea preparations (sulfa drugs) and biguanides.

Sulfanilamide sugar-reducing drugs.

Sulfanilamide drugs (SAP) are sulfonylurea derivatives. The mechanism of action of sulfa drugs is multifaceted. It is known that sulfonamides:

1) increase the sensitivity of beta cells to the release of insulin with an increase in blood glucose levels;
2) activate the absorption of glucose by cells of muscle tissue and liver;
3) inhibit gluconeogenesis;
4) inhibit lipolysis;
5) inhibit the secretion of glucagon;
6) increase the sensitivity of peripheral tissues of receptors to insulin.

In clinical diabetology, 2 groups of FAP are used: drugs of the first generation and drugs of the second generation, which have a pronounced hypoglycemic effect and give fewer side reactions.

SAP I generation:

/ group - D-860 (commercial drugs: orabet, rasinone, tolbutamide). The drugs are available in tablets of 0.25 and 0.5 g, the maximum therapeutic dose is 2.0 g / day. These are the weakest FAP preparations in terms of the hypoglycemic effect. They can cause side reactions in the form of dyspeptic disorders, skin rashes and other manifestations.

// group - BZ-55 (commercial drugs bucarban, carbutamide, oranil). The drugs are available in tablets of 0.5 g, the maximum therapeutic dose is 1.5 g / day. If there is no

effect when taking 1.5 g of the drug per day, it is inappropriate to increase the dose, since the sugar-lowering effect increases slightly, side effects are more pronounced, which, in addition to dyspeptic disorders and skin rashes, include inhibition of bone marrow hematopoiesis with the development of agranulocytic reactions.

/// group - R-607 (commercial drugs chlorpropamide, diabenez). Produced in 0.1 and 0.25 g. The listed drugs have the strongest hypoglycemic effect among the first generation SAP. The maximum dose is 0.5 g / day. The side effects are the same as in the BZ-55 group.

IV Group - K-386 (commercial drugs cyclamide, diaboral). Available in tablets of 0.25 and 0.5 g, the maximum daily dose is 1.5 g.

Have a group - N-717 (commercial drugs redul, glycodiazone). Available in tablets of 0.5 g, therapeutic dose - 1.5 g / day.

Preparations of the second generation have a sufficient hypoglycemic effect when used in small doses (from 5 to 15 mg). To sugar-reducing drugs
elevengenerations include:

/ group - glibenclamide: daonil, maninil, euglucon. Available in tablets of 0.005 g. Effective daily dose - 0.015 g. The maximum effect of the drug occurs 4 hours after administration, duration - up to 14 - 24 hours.
12 h. The effect of treatment should be taken into account after 5 days from the start of therapy.

// group - gliclazide: diamicron, predian, diabeton. Produced in 0.025 g. Daily dose - 0.05 g, duration of action 12 - 24 hours.

III group - glipizide: minidiab, antidiab. Produced in 0.005 g, daily dose - 0.01 g, duration of action 16 - 24 hours.

1U group - glycidone: glurenorm. Allowed for kidney damage, because 95% is excreted by the intestines and only 5% by the kidneys. Their duration of action is up to 10 hours.

In recent years, sulfanilamide preparations have been synthesized that differ from the previous ones in their ability to have a double pharmacological effect - metabolic (hypoglycemic) and hemovascular (angioprotective). Drugs with a similar effect are widespread. In our country, predian, diamicron, diabeton are used, they are available in tablets of 0.04 g - 0.08 g, the daily dose is 0.16 g.

Promising in the treatment of diabetes drugs that are excreted by the gastrointestinal tract (glurenorm) - 30 mg / tab., Daily dose - 120 mg. They can be used in patients with impaired liver and kidney function.

Generation III preparations.

Glimipiride group: amaryl is a more active drug, the effect is stable during the day.

Repaglinide group: Novonorm has a short period of hypoglycemic action (1 - 1.5 hours), which allows it to be used before each meal to eliminate postalimentary (postprandial) hyperglycemia.

Aktos - reduces insulin resistance.

Sulfonylurea preparations are the main after diet and exercise by the treatment of type 2 diabetes in patients with normal and overweight.

The appointment of SAP is contraindicated:

1. With precomatose and coma states.
2. During pregnancy and lactation.
3. In childhood.
4. In preoperative preparation.
5. With severe violations of liver and kidney function.

6. In diseases of the blood, accompanied by leukopenia, thrombocytopenia.

Complications during treatment with sulfa drugs can manifest themselves not only in the form of skin allergic reactions, but also in the form of hypoglycemic conditions. (develop mainly after taking chlorpropamide or maninil). Dyspeptic disorders (salivation, nausea, discomfortin the epigastric region, loss of appetite). It should be remembered that FAP of the first generation can affect hematopoiesis with the subsequent development of leukopenia and thrombocytopenia; in addition, FAPs increase the concentration of blood fibrinogen.

Some patients with type 2 diabetes are resistant to FAP.

Primary resistance is manifested by the lack of effect from the appointment FAP at the beginning of drug therapy. In order to avoid primary resistance to FAP, it is advisable to select strictly for the treatment of oral antihypertensives. The conclusion about the absence of the effect of FAP therapy can be made not earlier than after 5-7 days of using the drug.

Secondary resistance to FAP develops after 6-10 years or more from the start of treatment. The causes of this condition may be an inadequate dose of the drug, stressful situations, infectious diseases, as well as depletion of beta cells due to prolonged stimulation.

Biguanides.

In addition to SAP as a sugar reducing agent in nonin which cases biguanides, which are derivatives of guanidine, are used. According to their chemical structure, they are divided into 2 groups:

1. Dimethylbiguanides (metformin, glucophage).
2. Butylbiguanides (adebit, silubin, buformin). Biguanide preparations are produced with a duration of 6-8 or 10-12 hours. Prolongation is achieved by using granular forms in the form of capsules or multilayer tablets, which slows down the absorption of the drug.

The main mechanism of action of biguanides is to increase the utilization of glucose by the muscles due tovation of anaerobic glycolysis. In addition, biguanides inhibit gluconeogenesis, the breakdown of glycogen in the liver, and slow down the absorption of glucose in the small intestine. At the same time, these drugs have anorexigenic, lipotropic effect and help to reduce the level of triglycerides in the blood serum. They activate fibrinolysis.

Features of the mechanism of action of biguanides witnessThere is an argument about the advisability of prescribing these drugs for patients with type 2 diabetes if they are obese.

Side effects in treatment with biguanides are observed in the form of dyspeptic disorders. A dangerous complication is lactic acidosis due to the accumulation of lactic acid due to the activation of anaerobic glycolysis. In this connection, the appointment of biguanides is indicated for: heart failure; conditions accompanied by chronic hypoxia; acute inflammatory diseases; with damage to the liver, kidneys and blood.

Elderly persons should be prescribed biguanides with extreme caution, at a dose of no more than 50 mg / day and under strict control of the general condition. Because of the severe disorders associated with lactic acidosis, biguanides are used less and less.

Combined therapy with SAP and biguanides is possible, which allows the use of minimal doses of drugs. Combination therapy eliminates the predominance of anaerobic glycolysis, since SAP stimulates aerobic glycolysis.

Phytotherapy with diabetes mellitus.

Blueberries. Leaves and berries are harvested. They contain tannins, glycosomethylene, glucose, fructose, citric and malic acid, vitamins PP, C, Br, calcium, phosphorus, iron,

manganese. The healing property is manifested in the same wayin fresh and dried berries. Sharpens vision, lowers blood glucose levels, active angioprotector.

Preparation of the infusion: 60 g of leaves, stems per 1 liter of water, boil for 9-10 minutes, leave for 5-6 hours, drain. Take 1-2 tablespoons a day.

Lingonberry. Benzoic acid is contained in the berry, due to which the lingonberry is well stored, has an antiseptic, choleretic, sugar-reducing effect. An infusion of leaves is prepared as in the first case. Dried lingonberry berries are poured with 1 cup boiling water for 1-2 hours.Drink during the day. Fresh berries - 1 glass a day.

Bean pods. Harvested in August. For 100 g of bean pods, take 1 liter of water, which is boiled to 500 ml, cooled, filtered. Take 1 / g of a tablespoon 3-4 times a day.

Walnut leaves. Dries quickly in the sun. 60 g of leaves per 1 liter are boiled for 3 minutes. Drink '/ g glass after meals. You can rinse your mouth with periodontal disease.

Broth of oats. Grain with husk is collected. 100 g of grains - for 3 cups of water. Boil for 1 h, then cool for 7 hgive in a warm place, cover with a rag, towel. They are filtering. Take 1 tablespoon before meals.

Strawberries, strawberry tea. 3 g leaves per 400 g boiling water, close, insist. Drink in a day. Fresh berries: 3-4 tablespoons per 1 glass of water; 2 tablespoons of dry berries to insist as tea and drink (with atherosclerosis, diseases of the stomach, liver, diabetes).

Herbal mixtures: No. 1 - goat's rue, bean husk, blueberry leaves, dandelion root, young nettle leaves, 25 g of each herb. 1 tablespoon of the mixture in 1 glass of boiling water, brew like tea, infuse overnight, 1 glass per day.

No. 2 - field horsetail, 1 part; oat husk, 1 part; flax seeds, 1 part; the root of the bait, f% of the part. 1 tablespoon of the mixture in 1 glass of water. Boil for 45 minutes over low heat. Refrigerate. 1 tablespoon 3 times a day.

No. 3 - a set of herbs called "Arfazetin" - 10.0 g. Blueberry shoots - 2.0 g; aralia root (zamanihi) - 1.5 g; rose hips - 2.0 g; horsetail herb - 1.0 g; St. John's wort herb - 1.0 g; chamomile flowers - 1.0 g; sashbeans - 2.0 g. Pour boiling water over one packet, insist, take 1/3 cup 3 times a day.

Take all herbal preparations before meals.

***Compensation criteria* With D.**

With type 1 diabetes:

1. Lack of complaints and preservation of performance.
2. Normal parameters of physical development.
3. Absence of hypoglycemic conditions and acetonuria.
4. Fasting glycemia 5.0-5.5 mmol / l, during the day — not higher than 10.0 mmol / l.
5. Aglycosuria.
6. The content of glycosylated hemoglobin is not more than 7%.
7. Normal blood lipids and proteinograms.
8. Normal indicators of humoral and cellular immunity.

With type 2 diabetes:

1. Normal body weight.
2. Retention of ability to work and well-being.

No hypoglycemia.

4. Fasting glycemia 4.5-5.0 mmol / l, during the day - no more than 9.0 mmol / l.
5. Aglycosuria.
6. The content of glycosylated hemoglobin is not more than 6%.
7. Normal indicators of lipid metabolism.

Clinical examination...

The goal is to preserve the ability to work and prevent complications.

Methodology for registration of primary patients:

1. Genealogical history.
2. Determination of the HLA phenotype (with type 1 diabetes).
1. Objective clinical examination and laboratory tests.
2. Examination by an ophthalmologist, neuropathologist, dentist (for women - a gynecologist).
3. Self-control training.

In the process of further observation:

1. Control over the dynamics of the course of the disease, body weight.
2. Optimizing treatment to maintain compensation.
3. Examination of the insulin injection sites, the condition of the legs.
4. Specialist consultations for early diagnosis of complications.

The list of necessary studies to diagnose complications:

1. Retinopathy - examination by an ophthalmologist, ophthalmoscopy.
2. Nephropathy - glomerular filtration rate, urinary syndrome, microalbuminuria, blood pressure, creatinine and urea in the blood.
3. Neuropathy - definition of tactile, pain and vibration sensitivity, study of reflexes, heart rate, measurement of blood pressure lying and standing (orthostatic hypotension).

Laboratory research: fasting blood glucose and during the day, glycosuria and acetone in urine, triglycerides, cholesterol and beta-lipoproteins of blood, proteinogram, potassium, sodium, blood calcium, glycosylated hemoglobin, IRI and C-peptide, microalbuminuria.

Other studies: 1. Isotope renography. 2. Determination of tissue blood flow of the leg. 3. Rheovasography with nitroglycerin test. 4. ECG at rest and with veloergometry. 5. Chest X-ray.

The basics of self-control. The goal is the ability to supportlive compensation, take urgent action in case of hypoglycemia, acetonuria. Methodology - individual and group training. Trainers: endocrinologist, nutritionist, nurse, exercise therapy instructor, psychologist. The training program should include sections:

1. Fundamentals of etiology and pathogenesis with D.
2. Clinical manifestations of the disease ..
3. Emergencies and their causes.
4. Diabetes treatment (diet, exercise, hypoglycemic drugs).
5. Control over compensation at home.
6. Labor orientation, sports.
7. Family issues, pregnancy planning, child rearing.

Training is best done in an endocrinology department. For this, special"Diabetes schools", that is, schools for teaching self-management based on the diabetes department. According to the order № 197 of 01.08.94, the rates of doctors and nurses were allocated for the training of patients with diabetes mellitus.

COMATIC CONDITIONS IN DIABETES MELLITUS...

Acute complications of diabetes mellitus.

There are 3 hyperglycemic comas: hyperketoacidotic, hyperosmolar, hyperlacticidemic and 1 hypoglycemic coma.

Ketoacidosis and ketoacidotic coma...

Diabetic ketoacidotic coma is one of the most severe complications of diabetes mellitus and occurs as a result of increasing insulin deficiency. The development of diabetic

ketoacidosis (KA) is more typical for type 1 diabetes. However, it is important to remember that diabetic CA can also develop in patients with type 2 diabetes under conditions of stressful situations and intercurrent diseases leading to diabetes mellitus decompensation. The state of the spacecraft may be due to:

a) insufficient administration of insulin (incorrect calculation of the daily dose or inadequate distribution during the day);

b) changing the insulin preparation;

c) violation of the technique of insulin injection (injection into the site of lipodystrophy, into the inflammatory infiltrate);

d) cessation of insulin therapy;

e) an increase in the need for insulin (due to intercurrent diseases, pregnancy, trauma, surgical interventions);

f) violation of the diet, especially the abuse of fatty food.

In addition, to date, there are cases when the diagnosis of diabetes mellitus is first established in a state of ketoacidosis.

The development of diabetic CA in accordance with modernvariable data can be represented by the following stages.

Ketoacidosis Stage I *(KA I st.).*

Pathogenesis: first the stage is characterized by increasing hyperglycemia, which is the result of insulin deficiency. At the same time, against the background of an increase in blood sugar levels, cells experience energy starvation, since insulin deficiency prevents cells from utilizing glucose. Energy starvation of body cells leads to the activation of endogenous glucose formation due to gluconeogenesis and glycogen breakdown. These processes are stimulated by glucagon, catecholamines, glucocorticoids. However, the utilization of glucose by cells in this situation does not improve due to insulin deficiency, as a result of which hyperglycemia increases even more. Hyperglycemia is accompanied by an increase in the osmotic pressure of blood plasma, cell dehydration, polyuria and glycosuria (glucose begins to be excreted in the urine at a glycemic level of 10-11 mmol / l).

An obligatory factor in the pathogenesis of ketoacidosis isactivation of the formation of ketone bodies. Insulin deficiency and an excess of counterinsular hormones (primarily somatotropic, which has a lipolytic effect) lead to the activation of lipolysis and an increase in the content of free fatty acids (FFA), which are a ketogenic substrate. In addition, the synthesis of ketone bodies of beta-hydroxybutyric and acetoacetic acids occurs from "ketogenic" amino acids (isoleucine, leucine, valine), which accumulate as a result of activation of protein catabolism in conditions of insulin deficiency. The accumulation of ketone bodies leads to the depletion of alkaline blood reserves and the development of metabolic acidosis. In the first stage of CA, moderate acetonuria appears, which may be unstable.

Clinical manifestations…

Diabetic CA, as a rule, it develops slowly, over several days, against the background of diabetes mellitus decompensation. In the stage of moderate CA, there is a loss of working capacity, muscle weakness, and loss of appetite. Headaches, dyspeptic symptoms (nausea, diarrhea) appear. On thepolyuria and polydipsia will melt. Examination reveals dry skin, tongue and oral mucosa, a slight odor of acetone from the mouth, muscle hypotension, rapid pulse, muffled heart sounds, and may be arrhythmic. Sometimes abdominal pain is noted, an enlarged and painful liver can be palpated.

Laboratory criteria...

Laboratory research allow detecting hyperglycemia (up to 18-20 mmol / l), glycosuria, ketonemia (up to 5.2 mmol / l) and ketonuria (ketone bodies in the urine are weakly positive). The water-electrolyte balance at this stage of the development of ketoacidosis is characterized by insignificant hyperkalemia due to the release of potassium from the cell (up to 5 mmol / L), the potassium deficiency in the cell is confirmed by the data of electrocardiography (asymmetric decrease in the S - T interval, biphasic T wave, which may be negative). The acid-base state (ACS) does not change significantly, but the content of hydrocarbonates decreases to 20-19 mmol / l.

Treatment.

Patients with diabetes mellitus with Art. diabetic CA should be hospitalized in the endocrinology department. An important event at this stage is the change in dietary regimen. The patient is prescribed easily digestible carbohydrates, fruit juices, honey. The composition of foodoat broths, cereals, jelly are introduced into the new diet. The total amount of carbohydrates increases to 60-70% (instead of 50%) to suppress ketogenesis. Correction of hyperglycemia is carried out with short-acting insulin (Aktrapid, Iletin, Humulin, Rapitard), in divided doses, at least 6 times a day intramuscularly or subcutaneously. The calculation of the daily insulin dose is made from 0.7 U / kg of actual body weight. To eliminate acidosis, alkaline mineral waters (Borjomi), dimephosphon, and rehydron are prescribed. Also shown are cleansing enemas with 3% sodium bicarbonate solution. The listed measures, as a rule, are sufficient to remove patients from the state of diabetic CA 1st stage. In the case of pronounced signs of dehydration, intravenous fluid is prescribed.

It should be noted that early diagnosis and timely treatment help prevent the transition of the initial stage of ketoacidosis to a precomatose state, which is dangerous for the patient's life.

Precomatose state (CA II degree).

Pathogenesis...

In the development of diabetic CA II stage. the following pathogenetic furs join the previously listed onesnism: energy deficiency of cells in conditions of insulin deficiency is accompanied by activation of protein breakdown, which leads to disruption of nitrogen balance and contributes to the development of azotemia. The production of endogenous glucose from glycogen, fat and protein continues to increase, glycosuria and polyuria increase due to osmotic diuresis. First, glycosuria and hyperglycemia lead to cellular dehydration, and then to general dehydration with a decrease in tissue and renal blood flow and a deficiency of electrolytes (Na +, K +, C1 ~).

Violation of renal blood flow contributes to the growth of ketoacidosis, since the kidney stops the production of bicarbonate ion (HCO3). The activation of ketogenesis is aggravated by metabolic acidosis, which is accompanied by a decrease in blood alkalinity and a shift in pH. Discordthe presence of hydrogen ions in the respiratory center leads to the appearance of a characteristic noisy and infrequent respiration. In addition, as a result of the activation of lipolysis, FFA, NEFA, triglycerides accumulate in the blood, which increases the viscosity of the blood and contributes to the violation of the hemorheological properties of the blood and the deterioration of microcirculation.

Clinical manifestations…

In IIArt. of diabetic CA, the general condition of the patient deteriorates sharply, thirst and polyuria rapidly increase. The patient experiences progressive muscle weakness and cannot move independently, lethargy appears. Stubborn vomiting joins the nausea, abdominal pains appear and intensify, pains in the heart often occur. On examination, there

is a sharp dryness of the skin and mucous membranes, rubeosis of the face (the result of capillary paresis). Tongue dry, crimson or coated with a dark brown bloom, sharp smell of acetone from the mouth. Reduced muscle tone. The tone of the eyeballs is also lowered. Breathing becomes rare, deep, noisy (breathing of Kussmaul). On the part of the cardiovascular system, the following changes are noted: frequent and small pulse, tachycardia, heart soundshie, there may be arterial hypotension. Hypokalemia leads to decreased bowel motility. As a resultthe onset of intestinal atony, it is overstretched by the contents, which leads to the appearance of pain. In addition, there is tension in the muscles of the anterior abdominal wall. The listed changes may serve as a reason for an erroneous diagnosis of the "acute" abdomen syndrome. It should be borne in mind that diabetic ketoacidosis is accompanied by leukocytosis with neutrophilia and a shift in the formula to the left (due to hypovolemia). Therefore, the picture of the "acute" abdomen, confirmed by laboratory parameters, seems to be very convincing. This may result in unnecessary laparotomy, which worsens the course of diabetic ketoacidosis.

Depending on the prevalence of certain symptoms in the clinic of ketoacidosis, the following variants of the precomatose state are distinguished:

a) cardiovascular, or collaptoid, form;

b) gastrointestinal (abdominal), or pseudoperitoneal, form;

c) renal form;

d) encephalopathic form.

In the clinic of the cardiovascular form in the foreground there are phenomena of vascular collapse with severe cardiovascular insufficiency (cyanosis, tachycardia, inspiratory dyspnea, heart rhythm disturbances such as extrasystole or atrial fibrillation). The listed manifestations mimic the picture of acute myocardial infarction or thromboembolism of small branches of the pulmonary artery.

Abdominal form characterized by the predominance of dyspeptic symptoms and abdominal pain in the clinic, and also by tension of the muscles of the anterior abdominal wall, which imitates the picture of an "acute" abdomen. Indomitable vomiting and severe diarrhea can mimic acute gastroenteritis.

In the renal form, dysuric phenomena with pronounced urinary syndrome (abouttheinuria, hematuria, cylindruria, hypoisostenuria) in the absence of pronounced glycosuria and ketonuria, which may be a consequence of a decrease in glomerular filtration. Perhaps the development of anuria and acute renal failure with increasing azotemia. Usually, a similar course of ketoacidosis is observed in patients with diabetic nephropathy.

Encephalopathic form characterized by a clinic resembling an acute violation of cerebral circulation, which is due to insufficient blood supply to the brainha, intoxication, punctate hemorrhages.

Laboratory criteria: in the precoma stage, glycemia up toreaches 20-30 mmol / l, accompanied by severe polyuria and an increase in plasma osmolarity up to 320 mosm / l, the water-electrolyte balance is significantly disturbed, which is manifested by hyponatremia (less than 130 mmol / g), hypokalemia (less than 4.0 mmol / l). This stage of development of ketoacidosis is characterized by obvious violations of acid base balance (blood alkalinity decreases - less than 40 vol%, HCO3 - to 10-12 mmol / l, pH from 7.35 to 7.10). Protein catabolism due to gluconeogenesis is accompanied by an increase in blood urea and creatinine, proteinuria. It should be remembered that urinary syndrome and the accumulation of nitrogenous compounds in the blood are especially pronounced in patients with diabetic nephropathy.

If in a precomatose state the patient does not receive appropriate assistance, a ketoacidotic coma develops within 1-2 hours (stage III diabetic ketoacidosis).

Ketoacidotic coma...

Pathogenesis...

Progressive dehydration leads to hypovolemia, accompanied by impaired blood flow to the kidneys and brain. The progression of ketoacidosis aggravates hypoxia.Deficiency of sodium, chlorides and especially potassium is increasing.

Hypovolemia, hypoxia and potassium deficiency largely determine the course of a coma. One of the factors in the pathogenesis of hypoxia is an intense increase in the content of glycosylated hemoglobin (bound to glucose and intolerant of oxygen). Hypovolemia leads to the development of circulatory hypoxia. Electrolyte imbalance is accompanied by myopathy, including weakness of the respiratory muscles, which leads to the addition of a component of alveolar hypoxia. The progression of hypoxia promotes the activation of anaerobic glycolysis and the accumulation of lactic acid in the tissues.

The listed factors (hypovolemia, hypokalemia, hypoxia and an increase in lactic acid content) contribute to the development of cardiovascularvascular insufficiency and metabolic coagulopathy. Coagulopathy is manifested by disseminated intravascular coagulation, peripheral thrombosis and thromboembolism. Hypovolemia is usually accompanied byimpaired renal perfusion.

Clinical manifestations…

For ketoacidoticcoma is characterized by complete loss of consciousness. Breathing is noisy, such as Kussmaul, a sharp smell of acetone from the mouth and in the room where the patient is. On examination, the skin and mucous membranes are dry, cyanotic, and the facial features are pointed. The tone of the muscles and eyeballs is sharply reduced, the pupils are narrowed. Tendon, periosteal and cutaneous reflexes are absent. The pulse is quickened, threadlike, possibly a violation of the heart rhythm. Arterial hypotension is expressed. The tongue is dry, coated with a dark brown coating, the abdomen is slightly swollen, the abdominal wall may be tense against the background of general muscle hypotonia. On palpation, a dense, enlarged liver is determined. The body temperature is lowered (if the patient does not have concomitant infectious and inflammatory diseases). Urination is involuntary, may be oligo- or anuria.

Laboratory criteria.

Glycemia at the stage of ketoacidotic coma, as a rule, exceeds 30 mmol / L, which is accompanied by an increase in plasma osmolarity up to 350 and more mosm / l. In addition, at this stage, the deficiency of sodium, chlorides and potassium increases, and hyperazotemia increases. The acid-base state is characterized by progressive acidosis (pH drops to 7.1 or less), reserve alkalinity (30%) and blood hydrocarbons sharply decrease. The lactic acid content increases to 1.6 mmol / l or more.

Treatment of precomatose and ketoacidotic coma...

When a patient with diabetes mellitus develops a precomatose state or coma, emergency hospitalization is required in the intensive care unit, where intensive treatment immediately begins, which includes the following components:

a) insulinosisrapia;

b) rehydration;

c) correction of electrolyte balance,

d) restoration of the acid-base state;

e) correction of cardiovascular disorders;

f) elimination of the factor that caused ketoacidosis.

Insulin therapy.

Insulin therapy is carried out by administering short-acting insulin preparations (Aktrapid Rapitard, Iletin R, Humulin). Currently, the adopted method of insulin therapy, which is called the method of low doses. This technique is based on a continuous intravenous infusion of insulin. The dose of insulin is determined depending on the initial level of glycemia (if the glycemia is less than 20 mmol / l, 8-12 units of insulin are administered per hour;kemia from 20 to 30 mmol / l - 12-14 U / h; with glycemia above 30 mmol / l - 14-16 U / h). The optimal rate of glycemic decline is considered to be from 3.0 to 6 mmol / l per hour, depending on the initial level. After reaching a glycemic level of 11.0 mmol / l, insulin is prescribed subcutaneously or intramuscularly, 4-6 units every 3-4 hours under glycemic control. The glycemic level is maintained in the range of 5-9 mmol / l. This method of insulin therapy is considered the most effective and safest. The introduction of intravenous insulin at the beginning of treatment ensures its entry into the circulation in conditions of dehydration, and small doses prevent a sharp decrease in the level of glycemia, aggravating hypokalemia and leading to the development of cerebral edema.

Rehydration.

Rehydration is prescribed simultaneously with insulin therapy by the introduction of isotonic saline solutions (0.85% sodium chloride solution, Ringer's solution), plasma, low molecular weight dextrans. Rehydration speedtation is 1 l / h during the first two hours, then 500 ml / h during the next 2 hours (i.e., 3 liters for 4 hours of treatment). Infusion therapy should be carried out under the control of urine output, which should be at least 40-50 ml / h. After reaching a glycemic level of 11.0 mmol / l, when the diffusion of drugs into the cell begins, it is necessary to use a 5% glucose solution, which helps to inhibit ketogenesis. In addition, glucose infusion avoids hypoglycemic syndrome and reduces energy starvation of cells. The total volume of fluid administered to the patient during the day is 5-6 liters or more.

Correction of electrolyte disturbances.

An important component of the treatment of ketoacidosis is the correction of electrolithic disorders, and especially potassium deficiency. Before rehydration begins, patients may experience relative hyperkalemia due to the release of intracellularpotassium into the extracellular space and hemoconcentration. Serum potassium levels during rehydrationintracellular potassium deficiency decreases and increases, which is manifested by characteristic ECG signs. It should be noted that hypokalemia is one of the main causes of patient death.

*Correction of hypokalemia*carried out by the introduction of potassium chloride as part of a potassium polarizing mixture, usually begins 1 hour after the start of treatment, when, against the background of rehydration and a decrease in glycemia, the diffusion of potassium into the cell is ensured. If the serum potassium level on admission to the department is less than 4.0 mmol / l, potassium administration is started simultaneously with rehydration and insulin therapy. The rate of potassium administration is determined by the baseline serum potassium level. With a potassium content in the blood serum of 4.0 mmol / l and more infupotassium chloride is carried out at a rate of 2 g / h, and with a potassium content below 4.0 mmol / l - at a rate of 3 g / h. Serum potassium levels are maintained at 4.3–4.8 mmol / L. The intracellular potassium concentration is judged by ECG data. Correction of sodium and chloride ions is achieved in the process of rehydration by the introduction of physiological sodium chloride solution, Ringer's solution, and, if necessary, 10% sodium chloride solution (5.0-10.0 ml intravenously).

Normalization of acid-base balance.

Indications for intravenous bicarbonate administration are currently revised sodium. A

solution of sodium bicarbonate is introduced when the pH of the arterial blood is shifted to 7.2 and the reserve alkalinity is reduced to 30.0% by volume. Limitations of indications for infusion of soda solutions are associated with the possible development of alkalosis and aggravation of hypokalemia. The danger of alkalosis development is due to the fact that with the normalization of renal perfusion against the background of rehydration, the synthesis of endogenous hydrocarbonate ion in the kidneys is restored, which contributes to the elimination of acidosis due to its own hydrocarbonates.

The issue of the dose of sodium bicarbonate for intravenous administration has also been revised. On average, 400-600 ml of 2.5% sodium bicarbonate solution, 200-300 ml each, is injected during the day with an interval of 3-4 hours.

Correction of cardiovascular disorders.

Correction of vascular insufficiency is performed by rehydrationrationing (combating hypovolemia, as well as the introduction of mezaton, cordiamine, DOXA). The indications for the appointment of cardiac glycosides are signs of heart failure (mainly of the left ventricle), which develop with intensive rehydration against the background of a decrease in the filtration function of the kidneys, mainly in patients with diabetic nephropathy, myocardial dystrophy or ischemic heart disease.

In the treatment of precomatose state and ketoacidotic coma, three stages can be distinguished, which have methodological features. Stage I - until glycemia is reached 16.8 mmol / l; Stage II - glycemia from 16.8 to 11 mmol / L;Stage III - glucose is below 11 mmol / l. As therapeutic measures at the first stage, intravenous insulin therapy, intensive rehydration, correction of acid base balance and vascular insufficiency are required. At stage II, as mentioned above, it is necessary to introduce a potassium polarizing mixture on glucose, since the diffusion of glucose and potassium into the cell begins. After reaching a glycemia of 11 mmol / l, you can switch to subcutaneous insulin administration and begin feeding the patient with food containing easily digestible carbohydrates, mainly fructose (jelly, fruit juices, oat and semolina porridge, honey). Foods containing fats are excluded from the diet.

Success in the treatment of precomatose and coma is determined by the timeliness of the initiation of intensive care, the state of the cardiovascular system, kidneys, the patient's age and the cause of ketoacidosis.

It must be remembered that prognostically unfavorable signs in the course of diabetic ketoacidotic coma can be:

arterial hypotension, not amenable to correction with rehydration and the introduction of corrective blood pressure drugs,

decrease in urine output to 30 ml / h and less,

acute left ventricular failure,

hemorrhagic syndrome (often in the form of gastrointestinal bleeding), hypokalemia and an increase in the content of lactic acid in the blood.

At the same time, it should be emphasized that over the past 10 years, as a result of the introduction into practice of methods of internaltrivenous infusion of insulin in small doses, adequate correction of hypokalemia and limitation of indications for intravenous administration of sodium bicarbonate solutionstality from ketoacidotic diabetic coma decreased by more than 3 times.

Hypoglycemic conditions and hypoglycemic coma.

Hypoglycemia - state organism caused by a sharp decrease in blood sugar (glucose) levels and insufficient supply of glucose to the cells of the central nervous system. The most

severe manifestation of a hypoglycemic state is hypoglycemic coma.

Etiology...

Hypoglycemic conditions can occur in diabetes mellitus due to:

1) overdose of injected insulin or sulfa hypoglycemic tablet drugs;

2) eating disorders in case of untimely food intake after insulin injection or food intake with an insufficient amount of carbohydrates;

3) hypersensitivity to insulin (especially in childhood and adolescence);

4) lowering the insulin inactivating ability of the liver (insufficiency of insulinase production or activation of its inhibitors);

5) alcohol intoxication (slowing down the breakdown of glycogen);

6) chronic renal failure (an increase in the circulation time of sugar-reducing drugs as a result of a slowdown in their excretion in the urine);

7) intense physical activity, accompanied by increased glucose utilization;

8) compensatory hyperinsulinism in the early stages of diabetes mellitus;

9) taking salicylates, sulfa drugs, adrenergic blockers when prescribed in combination with insulin or antidiabetic drugs in tablets.

Pathogenesis...

The basis of the pathogenesis of hypoglycemia is a decrease in glucose utilization by cells of the central nervous system. It is known that free glucose is the main energy substrate for brain cells. Insufficient supply of the brain with glucose leads to the development of hypoxia with subsequent progressive impairment of the metabolism of carbohydrates and proteins in the cells of the central nervous system. Various parts of the brain are affected in a certain sequence, which causes a characteristic change in clinical symptoms as the hypoglycemic state progresses. First of all, the cerebral cortex suffers from hypoglycemia, then subcortical structures,the cerebellum, and ultimately the functions of the medulla oblongata are disrupted. The brain gets its nutrition from carbohydrates. At the same time, little glucose is deposited in the brain. The energy requirements of the cells of the central nervous system are very high. Brain tissue consumes 30 times more oxygen than muscle tissue. Deficiency of glucose is accompanied by a decrease in oxygen consumption by the cells of the central nervous system even with sufficient oxygen saturation of the blood, and therefore, the symptoms of hypoglycemia are similar to those of oxygen deficiency.

In the pathogenesis of hypoglycemia, the decisive factor isThere is an ability to utilize glucose, therefore, hypoglycemic states can be observed even with normal blood glucose levels, but with suppression of the processes of glucose entry into the cell. Due to the lack of glucose in the cells of the most differentiated parts of the brain (cortex and diencephalic structures), irritability, anxiety, dizziness, drowsiness, apathy, inappropriate speech or actions occur. In case of damage to phylogenetically more ancient parts of the brain (medulla oblongata, upper parts of the spinal cord), tonic and clonic seizures, hyperkinesis, suppression of tendon and abdominal reflexes, and nystagmus develop.

Hypoglycemia is an adequate stimulator of the sympathoadrenal system, which leads to an increase in blood levels of catecholamines (adrenaline and noradrenaline). This is manifested by characteristic vegetative symptoms - weakness, sweating, tremors, tachycardia. At the same time, hypoglycemia causes irritation of the hypothalamus, followed by activation of counterinsular neurohormonal systems (corticotropin - glucocorticoids - somatotropin). An increase in the activity of the contrainsular systems is a compensatory reaction of the body aimed at eliminating hypoglycemia. A significant place in the elimination of hypoglycemia by self-regulation belongs to the hormone of the

pancreas -glucagon, which activates the breakdown of glycogen, in the first turn in the liver.

Prolonged carbohydrate starvation and brain hypoxia withare carried out not only by functional, but also by morphological changes, up to necrosis or edema of certain parts of the brain. An excess of catecholamines in hypoglycemia leads to a violation of the tone of the vessels of the brain and blood stasis in them. A slowdown in blood flow leads to increased thrombus formation with subsequent complications. It has been suggested that one of the causes of neurological disorders in hypoglycemia may be a decrease in the formation of amino acids and peptides necessary for the normal functioning of neurons. It should be remembered that the hypoglycemic state promotes ketogenesis. The mechanism is as follows. With a decrease in blood glucose, the development of energy deficiency, the secretion of catecholamines and growth hormone increases, which enhances lipolysis,

Depending on the individual sensitivity of the central nervous system to a lack of glucose, hypothe glycemic state occurs at different levels of glycemia - from 4.0 to 2.0 mmol / l and below. In some cases, hypoglycemic states can develop with a rapid decrease from a very high level, for example, from 20 mmol / l or more, to normal and even slightly increased blood glucose (5.0-7.0 mmol / l).

Clinical picture...

The development of hypoglycemic coma is preceded by the following clinical stages of hypoglycemia:

/ stage. Pathogenetically caused by hypoxia of cells of the higher parts of the central nervous system, mainly of the cerebral cortex.

*Clinical signs*very diverse. They arecharacterized by excitement or depression, anxiety, mood changes, headache. An objective examination can be noted the moisture content of the skin, tachycardia. Unfortunately, not all patients feel hungry at the same time, and therefore, do not regard their condition as a manifestation of a hypoglycemic reaction.

// stage. Its pathogenetic basis is the defeat of the subcortical-diencephalic region.

Clinical symptoms characterized by inappropriate behavior, demeanor, motor excitement, tremors, profuse sweating, severe tachycardia and arterial hypertension.

III stage hypoglycemia is caused by a violation of the functional activity of the midbrain and is characterized by a sharp increase in muscle tone, the development of tonic-clonic seizures resembling an epileptic seizure, the appearance of Babinsky's symptom, and dilated pupils. Severe skin moisture, tachycardia and high blood pressure persist.

IV stage associated with dysfunction, regulatedby the upper parts of the medulla oblongata (coma itself).

Clinical symptoms hypoglycemic coma accompanied by a lack of consciousness. Tendon and periosteal reflexes are increased. The tone of the eyeballs is also increased, the pupils are dilated. The skin is moist, the body temperature is normal or slightly elevated. Breathing is normal, and the smell of acetone is usually absent. Heart sounds may be increased, pulse is rapid, and blood pressure is high or normal.

V stage pathogenetically associated with increasing hyperhidrosis and involvement in the process of regulatory functions of the lower part of the medulla oblongata.

*Clinic*reflects the progression of a coma. At the same time, areflexia is observed, muscle tone decreases, profuse sweating stops, there may be a violation of breathing of the central genesis, blood pressure drops, the heart rhythm is disturbed.

It should be emphasized that in some cases atypical hypoglycemic reactions are observed, the pathogenetic basis of which is lesions of the limbic-reticular region. In such cases, the clinical signs of hypoglycemia are characterized by nausea, vomiting,

bradycardia, and mental disorders are manifested by euphoria.

A life-threatening condition accompanying hypoglycemia is cerebral edema.

The development of cerebral edema is due to several factors: late diagnosis, mistaken insulin administration or overdose hypertonic (40%) glucose solution. The clinic of cerebral edema is characterized by meningeal symptoms, vomiting, fever, respiratory and heart rhythm disturbances.

The consequences of hypoglycemic conditions can bedivided into nearest and distant. The first ones develop a few hours after a hypoglycemic reaction. These include hemiparesis and hemiplegias, aphasia, myocardial infarction, and cerebrovascular accident.

Long-term effects are observed several days, weeks or months after the development of hypoglycemicstate. They are manifested by encephalopathy, progressing with repeated hypoglycemic reactions, epilepsy, parkinsonism.

A hypoglycemic state against the background of alcohol intoxication has a particular danger in terms of adverse effects.

Diagnostics. Diagnostic criteria for hypoglycemia are divided into clinical and laboratory ones.

The first includes the listed symptoms of each of the five stages.

Laboratory criteria.

It is generally accepted that a hyperglycemic reaction is observed with a glycemia of 3.0 mmol / l or less. However, the clinic of hypoglycemia can be with blood glucose figures of 5.0-7.0 mmol / l in cases of impaired utilization of glucose by cells of the central nervous system and with a rapid drop in glucose levels from high figures.

An essential diagnostic criterion for a hypoglycemic state is a positive reaction for intravenous glucose.

Treatment.

At the prehospital stage. To stop the first stage of the hypoglycemic state, it is enough to eat food containing carbohydrates that are part of the patient's usual diet (roll, porridge, potatoes, jelly).

In the second stage of hypoglycemia, additional easily digestible carbohydrates are required (sweet tea, fruit syrup, compote with sugar, sweets, jam). As a rule, a quick intake of food containing sucrose and fructose can prevent the progression of the hypoglycemic state and normalize the glycemic level and the patient's condition. If there are no special indications, patients do not need hospitalization.

To provide effective emergency care in the third stage of hypoglycemia, immediate intravenous administration of a 40% glucose solution is required in an amount necessary to completely eliminate the clinical symptoms of a hypoglycemic reaction, but not exceeding 80.0 ml, in order to avoid cerebral edema. Sickis subject to hospitalization to prevent the early effects of hypoglycemia and adjust the dose of sugar-lowering therapy.

Stage IV and V treatment (hypoglycemic coma) is carried out in the intensive care unit. Treatment beginsit is taken from a jet intravenous injection of 80/100 ml of a 40% glucose solution, then they switch to a drip infusion of 200-400 ml of a 5% glucose solution. In this case, the level of glycemia is maintained in the range of 6.0-9.0 mmol / l. In the absence of effect, resort to subcutaneous administration of 0.5-1% solution of epinephrine or intramuscular or subcutaneous administration of 1-2 ml of 10% glucagon solution. After these measures, consciousness is restored in 15-20 minutes. It should be remembered that the action of the hormones administered is associated with the mobilization of endogenous glucose and glycogen from the depot, mainly from the liver, therefore, the frequent use of these drugs can contribute to the depletion of glycogen stores and the subsequent

deterioration of the course of diabetes, especially with concomitant liver damage.

If, as a result of the measures taken, consciousness is not restored, hydrocortisone is prescribed in a dose of 150-200 mg intravenously. As a rule, these measures are sufficient to stabilize glycemia. However, in some cases, after the restoration of glycemia, the patient does not immediately regain consciousness. In such cases, drip infusion of 5% glucose solution with 100 mg of cocarbok is continuedsilase, insulin and potassium preparations to improve the transport of glucose into the cell. Helps to improve the utilization of glucose intramuscular injection of 3-4 mg of a 5% solution of ascorbic acid.

For the prevention of cerebral edema, 5 ml of a 25% solution of magnesium sulfate is administered intravenously (enter slowly) or drip intravenous administration of mannitol 0.5-1 g / kg in the form of a 15% or 20% solution (200- 250 mg). Patients need oxygen therapy. Sometimes they resort to transfusion of fresh donor blood, since blood transfusion replaces some of the missing respiratory enzymes.

After removal from a hypoglycemic coma, it is recommended to use agents that improve microcirculation and stimulate the metabolism of carbohydrates and proteins in cells. - central nervous system (glutamic acid, aminalon, stugerone, cavinton, cerebrolysin) for 3-6 weeks, depending on the patient's condition and the course of diabetes mellitus. With the development of hypoglycemic reactions as a result of treatment with sugar-reducing tablet drugs or prolonged-release insulins, one should remember about the possibility of recurrence of hypoglycemia during the duration of the action of the corresponding drug.

Prevention.

Prevention of hypoglycemic conditions in patients with diabetes mellitus consists of two main components. The first of them consists in the appointment of adequate antihyperglycemic therapy with insulin (to avoid overdose syndrome) or tablet preparations, taking into account the functional state of the liver and kidneys.

The second component of prevention is largely due to the distribution of carbohydrates in the diet. diet in accordance with the effect of the deployment of sugar-reducing drugs, as well as the correct regulation of physical activity during the day.

Hyperosmolar hyperglycemic coma.

An acute complication of diabetes mellitus, the pathogenetic basis of which is dehydration, hyperglycemia and hyperosmolarity.

Etiology...

The development of a hyperosmolar coma methodthe following factors are involved:

1. Long-term use of diuretics, immunosuppressants, glucocorticoids.
2. Acute gastrointestinal diseases accompanied by vomiting and diarrhea (gastroenteritis, pancreatitis, food intoxication).
3. Extensive burns.
4. Massive bleeding.
5. Hemodialysis or peritoneal dialysis.

Excessive intake of carbohydrates.

7. Introduction of hypertonic glucose solutions.

Any fluid loss condition can lead to hyperosmolar coma.

Hyperosmolar coma usually occurs in patients non-insulin dependent type of diabetes mellitus against the background of insufficient treatment or with a previously unrecognized disease.

Pathogenesis...

The trigger mechanism in the development of coma is dehydration and

hyperglycemia. Hyperglycemiaat first it is accompanied by glycosuria and polyuria, and also promotes the flow of fluid from cells into the extracellular space. Osmotic diuresis increases dehydration. Fluid loss occurs not only with osmotic diuresis, but also as a result of a decrease in tubular reabsorption, as well as a decrease in the secretion of antidiuretic hormone. Increased diuresis causes intracellular and intercellular dehydration and a decrease in blood flow in organs, including the kidneys. Dehydration hypovolemia develops. Dehydration is accompanied by stasis of blood cells, aggregation of platelets and erythrocytes, hypercoagulation. In response to dehydration hypovolemia, the secretion of aldosterone increases and sodium ions are retained in the blood. Reduced renal blood flow makes sodium excretion more difficult. Hypernatremia contributes to the formation of small cell hemorrhages in the brain. In conditions of hyperglycemia and dehydration, the osmolarity of the blood sharply increases. An increase in osmolarity is accompanied by intracerebral and subdural hemorrhages. A characteristic feature of the pathogenesis of hyperosmolar coma is the absence of ketoacidosis. This feature is explained by the following factors: the presence of endogenous insulin, a decrease in lipolysis due to dehydration, inhibition of ketogenesis due to hyperglycemia. So, the pathogenesis of hyperosmolar coma consists of the following stable components: dehydration, hyperglycemia, hyperosmolarity, hypernatremia, and hypercoagulation. An increase in osmolarity is accompanied by intracerebral and subdural hemorrhages. A characteristic feature of the pathogenesis of hyperosmolar coma is the absence of ketoacidosis. This feature is explained by the following factors: the presence of endogenous insulin, a decrease in lipolysis due to dehydration, inhibition of ketogenesis due to hyperglycemia. So, the pathogenesis of hyperosmolar coma consists of the following stable components: dehydration, hyperglycemia, hyperosmolarity, hypernatremia, and hypercoagulation. An increase in osmolarity is accompanied by intracerebral and subdural hemorrhages. A characteristic feature of the pathogenesis of hyperosmolar coma is the absence of ketoacidosis. This feature is explained by the following factors: the presence of endogenous insulin, a decrease in lipolysis due to dehydration, inhibition of ketogenesis due to hyperglycemia. So, the pathogenesis of hyperosmolar coma consists of the following stable components: dehydration, hyperglycemia, hyperosmolarity, hypernatremia, and hypercoagulation.

The clinical picture is characterized by narasmelting polyuria, accompanied by severe polydipsia, dry skin and mucous membranes, a decrease in skin and subcutaneous tissue turgor. The body temperature rises. Breathing becomes surfacenym, speeded up. Blood pressure decreases, tachycardia, extrasystole are observed.

Neurological disorders deserve special attention. They largely determine the clinical picture of hyperosmolar coma. Sopor is noted,hallucinations, pathological reflexes appear (Babinsky, Rossolimo), vestibular disorders, meningeal signs, hemiparesis, paralysis. Due to hypercoagulability, thrombosis is possible. With the progression of renal failure, oliguria and azotemia appear.

Clinical symptoms develop slowly over several days, gradually leading to the stage of hypovolemic shock.

Diagnostics.

Diagnosis of hyperosmolar coma osnoshows up on characteristic clinical symptoms with inspiratory dyspnea, hyperthermia, and neurological disorders.

Laboratory diagnostic criteria: hyperglycemia - 40.0-50.0 mmol / l or more; hyperosmolarity - 350 and more mlosm / l; hypernatremia - more than 150 mmol / l; uvean increase in the content of blood urea; expressed glycosuria; signs of blood thickening.

During insulin therapy and rehydration, hypoglycemia can develop, which requires monitoring of blood glucose levels during intensive care.

Treatment.

At the prehospital stage, an emergency correction of hemodynamics is required to ensure the transportation of the patient. In all cases, urgent hospitalization is required in the intensive care unit. Treatmentconsists of the following components: rehydration, insulin therapy, correction of electrolyte disorders (hypovolemia and hypernatremia), elimination of hypercoagulation, prevention of the development of cerebral edema.

1. Rehydration. It is carried out by drip intravenous administration of a hypotonic solution of sodium chloride (0.45%) - up to 3 liters during the first three hours. Then,with a decrease in blood osmolarity, - sodium chloride. In the first 8 hours of treatment, the amount of injected fluid can reach 5 liters, and during the day - 8-10 liters.

Insulin therapy. Insulin therapy of hyperosmolar soporous and coma should be carried out with the introduction of small doses of insulin, namely:

8 U / h intravenously during the first 3 hours. After 3 hours from the start of treatment, the tactics of insulin therapy is determined by the level of glycemia and corresponds to the method of insulin therapy discussed in the section on ketoacidotic coma. In the process of insulin therapy, the glycemic level is maintained in the range of 6-10 mmol / l.

3. Correction of electrolyte disorders. Elimination of potassium deficiency is carried out according to the treatment regimen for ketoacidotic coma presented in the previous section. Hypernatremia is eliminated by rehydration of hypotensioncic solution.

4. Heparin treatment. For the prevention of thrombosis and the improvement of microcirculation requires the introduction of heparin. In the first 3 hours, in combination with a hypotonic sodium chloride solution, up to 6000 U of heparin is administered. Further administration of heparin is controlled by coagulogram indicators.

5. Prevention of cerebral edema. In order to prevent cerebral edema and to correct metabolism in the cells of the central nervous system, glutamic acid is prescribed acid intravenously in the form of a 1% solution of 30.0-50.0 ml. Oxygen therapy is a mandatory component of treatment.

Prevention of hyperosmolar coma consists of adequate therapy of diabetes mellitus with stable compensation and prevention of dehydration by careful prescription of diuretics, timely replenishment of fluid loss in diseases and conditions, accompanied by dehydration.

Lactic acidosis and hyperlactacidemic coma*...*

An acute complication of diabetes mellitus, caused by the accumulation (potentiation) in the body of lactic acid.

Etiology.

As factors contributing to the development of lactic acidosis, the following conditions can be considered:

1. Infectious and inflammatory diseases.
2. Massive bleeding.
3. Acute myocardial infarction.
4. Chronic alcoholism.
5. Severe physical injuries.
6. Chronic liver disease.

Lack of kidney function.

Taking biguanides occupies a special place among the etiological factors. It should be

emphasized that in case of liver or kidney damage, even a minimal dose of biguanidescan cause lactic acidosis as a result of the accumulation of the drug in the body.

Pathogenesis...

The pathogenesis of lactic acidosis is based on hypoxia. In conditions of oxygen deficiency,This results in activation of the anaerobic pathway of glycolysis, which is accompanied by the accumulation of excess lactic acid. As a result of insulin deficiency, the activity of the enzyme pyruvate dehydrogenase decreases, which promotes the conversion of pyruvic acid into acetyl coenzyme A. Instead, pyruvic acid is converted into lactate, which aggravates the state of lactic acidosis. At the same time, under conditions of hypoxia, the resynthesis of lactate into glycogen is inhibited.

Pathogenesis of hyperlactacidemia in treatment with biguanides associated with a violation of the passage of pyruvic acid through the membranes of mitochondria and the acceleration of the transition of pyruvate to lactate.

As a result of anaerobic glycolysis, a lot of lactic acid is formed in the tissues, which enters the bloodstream. From the blood, lactic acid enters the liver, where glycogen is formed from it. But the formation of lactic acid exceeds the ability of the liver to use it for glycogen synthesis.

Clinical picture…

Clinical signs are due to a violation of the acid-base balance. The leading syndrome is progressive cardiovascularvascular insufficiency. It is not associated with dehydration, but with acidosis. The development of coma is very rapid, however, dyspeptic disorders, muscle pain, angina pectoris can be precursors. As the acidosis progresses, abdominal pain increases, mimicking surgical conditions. Shortness of breath increases, collapse develops, Kussmaul breathing (due to acidosis) joins. Consciousness (stupor and coma) is impaired due to hypotension and hypoxia of the brain.

Diagnostics.

It is very difficult to diagnose hyperlactacidemic coma due to the nonspecificity of symptoms. However, an acute onset, dyspeptic disorders, pain in the region of the heart in a patient with diabetes mellitus with liver and kidney damage can serve as auxiliary signs in the diagnosis of this life-threatening condition.

Laboratory diagnostic criteria include:

1. An increase in the content of lactic acid in the blood (more than 1.5 mmol / l).
2. Decrease in blood bicarbonates (indicative of less than 12 mmol / l).
3. Decrease in reserve alkalinity (below 50%).
4. Moderate hyperglycemia (12-14 mmol / l) or normoglycemia in the general serious condition of the patient.
5. Lack of acetonuria.
6. The degree of glycosuria depends on the functional state of the kidneys.

Treatment.

The patient should be hospitalized in the intensive care unit. Emergency care is aimed primarily at eliminating acidosis and combating hypoxia.

1. 4% sodium bicarbonate solution - 1.0-1.5 l / day.
2. Intravenous drip of small doses of insulin and 5% glucose solution to stimulate aerobic glycolysis. For 500 ml of 5% glucose solution - 8 units of insulin.
3. Transfusion of plasma and rheopolyglucin in an amount of 500 ml / day.
2. Oxygen therapy.
4. The fight against vascular insufficiency is generallyby well-known means.
5. Intravenous drip of cocarboxylase — not less than 200 mg / day.

6. A patient with any type of sugar-lowering therapy is transferred to treatment with short-acting insulin preparations - 6-8 injections per day.

Prevention of hyperlactacidemic coma consists in the prevention of hypoxia and in an individual approach to the appointment of biguanides to patients with diabetes.

Diabetes mellitus and pregnancy

... Pregnancy for the purpose of bearing and giving birth to a child is permissible only against the background of compensation for diabetes mellitus,the criteria of which should be indicators of the glycemic profile in the range from 5.0 to 9.0 mmol / l during the day, the absence of acetone in the urine, the normalization of the proteinogram and blood lipids, the content of glycosylated hemoglobin is not more than 6.0-8.0%. Pregnancy must be planned, since a successful outcome can only be after the mother's body is properly prepared for conceiving a child.

When pregnancy occurs, hospitals are neededplacement in the endocrinology department in order to:

1) transfer to the technique of 4-time insulin injections and revision of the daily dose; 2) establishing the functional state of the kidneys and liver;

3) examination by a neurologist and gynecologist.

After discharge, a visit to the clinic is shown every 2 weeks in the first half of pregnancy, and then weekly.

Parameters monitored on an outpatient basis:

1) fasting blood glucose and 2 hours after meals, daily;
2) urine analysis for sugar and acetone, once a week;
3) urine analysis for albuminuria and sediment microscopy, once every 2-3 weeks;
4) ophthalmoscopy, at least 3 times during pregnancy;
5) blood pressure, daily;
6) body weight, once every 7-10 days.

During pregnancy, the following are possible:

1) urinary tract infections;
2) hypoglycemia;
3) acetonuria;
4) the progression of nephropathy;
5) progression of retinopathy;
6) gynecological complications.

In all these cases, immediate hospitalization is required in the endocrinology department or in the department of pregnancy pathology.

In the case of a successful pregnancy, re-hospitalization is indicated at the twentieth week of pregnancy.

Research on readmission:

1) body weight 2 times a month
2) blood pressure, daily;
3) blood glucose 4-6 times a day, daily
4) glycosuric profile 6 servings daily;
5) urine acetone, daily;
6) estradiol, blood prolactin, once a week;
7) obstetric examination, at least 2 times a week.

Insulin requirements during pregnancy. In the first trimester, there is a tendency to hypoglycemia, so the daily dose of insulin is reduced by 4-6 units. In the second trimester, the insulin dose is increased due to the tendency to ketosis due to placental functiontary hormones and increased lipolysis. Starting from the 35th to 36th week, the need for insulin decreases due to fetal hyperinsulinemia, more stable glycemic parameters are observed during the day.

It must be remembered that all pregnant women with diabetes mellitus can count on a successful pregnancy outcome only when treated with adequate doses of insulin.

With type 2 diabetes for the entire period of pregnancy, sugar-reducing tablets should be canceled and insulin therapy should be prescribed. When diabetes is diagnosed for the first time during pregnancy, insulin therapy is also necessary.

For pregnant women with a violation of carbohydrate tolerance, in addition to a diet with the exception of refined, easily digestible carbohydrates, it is advisable to administer small doses of insulin (4-6 U) before the main meals (breakfast, lunch, dinner).

Insulin therapy during pregnancy...

Regardless of the type of diabetes mellitus, the best option is the appointment of short-acting insulin before breakfast, lunch and dinner, and at 21-22 hours - the introduction of medium-duration insulin to stabilize glycemia at night. The dose distribution takes into account the need for insulin during the daytime (two-thirds of the daily dose) and at night (the remaining one-third). The ratio of short-acting and prolonged-acting insulin in a daily dose varies from 3: 1 to 4: 1, depending on fluctuations in glycemia during the day. Optimal values range from 4.5-5.0 to 8.5-9.0 mmol / l during the entire period of pregnancy.

Timing and method of delivery...

Admission to the maternity hospital is desirable no later than 1-2 weeks before the expected date of birth of the child. In case of complications of pregnancy, earlier hospitalization is indicated. The time and method of delivery is determined by the gynecologist.

On the day of childbirth, 8 units of short-acting insulin are injected in the morning and breakfast is given with a sufficient carbohydrate content (rice or semolina porridge, fruit juice, bread). During childbirth, a 5% glucose solution with insulin is injected intravenously, maintaining the glycemic level within 6-8 mmol / l. Glycemia is monitored every hour.

Indications for drug delivery:

1. Late toxicosis of pregnancy, difficult to treat.
2. Increasing polyhydramnios.
3. Frequent hypoglycemia in a pregnant woman.
4. Discharge of amniotic fluid.

Indications for a planned cesarean section:

1. Transverse and oblique position of the fetus.
2. Breech presentation of the fetus.
3. Narrow pelvis.
4. Large fruit.
5. The first childbirth in a woman over 30-35 years old.

Indications for emergency delivery by cesarean section:

1. Progression of diabetic retinopathy with repeated fresh hemorrhages.
1. Increased renal failure.
2. Violation of the fetus.

2. Bleeding due to previa or placental abruption.

Newborn period...

The first day after During childbirth, approximately 1/2 - 2/3 of the dose of insulin that the patient received before pregnancy is administered. During the stay in the maternity hospital, the dose is adjusted to the required level.

In the case of a safe condition of the newborn and the baby's mother is delivered for breastfeeding after 24 hours. The mother must eat before feeding to avoid the risk of hypoglycemia.

Newborn care…

In children born to mothers with diabetes mellitus, asphyxia occurs 3 times more often and is more severe, and mortality 10 times higher than that of newborns from healthy mothers.

As a result of atelectasis and hyalinosis of the membranes of the lungs, respiratory disorders are frequent, to which cardiac and vascular disorders are added.

In cases of asphyxiation, the following is urgent: suction of mucus from the respiratory tract, artificial ventilation of the lungs, sometimes in combination with hyperbaric oxygenation.

To prevent the development of atelectasis of the lungs and respiratory disorders, it is necessary to inject intramuscularly hydrocortisone at the rate of 5 mg per 1 kg of body weight (in order to form a surfactant) 2 times a day during the first 4-5 days of life. In addition, glucocorticoids prevent the development of hypoglycemia.

With the threat of intracranial hemorrhage, it is necessary to administer a 1% solution of vicasol at the rate of 0.2 ml per 1 kg of body weight (the first 3 days of life). If the child's blood sugar is low (1.6 mmol / l), then intravenous glucose is injected at the rate of 1 g of dry matter per 1 kg of body weight, first in the form of 20%, and then in the form of 10%solution until blood sugar rises to 2.5 mmol / l. With a glycemia of 2.0 mmol / l and above, glucose is administered by mouth in the form of a 10% solution, 1-2 teaspoons every hour. To prevent cerebral edema, lasix is injected, 0.1 ml per 1 kg of body weight in combination with 5 ml of 5% potassium acetate solution. If an intrauterine infection is suspected, broad-spectrum antibiotics are indicated.

From the first day of life, the child receives an aqueous 10% glucose solution every 2-3 hours and breast milk at the rate of 60 ml per 1 kg of body weight (the first 48 hours - after 2 hours, from the third day - after 3 hours).

After being discharged from the maternity hospital, children must findlive under the systematic supervision of a pediatrician, as well. for children born in asphyxia, supervision and a neuropathologist is necessary.

In the system of organizing medical and preventive care for women with diabetes mellitus and their children, 4 stages can be distinguished:

I stage - training in self-control and effective clinical examination of women of childbearing age with diabetes mellitus in order to prevent complications and achieve full clinical and metabolic compensation of the disease. Pregnancy must be planned. The condition for the successful course and outcome of pregnancy is long-term (at least 1 year) compensation for diabetes before pregnancy.

II stage - careful monitoring of the course of diabetes and pregnancy. Timely hospitalization for correction of insulin dose, transfer to 4-fold insulin administration. Prescribing only highly purified drugs. Prevention of hypoglycemic conditions and ketoacidosis.

III stage - timely determination of the timing and method of delivery. Stabilization of

glycemic levels during childbirth, appropriate care of the newborn.

stage - dispensary observation of children of women with diabetes mellitus in order to prevent the disease in a child.