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Faculty: medical №1
Department of propaedeutics of internal diseases and therapy

APPROVED BY.

Vice-rector for scientific and pedagogical work

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METHODOLOGICAL DEVELOPMENT
FOR A LECTURE ON THE DISCIPLINE

Faculty, course: dentistry, 3
Discipline: Endocrinology

Approved:
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Lecture 1 "Diabetes. Etiology, pathogenesis, classification, diagnosis, complications. The role of the dentist in prevention and early diagnosis. Changes in the dental and jaw system and manifestations on the mucous membranes. Principles of treatment"

Actuality of theme.

Diabetes is a major part of the structure of endocrinological pathology. Suffice it to say that the prevalence of diabetes mellitus is 1.5-4% among the population, and with age, in people over 60 years old, up to 7-8%. Such prevalence, chronic course of this disease, possibilitylife is dangerous acute complications (diabetic coma), the presence of such complications as angiopathy, neuropathy, whichlead todeterioration the quality of life and early disability of patients determine the relevance of the subject of the lesson. Early and correct diagnosis of diabetes is the key to timely treatment, which will slow down the progression of the disease and prevent complications. The student's knowledge of the main clinical manifestations of diabetes, the formation of skills and abilities in diagnostics will serve as the foundation for future successful medical activity.

The purpose of the lecture (goals):

To acquaint the student with the tasks of endocrinology as a scientific discipline:

1. Recognition of endocrine diseasessystems (diagnosis).
2. Study of etiology and pathogenesis of diseases of endocrine organs.
3. Development of issues of prevention and treatmentendocrine diseases bodies

Determine the main tasks for students at the department of propaedeutics of internal diseases, which are:

1) method of clinical examination of patients;

2) symptomatology of diseases;

3) basics of laboratory and instrumental diagnostic studies for diseases of internal organs;

4) when familiarizing students with the main nosological units (diseases) and syndromes, teach the ability to use, for example, the data obtained during the examination of a patient for the diagnosis of specific diseases.

Basic concepts:diabetes, glucose, insulin, insulin receptor, insulin resistance.

Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of lecture material (lecture text)

Diabetes was known in ancient Egypt. Described by Celsus (30 BC - 50 AD), Galen (129 - 201 AD), Avicenna (980 - 1037 AD). The disease was called "diabetes mellitus" by Thomas Willis (1674) after the taste of urine. Langerhans in 1869 described islets in the pancreas, and Sobolev in 1900 proved their connection with diabetes. Bunting and Best isolated insulin in 1921, and Sanger established its structure in 1955. The synthesis of insulin was carried out in 1964 by Katsoyannis (USA).

Diabetes mellitus (DM) is defined by WHO experts as a state of chronic hyperglycemia caused by impaired insulin production or action. This is a chronic endocrine-metabolic disease caused by an absolute (impairment of formation) or relative (impairment of action) insulin deficiency as a result of the action of various endogenous (genetic) and exogenous factors, which is accompanied by a violation of all types of metabolism, primarily carbohydrate with its most characteristic manifestation - hyperglycemia, damage to various organs and tissues, primarily blood vessels and nerves.

EPIDEMIOLOGY OF DIABETES

The prevalence of DM in individual countries varies: in Japan, China – 0.7–0.9%, in Western European countries 3–8%, among some American Indian tribes 15–20%. Today, there are more than 170 million people with diabetes in the world.

At the end of 2006, there were about 1 million people with diabetes in Ukraine.

As of January 1, 2000, 902,700 patients with diabetes mellitus were registered in Ukraine, of which 118,100 patients suffered from type I diabetes mellitus, and 784,600 patients suffered from type II diabetes mellitus.

The costs of treating patients are significant. Thus, in the USA (1992), the losses from diabetes amounted to 102 billion dollars (direct costs for treatment - 45 billion dollars, and indirect costs, as a result of disability, etc. - 47 billion dollars). DM is the leading cause of blindness, 50% of all amputations (without trauma). Patients with diabetes are 2-3 times more likely to be diagnosed with CHD, and mortality from CHD is 2-5 times higher, compared to the general population. There is a constant increase in the number of patients with diabetes in the world, which increases by 1.5-2 times in 10 years.

Compared with other diseases, DM is characterized by the earliest disability. Thus, 60-85% of associations of the blind are formed by patients with diabetes mellitus. Approximately 50% of patients with type 1 diabetes die from CKD. Among such

patients, myocardial infarction is diagnosed 3-5 times more often than in the population of the same age, and stroke is 2-3 times more often. Gangrene of the lower extremities occurs 200 times more often, 50-70% of amputations of the lower extremities not related to trauma occur in

The reasons causing the increase in the incidence of diabetes are as follows:

· an increase in the population structure of persons with a hereditary predisposition to diabetes;

· an increase in the average life expectancy of people with an increase in the percentage of elderly people who are more likely to have diabetes;

· intensification of life;

· deterioration of the environmental and social situation, especially in developing countries;

· life-sustaining treatment for diabetic patients;

· the nature of the diet of the population, which, in combination with hypodynamism, leads to an increase in the number of obese people;

· increase in the frequency of chronic cardiovascular diseases (hypertensive disease, atherosclerosis), which are also risk factors;

· improved disease detection.

Risk factors for diabetes

Persons with risk factors for the development of diabetes include:

1. – elderly and senile persons;

2. – identical twins, one of whom has diabetes;

3. – persons in whom both or one of the parents suffers from diabetes and there are patients with diabetes in the pedigree of the other parent;

4. – women who gave birth to a live child weighing 4.5 kg or more;

5. – mothers of children with developmental disabilities;

6. women with glycosuria during pregnancy, as well as after miscarriage or stillbirth;

7. – persons suffering from obesity, atherosclerosis, hypertension, hyperuricemia, gout;

8. – patients with manifestations of metabolic syndrome (insulin resistance, hyperinsulinemia, dyslipidemia, arterial hypertension, hyperuricemia, microalbuminuria, increased aggregation ability of platelets, central or android obesity);

9. patients with diseases of the liver and biliary tract, pancreas, chronic infections of the urinary tract, chronic lesions of the kidneys, respiratory organs;

10. Individuals with renal and alimentary glycosuria, episodic glycosuria and hyperglycemia, manifested in stressful situations;

11. patients with persistent periodontitis and furunculosis;

12. patients with neuropathies of unclear etiology;

13. Patients with spontaneous hypoglycemia.

In its development, diabetes usually goes through 3 stages, which are more clearly distinguished in patients with type 2 diabetes.

1. Prediabetes is a condition (period of the patient's life) that precedes the disease. Laboratory indicators do not go beyond the norm. Risk factors for diabetes mellitus are often observed in patients:

presence of diabetes in relatives (if both relatives have diabetes, the risk of developing it in children increases to 50%)

· obesity (the risk increases with increasing degree of obesity)

· hyper- and dyslipoproteinemia

birth of a child with a large (over 4 kg) body weight - both for the child and for the mother (the risk increases with increasing weight)

· in women, glucosuria during pregnancy; miscarriages and stillbirths

· viral diseases

acute and chronic stress

atherosclerosis, hypertension

· acute and chronic intoxications

acute and chronic diseases of the liver and pancreas

· iatrogenic factors (thiazide diuretics, glucocorticoids, etc.)

2. Impaired glucose tolerance (latent diabetes) is a condition in which there is no clinical picture of diabetes, fasting normoglycemia, and aglucosuria. Violations are detected during STTG. During this period, patients may show the so-called "small symptoms" of diabetes:

· chronic gingivitis, stomatitis, periodontitis, tooth loss

· chronic pustular skin diseases, furunculosis

· itching of the skin, genitals

· skin trophic disorders, skin wounds that do not heal for a long time

· unmotivated sexual weakness, violation of the menstrual cycle

polyneuritis

· cataract

3. Overt (manifest) diabetes mellitus – a condition when clinical symptoms become manifest, changes in laboratory parameters, primarily hyperglycemia and glycosuria. Clinical symptoms of diabetes, primarily complaints of patients, are caused by the disease itself, the degree of its compensation and complications.

Patients are observed:

polydipsia, thirst, dry mouth;

· polyuria, frequent urination, leading to dehydration:

· muscle weakness, increased fatigue;

weight loss (type 1 diabetes), weight gain (type 2 diabetes);

· loss of appetite (polyphagia is possible at first);

· pains in the legs, paresthesias, loss of sensitivity;

· itching (local - perineum, hairy part of the head or generalized);

· impaired vision, impaired sexual function;

· chronic pustular or fungal skin lesions

Depending on the clinic and laboratory indicators, there are 3 degrees of severity of diabetes: mild, moderate and severe

Clinical manifestations of diabetes

• Hyperglycemia is a cardinal manifestation of the disease. Diabetes is chronic hyperglycemia.

Glucosuria -

• Polyuria –

• Polydipsia-

Loss of body weight-

• Polyphagia

• Hyperketonemia

• Ketonuria

Type 1 diabetes (insulin-dependent) is a polyetiological disease. The factors that cause its development most often act in a complex way: environmental factors (viruses, toxins, stress) act on a genetically predisposed organism. Several genes are involved in the implementation of the action, and only a certain combination of them leads to a predisposition to diabetes. A proven link between the development of diabetes and HLA - DR3 antigens; DR4; Dw3; Dw4; B8; B18, Bw15; DQB10302; DQA10301, in the presence of which the risk of developing diabetes increases 7-14 times (the greater the number of antigens, the higher the risk of developing diabetes).

According to modern concepts, IZD is considered an autoimmune disease. Against the background of genetic predisposition, various diabetogenic factors of the external environment act on b-cells - viruses (Coxsackie, retroviruses, mumps, hepatitis A), toxins, food factors (excessive amount of nitrates and nitrites in food, etc.), drugs

(cimetidine, glucocorticoids, indomethacin etc.), which lead to the destruction of b-cells. The formed antigens act on the multinuclear phagocytic system, which leads to their inclusion in immune and inflammatory processes and modulation of the immune response through T- and B-lymphocytes (B-lymphocytes produce antibodies, and T-lymphocytes produce growth factors - interleukin IL, g-interferon , tumor necrosis factor). As a result, the production of autoantibodies to b-cells in macrophages increases, which leads to their destruction and continuation of the autoimmune reaction. When type 1 diabetes lasts up to 5–6 years, b-cells in the islets of Langerhans disappear almost completely. The clinic of diabetes is manifested when more than 2/3 of b-cells are destroyed.

Classification of type I diabetes

There are two main subtypes of type I diabetes - autoimmune and idiopathic. With autoimmune diabetes, specific autoantibodies to insulin-producing cells are detected in the blood of patients

The diagnosis of type 1 diabetes is made only with typical clinical symptoms (progressive weight loss, development of ketoacidosis, progressive physical weakness), detection of glycemia.

A diagnosis of diabetes is made with fasting blood glucose >6.1 mmol/L or with glycemia >11 mmol/L in capillary blood at any time of the day.
When performing TSH, the diagnosis of diabetes mellitus is made when the blood glucose level is >11.1 or more mmol/l (after 2 hours from the start of the test

Criteria for compensation of carbohydrate metabolism
with type I diabetes

- Compensation: Glycated hemoglobin 5-7%, fasting glycemia 5-6 mmol/l, postprandial glycemia 7.5-8 mmol/l, glycemia before sleep 6-7 mmol/l.

Subcompensation: Glycated hemoglobin 7.1-7.5%, fasting glycemia 6.1-6.5 mmol/l, postprandial glycemia 8.1-9 mmol/l, bedtime glycemia 7.1-7.5 mmol/l.

- Decompensation: Glycated hemoglobin >7.5%, fasting glycemia >6.5 mmol/l, postprandial glycemia >9 mmol/l, bedtime glycemia >7.5 mmol/l.

Type 2 diabetes is also a polyetiological disease, but its causes and pathogenesis differ from type 1 diabetes

. is a heterogeneous disease, the basis of which is insulin resistance and insufficient function of ß cells. This type of disease is based on genetic predisposition, and the main provoking factor is obesity.
Risk factors are burdened heredity, obesity, hypertension, gestational diabetes, increased triglycerides, decreased HDL cholesterol.

Persons with risk factors for the development of diabetes include:

elderly and senile persons;

– identical twins, one of whom has diabetes;

- persons in whom both or one of the parents suffers from diabetes and there are patients with diabetes in the genealogy of the other parent;

- women who gave birth to a live child weighing 4.5 kg or more;

- mothers of children with developmental disabilities;

- women with glycosuria during pregnancy, as well as after miscarriage or stillbirth;
- persons suffering from obesity, atherosclerosis,

hypertension, hyperuricemia, gout;

- patients with manifestations of metabolic syndrome (insulin resistance, hyperinsulinemia, dyslipidemia, arterial hypertension, hyperuricemia, microalbuminuria, increased aggregation ability of platelets, central or android obesity);

- patients with diseases of the liver and biliary tract, pancreas;
- persons with renal and alimentary glycosuria, episodic glycosuria and hyperglycemia, which manifests itself in stressful situations;
- patients with persistent periodontal disease and furunculosis;
- patients with neuropathies of unclear etiology;
- patients with spontaneous hypoglycemia.

DIABETES CLINIC

Type 2 diabetes is characterized by a slow development of the disease, especially in the elderly, when the clinical picture is erased, diabetes is not noticeable for years and appears accidentally, against the background of already existing diabetic vascular or nerve damage. Complaints caused by diabetes decompensation are not so demonstrative, they can be episodic. Thirst, polyuria intensifies in the evening, after a meal and becomes

pronounced only against the background of pronounced decompensation. However, type 2 diabetes can manifest itself quite acutely, especially when it manifests against the background of infection, intoxication, trauma, etc. It is not uncommon for the development of obvious stages of diabetes, especially type 2, to be preceded by a long period of hidden hypoglycemic states of varying severity caused by hypersecretion of insulin. Clinically, they are manifested by a feeling of hunger, weakness, sweating, tremors, headache, occur after long breaks in food or on the background of physical exertion, are leveled by taking food, especially with carbohydrate content.
Clinical manifestations of type 2 diabetes are often characterized by polymorphic symptoms from the first years, reflecting the presence of angioneuropathy of varying degrees of severity, progression, and localization.
Decompensation of diabetes mellitus, especially pronounced, is characterized by a clinic common to both types of diabetes mellitus, however, in severe type 1 diabetes, it develops faster, has a more severe course, and is more difficult to treat.

An objective, stable indicator of the state of compensation for diabetes is glycosylated (glycated) hemoglobin (or glycohemoglobin, or HbA1c - a test where Hb is hemoglobin, A1c is attached glucose). Hemoglobin and other proteins combine with glucose in a slow, non-enzymatic reaction that depends on the concentration of glucose. The more glucose is in the blood, the more glycosylated hemoglobin accumulates in erythrocytes. The test for the determination of glycosylated hemoglobin reflects the average level of glucose in the blood during the life of erythrocytes for the last 2-3 months, during which the interaction of hemoglobin and glucose takes place. Normally, the content of HbA1c in the blood is 5-7% of the total level of hemoglobin.

Thus, hyperglycemia is a cardinal symptom of type 2 diabetes, develops and progresses due to three main mechanisms:

decrease in insulin secretion due to functional insufficiency of the insular apparatus;
- tissue resistance to insulin and insufficient utilization of glucose;

- compensatory increase in glucose production by the liver.

The European group on insulin-dependent diabetes in 1993 proposed the following biochemical parameters of control (compensation of diabetes).

A mild (I degree) form of the disease is characterized by low levels of glycemia, not exceeding 8 mmol/l on an empty stomach, when there are no large fluctuations in blood sugar during the day, slight daily glucosuria (from traces to 20 g/l). The state of

compensation is supported by dietary therapy. With a mild form of diabetes, angioneuropathy of the preclinical and functional stage can be diagnosed.

With average (II degree) severity of diabetes, fasting blood glucose rises, as a rule, to 14 mmol/l, there are fluctuations in blood glucose during the day, daily glucosuria usually does not exceed 40 g/l, ketosis or ketoacidosis develops episodically. Compensation of diabetes is achieved by following a diet and taking oral hypoglycemic agents or by administering insulin (in the case of secondary sulfamide resistance) in a dose not exceeding 40 units per day. Diabetic angioneuropathy of various localization and functional stage may be detected in these patients.

Severe (III degree) form of diabetes is characterized by high levels of glycemia (fasting more than 14 mmol/l), significant fluctuations in blood sugar during the day, high level of glucosuria (over 40-50 g/l). Patients require constant insulin therapy at a dose of 60 IU or more, they have various diabetic angioneuropathy.
With type 2 diabetes, the development of severe organic lesions of blood vessels with impaired vision, kidney, heart, brain, and lower limbs function makes it possible to consider the disease as severe, regardless of glycemia and glycosuria indicators, the dose and nature of hypoglycemic therapy.

When diabetes is detected for the first time, a conclusion about the severity of the disease can be made only on the basis of dynamic observations against the background of adequate therapy. With a long-term course of type 2 diabetes, especially against the background of the development of severe vascular lesions, the clinical picture is practically no different from the symptoms of type 1 diabetes, patients are forced to be treated with insulin for a number of reasons, despite the original, non-insulin-dependent nature of diabetes. In such patients, in the diagnosis, the original terminology for determining the type of diabetes should be left, that is, diabetes, as before, should be classified as type 2 non-insulin-dependent diabetes with secondary sulfamide resistance.

Indications for a glucose tolerance test

- Temporary violations of carbohydrate metabolism:
- - glycosuria of pregnant women
- - hyperglycemia after a meal up to 9.9 mmol/l
- - reactive hypoglycemia
- 2. Presence of diabetes risk factors:

- - hereditary predisposition
- - overweight
- - pathological pregnancy and childbirth (miscarriage,
- polyhydramnios, stillbirth, toxicosis of pregnant women,

newborn weight > 4.1 kg)

- - damage to peripheral vessels, arterial hypertension
- - chronic infections
- - dermatopathies
- hyperlipidemia, hyperuricemia

retinopathy and neuropathy of unknown cause

Diagnostics

For the diagnosis of diabetes, assessment of the severity and state of compensation of the disease, it is important to determine the level of sugar in the blood, its repeated determinations throughout the day, the study of daily and fractional glucosuria in separate portions, the determination of the content of ketone bodies in urine and blood, the study of the dynamics of the level of glycemia in various forms glucose tolerance test.

Overt diabetes mellitus is diagnosed based on the detection of an increase in blood sugar and the appearance of sugar in the urine. Blood is examined on an empty stomach. Glucosuria is determined in daily urine or daytime, or in a portion of urine collected 2 hours after a meal. Examining only morning urine is not indicative, since glucosuria is usually not detected in urine collected on an empty stomach in mild forms of diabetes. In the case of a slight increase in the level of fasting blood sugar, the diagnosis can only be established if unambiguous results are obtained again, supported by the detection of glucosuria in the daily urine or in its separate portions. To clarify the diagnosis in such cases, determination of glycemia during the day against the background of the patient's food consumption helps. With untreated overt diabetes, the blood sugar level during the day exceeds 10 mmol/l (180 mg%), which is the basis for the appearance of glucosuria, since the renal threshold for glucose permeability is on average 9.5 mmol/l (170-180 mg%) .

If there is a slight increase or normal values of blood sugar in combination with episodic glucosuria during repeated examinations, or there are any other doubts about the diagnosis, a glucose tolerance test (TSG) is used.Even an incidental, one-time finding of hyperglycemia or glycosuria requires careful investigation to rule out or confirm diabetes.

If the subject has an infectious disease or fever, the test cannot be performed. TSH indicators are affected by the following factors: prolonged bed rest, diseases of the digestive tract with impaired glucose absorption, malignant diseases, taking certain drugs (adrenaline, glucocorticoids, caffeine, diuretin, morphine, thiazide diuretics, diphenine, psychotropic drugs and antidepressants). They should be taken into account during the diagnosis.

According to the report of the WHO Expert Committee "Diabetes" (1999), the criteria for diagnosing diabetes are: fasting capillary blood glucose > 6.1 mmol/l (in blood plasma > 7.0 mmol/l); 2 hours after glucose load in capillary blood > 11.1 mmol/l (in blood plasma > 11.1 mmol/l); glucose concentration in a randomly taken blood sample > 11.1 mmol/l in combination with characteristic clinical symptoms (polydipsia, polyuria, weight loss).

Criteria for diabetes mellitus and impaired glucose tolerance (WHO Expert Committee on Diabetes Mellitus)

An auxiliary diagnostic criterion for verifying the diagnosis of diabetes is the determination of glucose in urine collected after a glucose load.
In such patients, it is advisable to examine blood glucose levels after a meal, only with their normal fluctuations (7.7-8.0 mmol/l) to conduct the test. If one of the indicators of the 2-hour simplified TSH is elevated, it is recommended to conduct a classic TSH after 1 month under the condition of normal nutrition. At least 3 days before the test, the subject should receive 150-200 g of carbohydrates per day, including easily digestible ones.

SKIN, MUSCLE AND BONE DAMAGE IN PATIENTS WITH DIABETES.

Skin lesions in patients with diabetes mellitus are diverse and are united under the single term "diabetic dermopathy": dry skin, rubeosis on the face, yellowness of the palms and soles (due to impaired carotene metabolism), hyperkeratosis (primarily on the feet), thickening of the nails. Often pustular diseases that are poorly treated. Xanthomatosis - yellowish fat nodules on the palms and feet, the back of the elbow joints.

Lipoid necrobiosis is characteristic - painful red-purple nodes, smooth to the touch with a waxy sheen and clear edges, more often on the lower legs. Merging, they can form rings with a diameter of up to 10 cm. An ulcer can form in the center of the focus. It is believed that this is a kind of metabolic disorder in the skin with deposition of glycogen and lipids against the background of microangiopathy of the skin.

Damage to the bone system is a consequence of metabolic disorders and is observed in the majority of patients with a long course. Systemic osteoporosis, osteoarthritis, osteoarthropathies are characteristic. There is a gradual destruction of the bones. Pathological dislocations and deformations are possible, mainly in the feet. Patients complain of pain in the lower back, in the bones, often pain when percussing the bones. Radiologically, osteoporosis, osteosclerosis, gradual destruction of the bone structure and their reconstruction are observed. Schmorghl's hernia and Dupuytren's contracture occur 2-3 times more often in patients with diabetes.

INFRINGEMENT OF INTERNAL ORGANS (GASTROINTESTINAL TRACT, CARDIOVASCULAR SYSTEM, LUNGS, URINARY SYSTEM) AND FEATURES OF THEIR COURSE IN PATIENTS WITH DIABETES

1. Damage to the digestive organs in diabetes is often observed. Patients have tooth loss, gingivitis, stomatitis. The acid-forming and secretory functions of the stomach are reduced (as a result of hyperglycemia, lack of insulin, microangiopathy of the stomach, etc.). Chronic gastritis, gastroduodenitis, and possible gastroparesis with impaired evacuation are observed in 60% of patients with a disease duration of more than 4 years. Chronic diseases (ulcer disease, cholecystitis, appendicitis, etc.) in patients with diabetes mellitus often have a mild course of symptoms.

Damage to the cardiovascular system is the most frequent in patients with diabetes and in the long course reaches 100%. Atherosclerosis in patients with diabetes mellitus is observed much more often than among the general population and has its own characteristics:

· generalized character;

· early onset (at a young age in patients with type 1 diabetes);

· heavy course;

equally often in patients of both sexes.

Atherosclerotic lesions of coronary vessels, brain vessels and lower extremities are most often observed. Atherosclerosis is the main reason for the development of myocardial infarction, cerebral strokes and gangrene of the lower extremities in patients with diabetes. These lesions are the most common cause of death.

In patients with diabetes mellitus, acute myocardial infarction (MI) develops 3–5 times more often and its course, compared to persons without diabetes mellitus, has its own characteristics:

MI clinic is asymptomatic, often painless forms or with atypical pain syndrome;

· often atypical variants (abdominal, asthmatic, arrhythmic, colaptoid, etc.);

· frequent MI is transmural, large-focal, recurrent (repeated MI is 1.5–2 times more likely);

· the back wall of the myocardium is more often affected;

· the course of MI is more severe and the prognosis is worse (mortality is twice as high);

· inconsistency between clinic and ECG: ECG – dynamics are slowed down, negative "T" is formed late;

· complications often occur: shock, aneurysm, arrhythmias, pulmonary edema, etc.

Features of the course of coronary heart disease (angina) in patients with diabetes mellitus:

- CHD is equally common in men and women
- seizures are not intense, atypical (its manifestation may be weakness, sweating, palpitations, shortness of breath, which are relieved by nitroglycerin);
- irradiation is inconsistent, atypical;
- seizures often occur with hypoglycemia, at rest.

CHD in patients with diabetes should be differentiated from diabetic cardiomyopathy, which is characterized by:

- occurs more often at a young age;
- mainly in patients with diabetes type 1 with its duration of more than 5 years

- often in persons without increased blood pressure and obesity
- pain in the heart according to the type of cardialgia, not angina pectoris, often without pain;
- pronounced microangiopathies;
- more often with a decompensated, labile course;
- CNS is more often of the right ventricular type;
- existing diabetic autonomic neuropathy;
- ECG: the voltage is reduced, T±, a test with dosed exercise without characteristic changes (in case of coronary heart disease, a decrease in the S–T segment, inversion and discordance of T), a decrease in the coronary reserve, a positive test with K, a negative test with obsidan)

Features of the course of bronchopulmonary pathology in patients with diabetes:

1. Chronic processes often occur, pneumonias have a propensity for a protracted course, the development of abscesses and gangrene. The course is often atypical, with few symptoms.

2. Tuberculosis of the lungs:

a) more often in young people;

b) localization of the process is atypical - in the center or at the gates of the lungs, often bilateral damage;

c) the course is mildly symptomatic with the formation of "silent" caverns;

d) the process progresses rapidly, often the development of exudative forms (up to 50%).

Damage to the urinary and reproductive systems.

`1. Infection of the urinary tract - acute and chronic pyelonephritis, suppuration of the kidneys (carbuncle, abscess, apostematosis), which can lead to bacteremic shock. A feature of their course is mild symptoms, often latent forms, without a temperature reaction with severe intoxication. The course of diabetes mellitus becomes more severe, the infection contributes to decompensation of diabetes mellitus, ketoacidosis.

2. Diabetic nephropathy.

3. Diabetic cystopathy - the basis is visceral neuropathy, which can lead to bladder paresis. The urge to urinate is rare, the bladder is full (up to 500-1000 ml).

4. The function of the genital organs is disturbed. Men often have a decrease in libido, potency; in women - infertility, miscarriages, stillbirths, amenorrhea, vulvitis, vaginitis.

Diabetic angiopathy is a generalized lesion of blood vessels in patients with diabetes mellitus. Damage to small vessels (arterioles, capillaries, venules) is called microangiopathy and has a specific character, damage to large vessels is called macroangiopathy and is considered early and widespread atherosclerosis.

1. Diabetic nephropathy

Diabetic nephropathy is one of the main causes of disability and mortality in patients with diabetes mellitus: up to 50% of patients with diabetes mellitus type 1 die before the age of 50 due to nephropathy. Nephropathy occurs more often in patients with type 1 diabetes (40-50% versus 20-30% in patients with type 2 diabetes). Histologically, nodular (intercapillary) glomerulosclerosis is pathognomonic for nephropathy.

Proteinuria is an early clinical sign. which indicates deep damage to the kidneys, which is irreversible. The main preclinical criterion of nephropathy is microalbuminuria (30-300 mg/day), which is determined by the radioimmunoassay method or with the help of special strips.

Diabetic retinopathy (DR) is the main cause of blindness, which occurs 25 times more often in patients with diabetes compared to the general population, and visual disability occurs in more than 10% of patients. DR develops in patients with both types of diabetes.

Diabetic foot is one of the manifestations of damage to the lower extremities in patients with diabetes. Diabetic foot is a complex complex of anatomical and functional changes caused by micro- and macroangiopathies and neuropathy in the long course of diabetes. It is based on neuropathy, ischemia and infection. It occurs in 10-20% of patients. There are 3 clinical forms of diabetic foot syndrome: neuropathic, ischemic and mixed.

Gestational diabetes includes only diabetes detected during pregnancy (about 2% of pregnant women). In general, the manifestations of gestational diabetes disappear after childbirth and carbohydrate tolerance normalizes. However, after 5-15 years, 20-40% of women with gestational diabetes develop clinical diabetes. Pregnancy with gestational diabetes has an increased risk of perinatal mortality and fetopathy.

Xerostomia (dry mouth) is one of the early symptoms of diabetes, the development of which is a consequence of dehydration. Constant thirst and increased appetite are often noted. CO becomes poorly hydrated or dry, cloudy, with a significant accumulation of plaque, often hyperemic.

Catarrhal stomatitis, glossitis are caused by infection, easy vulnerability, a sharp decrease in the barrier function of CO and unsatisfactory cleaning (Fig. 8). This is facilitated by a decrease in salivation. In places of minor mechanical trauma, CO damage is observed in the form of hemorrhages, sometimes erosions.

Fungal stomatitis, mycotic stomatitis develop as a result of dysbacteriosis against the background of a sharp decrease in the body's resistance, a decrease in the content of many enzymes in saliva, especially lysozyme. The development of these lesions is facilitated by a violation of the acid-base state, which is caused by an increase in the number of underoxidized metabolic products (pyruvic and lactic acids)

Fungal lesions of various parts of the oral cavity are permanent. Mycotic infection is especially common, and cracks covered with whitish-gray crusts appear in the corners of the mouth.

Paresthesia CO occurs along with its dryness. The burning sensation of CO is combined with itching of the skin in the genital area and in other areas. Damage to the nervous system is manifested by neuritis and neuralgia of the branches of the trigeminal nerve. Taste sensitivity to sweet, salty, sour may decrease. This disorder has a functional nature, and therefore, after the treatment, taste sensitivity is normalized.

Trophic disorders of COPD are characterized by the appearance of trophic ulcers, which are characterized by a long course with slow regeneration. A decrease in the regenerative properties of CO is due to a violation of oxidation-reduction processes.

The described changes in CO do not have specific features that are unique to diabetes. Therefore, the history and general examination of the patient, including laboratory tests, are of great importance in diagnosis. With diabetes, there is an increase in the content of glucose in the blood, its appearance in the urine.

DIAGNOSIS IN DIABETES

Examination procedure	Identified symptoms	Pathogenetic justification of symptoms
Complaints	Dry mouth, increased thirst. Pain when eating, especially when eating hot solid food. Paresthesias of the mucous membrane, burning sensation, decreased taste sensitivity to sweet, salty. Burning and itching of the genitals and other parts of the body. Neuralgic and neuritic pain along the course of the branches of the trigeminal nerve. Bleeding gums when brushing teeth	Violation of carbohydrate metabolism with subsequent violation of protein and fat metabolism, dehydration of tissues. The mucous membrane of the oral cavity is thinned, highly vulnerable, inflamed due to a decrease in the barrier function. Disorders of the nervous system as a result of impaired carbohydrate metabolism. Catarrhal gingivitis due to a decrease in the barrier function of the mucous membrane and unsatisfactory oral hygiene
Anamnesis	Diabetes mellitus of different severity and duration	
Review		

Examination of the mucosa **lining of the oral cavity**	The mucous membrane of the oral cavity is slightly moist or dry, shiny, slightly hyperemic. Erosions, hemorrhages, trophic ulcers are possible	The consequence of tissue dehydration in diabetes, dryness and thinning of the epithelium
Examination of the salivary glands	There are no organic changes. Palpation of the salivary glands is painless. Excretory ducts without pathological changes, the secretion is clean, but in insufficient quantity.	Dryness in the oral cavity is not associated with damage to the parenchyma of the salivary glands, but with tissue dehydration.
Examination of dental rows	The hygienic condition is unsatisfactory, dental deposits, high KPU index	Patients spare their gums and brush their teeth poorly. Formation of dental plaque and increased destruction of teeth as a result of impaired carbohydrate metabolism and hyposalivation (xerostomia)
Examination of periodontal tissues	Catarrhal inflammation of periodontal tissues of varying severity	The result of unsatisfactory hygiene, impaired carbohydrate metabolism and lysis of bone tissue
Additional examination methods		

Blood and urine tests	Laboratory studies of blood and urine for glucose content.	An increase in the level of glucose in the blood (the norm is 3.9-8.6 mol / l) and the appearance of glucose in the urine
Bacterioscopic examination of plaque on the tongue.	In most cases, the fungus Candida is detected.	Dysbacteriosis of the oral cavity and acidification (decrease in pH) of tissues with impaired carbohydrate metabolism.
X-ray examination of alveolar processes and teeth	Pathology of periodontal bone tissue of various degrees	The result of osteolysis processes

Materials on the activation of students of higher education during the lecture: questions, situational tasks, etc

1. A 50-year-old diabetic patient, after the occurrence of furunculosis of the skin and the appointment of antibiotics, discontinued glibenclamide. The patient's condition worsened, increased thirst, dryness, diuresis - 4.5 l/day, fainted. Objectively: The skin is dry. Breathing is superficial, accelerated. RS - 100/min, BP - 90/40 mm Hg. Tones of the heart are deaf. The stomach is soft. Liver - +5 cm. Glycemia 43 mmol/l, reaction to acetone in urine is negative, glucose - positive. Determine the nature of the condition. A Hypersmolar coma B Ketoacidotic coma C Infectious-toxic shock D Diabetic ketoacidosis E Lactoacidotic coma 3 Patient K., 42 years old. He is 162 cm tall, weighs 87 kg, BMI = 33 kg/m2. The general condition is satisfactory. Heart sounds are dull, no murmurs are heard during auscultation. Heart rate - 72/min. In the lungs, breathing is vesicular. The lower edge of the liver protrudes 1.5-2 cm below the edge of the right hypochondrium. Swelling is not observed. I consulted an endocrinologist to determine obesity, its degree, and treatment methods. Blood pressure: on the right - 140/90 mm Hg, on the left - 145/85 mm Hg. Determine the type of obesity and its degree?
A *Alimentary and constitutional obesity, 1 st.
B Alimentary and constitutional obesity, 2nd art.
C Alimentary and constitutional obesity, 3rd century.
D Hypothalamic obesity 2 st.

E Overweight

General material and mass-methodological support
lectures:

1. work program of the academic discipline
2. synopsis (plan-summary) of the lecture
3. multimedia presentation of the lecture

Questions for self-control:

1. Define the concept of diabetes.
2. Complaints of the patient about diabetes.
3. Specify the main etiological factors, features of pathogenesis.
4. Modern classification of diabetes.
5. The main clinical signs classification of diabetes.
6. Laboratory and instrumental examination of patients, interpretation of the obtained results.
7. Basic principles of treatment.
1. Define type 2 diabetes.
2. Complaints of the patient about type 2 diabetes.
3. Specify the main etiological factors, features of pathogenesis.
4. Modern classification of type 2 diabetes.
5. The main clinical signs and classification of type 2 diabetes.
6. Laboratory and instrumental examination of patients, interpretation of the obtained results.
7. Basic principles of treatment.
8. main syndromes in macro- and microangiopathies.
9. Chronic complications of diabetes.
10. Emergency conditions of diabetes.

References:
Basic literature:

1. Internal diseases: study guide / O. O. Yakymenko, V. V. Klochko, O. E. Kravchuk and others. ; under the editorship Prof. O. O. Yakymenko. — 2nd ed., corrected. and additional — Odesa: ONMedU, 2023. — 436 p.
2. Endocrinology in dental practice: training. manual [for students of higher education. institutions of the Ministry of Health of Ukraine] / edited by L. E. Bobireva, A. K. Nikolishina; L.E. Bobiryova, V.M. Bobiryov, L.P. Gordienko [and others] ;

Ministry of Health of Ukraine, UMSA. – Poltava: Publisher S. V. Govorov, 2021. – 176 p.
3. Perederii V.G., Tkach S.M. Basics of internal medicine in 3 volumes. Manual. Vinnitsa. New book. 2018. - 784 p.
4. Davidson's Principles and Practice of Medicine 23rd Edition. Editors: Stuart Ralston, Ian Penman, Mark Strachan Richard Hobson. Elsevier. - 2018. – 1440 p.
5. Endocrinology: textbook /Ed. by prof. Peter M. Bodnar.- 4th ed. updated – Vinnitsa: Nova Knyha, 2017. – 328 pages.

Additional:

1. Endocrinology: a textbook for university students. honey. education closing IV level of accreditation / [P. M. Bodnar, G. P. Mikhalchyshyn, Yu. I. Komisarenko and others] ; under the editorship P. M. Bodnar; National Acad. honey. of Sciences of Ukraine. - 4th ed., updated and supplemented. - Vinnytsia: New book, 2017. - 500 p. : fig. 2.
2. Standards of providing medical assistance to patients with pathological conditions of the thyroid and parathyroid glands under the influence of negative environmental factors (third edition, expanded) / Ed. O.V. Kaminsky - Kharkiv: "Juright", 2017. - 312 p.
3. Order of the Ministry of Health of Ukraine dated December 29, 2014 No. 1021 "Unified clinical protocol of primary, emergency, secondary (specialized) and tertiary (highly specialized) medical care "Type 1 diabetes in young people and adults."
4. Macleod's Clinical Examination / Ed. G.Douglas, F.Nicol, C.Robertson.– 13th ed.– Elsevier. 2013. – 471 p.
5. Bates' Guide to Physical Examination and History Taking /Ed. Lynn S. Bickley, Peter G. Szilagyi. – Wolters Kluwer, 2017. – 1066 p.
6. Harrison's Endocrinology. Ed. by J. Larry Jameson, Mc Graw – Hill., New York, Chicago, Toronto. e.a. 4th edition, 2016. - 608 p.

Electronic information resources

1. http://moz.gov.ua - Ministry of Health of Ukraine
2. www.ama-assn.org – American Medical Association /American Medical Association
3. www.who.int - World Health Organization
4. www.dec.gov.ua/mtd/home/ - State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk – British Medical Association

6. www.gmc-uk.org - *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association
8. https://onmedu.edu.ua/ - Odesa National Medical University

Lecture No. 2 "Diseases of the thyroid and parathyroid glands. Etiology, pathogenesis, clinic, diagnosis, complications, principles of treatment. The role of the dentist in prevention and early diagnosis"

Actuality of theme.
Most endocrine diseases are chronic lifelong diseases. Early diagnosis of endocrine diseases can not only be a guarantee of timely and effective treatment, but also prevent the development of manifest forms of the disease, or the transition from milder to severe forms. He who diagnoses well treats well - this classic form is most relevant for endocrine diseases. The ability to evaluate clinical manifestations, data from additional studies, identify risk factors, the most important aspects of medical activity in terms of diagnosing endocrine pathology. This requires good theoretical training, professional skills and abilities from the doctor.

The purpose of the lecture (goals):
To acquaint the student with the tasks of endocrinology as a scientific discipline:

1. Recognition of diseases of the endocrine system (diagnosis).
2. Study of etiology and pathogenesis of diseases of endocrine organs.
3. Development of issues of prevention and treatment of diseases of endocrine organs.

Determine the main tasks for students at the department of propaedeutics of internal diseases, which are:

1) method of clinical examination of patients;

2) symptomatology of diseases;

3) basics of laboratory and instrumental diagnostic studies for diseases of internal organs;

4) when familiarizing students with the main nosological units (diseases) and syndromes, teach the ability to use, for example, the data obtained during the examination of the patient for the diagnosis of specific diseases.

Basic concepts: thyroid gland, parathyroid gland, hormone, hypothyroidism, hyperthyroidism, goiter.

Plan and organizational structure of the lecture.

1. Determination of the educational goal.
2. Providing positive motivation.
3. Presentation of the lecture material according to the plan:
4. Summary of the lecture. General conclusions.
5. The lecturer's answer to possible questions.
6. Tasks for self-training.

Content of the lecture material

Anatomical and physiological data. The thyroid gland (thyroid gland) is an unpaired endocrine organ (weighing 25-30 g), located on the front surface of the neck, fixed by a fibrous capsule to the trachea, therefore it moves with the latter during swallowing.

The structural and functional unit of the thyroid gland is: a follicle, the cells of which synthesize iodinated hormones - triiodothyronine (T3) and tetraiodothyronine or thyroxine (T4) and the protein thyroglobulin (TG), a third iodinated hormone - reverse T3, which has no significant effect on the body, is described. . Interfollicular C-cells produce a hormone - calcitonin, which, together with parathyroid hormone, has the ability to regulate calcium metabolism.

Thyroglobulin is a component of the colloid of follicles that deposits iodinated hormones. The release of the latter into the blood is regulated by hypothalamic releasing hormone (TRH) and pituitary thyroid-stimulating hormone (TSH). When the function of the thyroid gland decreases, the activity of these hormones increases, and when its function increases, the activity of tropic hormones decreases.

Thyroid activity is also directly influenced by the sympathetic nervous system, somatotropin, corticotropin, as well as calcium ions, prostaglandins, and long-acting thyroid stimulator (TDTS).

In the peripheral blood, mainly thyroxine (T4) circulates, which contains ¾ of all the iodine in the blood, as well as triiodothyronine (T3), which contains only ¼ part of iodine. Physiological activity of T3 is five times higher, its exchange is faster, distribution is wider, so half of the activity of thyroid hormones is due to its content.

The effect of thyroid hormones on tissues is carried out through receptors intracellularly, a large part of receptors is located in the nucleus of cells. Receptors bind triiodothyronine much more intensively than thyroxine.

Thyroid hormones have a complex multifaceted effect on all organs and tissues, on all types of metabolism: they stimulate heat generation, enhance oxidative processes in the body, increase oxygen absorption by tissues, and disconnect oxidative phosphorylation in mitochondria. In physiological doses, thyroid hormones stimulate the synthesis of proteins, and in excessive doses, they increase their dissimilation.

Thyroid hormones affect tissue growth and differentiation. With their deficiency, growth retardation and mental disorders are noted.

Diffuse toxic goiter

Definition. Diffuse toxic goiter (Bazedov's disease) is a disease based on hyperfunction of the thyroid gland, its hyperplasia and hypertrophy, and is primarily characterized by changes in the cardiovascular and nervous systems.

The disease occurs more often among women aged 20-50. The ratio among sick men and women is 1:51:7.

Etiology: mental trauma (80%); neurocirculatory dystonia; infection (flu, sore throat, chronic tonsillitis, measles, whooping cough, rheumatism, SLE); hereditary factor (genetic); influence of physical and hormonal factors – overheating in the sun, pregnancy, menopause, hypothalamus pathology. But in general, the disease is considered as an autoimmune process with delayed type hypersensitivity. This is evidenced by the following facts: the presence of protein thyrostimulator in the blood of patients; increase in the titer of antibodies to thyroglobulin, microsomal fraction; violation of cellular immunity; infiltration of lymphocytes and plasma cells of the thyroid gland and retrobulbar tissue; hyperplasia of the thymus gland; a decrease in the absolute and relative number of T-suppressors and an increase in B-lymphocytes. Leukocyte antigens HLA-B8 are detected much more often.

A thyrostimulating agent is an immunoglobulin (antibody) of class G (IgG), which is formed in B-lymphocytes under the stimulating effect of T-lymphocytes. It was called a long-acting thyroid stimulator (LTTS or LATS). Recently, a TDTS-protector specific only for humans has been found.

Under the influence of TDTS, a factor that acts through TSH receptors of the thyroid gland, hyperproduction of thyroid hormones occurs - the main link in the pathogenesis of diffuse toxic goiter.

Pathogenesis clinical symptoms are due to the effect of thyroid hormones on the nervous, cardiovascular systems, digestive organs, and various types of metabolism.

Exchange violation:

- carbohydrates is manifested by an increase in the absorption of glucose in the intestines, inhibition of the transition of carbohydrates into fats;
- proteins: increasing protein breakdown; the occurrence of a negative nitrogen balance.
- fats: strengthening the mobilization of fat from the depot and weight loss of patients.

In addition, there is a violation of vitamin metabolism and water-salt metabolism, including micro- and macroelements.

Pathomorphology. The altered thyroid gland is made up of hyperplastic follicles, the epithelium of which grows from single-layered to multilayered cylindrical. The connective tissue stroma of the gland is excessively vascularized, infiltrated by lymphoid cells. The electrogram shows significant changes in epithelial cells: an increase in the number of colloid droplets, the size of mitochondria and the Golgi complex, and the spread of tubules of the endoplasmic reticulum.

Parenchymatous organs undergo dystrophic changes, areas of necrosis and sclerosis are found in them. This applies to the heart muscle (myocardiodystrophy, myocardiosclerosis), skeletal muscles and liver (fatty degeneration with toxic hepatitis). The central nervous system, gonads, adrenal cortex also undergo changes.

Classification(WHO, 2001)

- 0 - there is no goitre

§ I - the goiter is palpated, but not visualized.
§ II - the goiter is palpable and available for examination.

According to the degree of severity, toxic goiter is divided into light, medium and severe forms.

The mild form is characterized by neurological symptoms, moderate tachycardia (heart rate about 100 per 1 min.), loss of body weight up to 10%, increase in basic metabolism (up to +30%), total thyroxine up to 190-200 nmol/l and absorption of triiodothyronine (PET3) by erythrocytes) by 10-12%.

The moderate form is characterized by significant emotional and autonomic disorders, tachycardia up to 120 beats per minute, heart failure of the I-IIA stage, loss of body weight up to 20%, increase in basic metabolism up to 40%, total thyroxine up to 245-258 nmol/l, PET3 by 12-13%.

Severe form - heart rate more than 120 per 1 min., arrhythmia, heart failure II-III stage, loss of body weight by 30%, increase of thyroxine to 258-270.9 nmol/l, PET3 more than 14%.

Examples of formulation of the diagnosis.

1. Diffuse toxic goiter of the III degree, medium severity, state of decompensation (compensation). Thyrotoxic heart, NC I-IIA st.

2. Diffuse toxic goiter of the II degree, severe form, state of decompensation

(compensation). Thyrotoxic heart, atrial fibrillation, tachysystolic form of NK IIA-IIV century. Anovulatory cycles.

Clinical picture. Already during a conversation with the patient, attention is drawn to fussiness, body tremors, verbosity, tearfulness, weight deficit, and in severe cases, muscle volume decreases.

The skin is warm, moist, pigmented. Body temperature is elevated. Hair is brittle. The thyroid gland is enlarged to degrees I-III. Eye symptoms occur in 70% of patients. The eye slits are wide open, a characteristic look reminiscent of anger, surprise (Delrymple's eye), exophthalmos up to 25-26 mm (normally 12-14 mm), eye shine, symptoms of Graefe, Kocher, Mobius, Elinek. Eye symptoms appear as a result of a violation of the tone of the eye muscles due to increased activity of the sympathetic-adrenal system, accumulation of glycosamines in the retrobulbar tissue with its subsequent swelling/

Cardiovascular system. The main symptoms in the clinic of diffuse toxic goiter are changes in the cardiovascular system. "One should never forget that a patient with thyrotoxicosis is, first of all, a patient with heart damage, and taking care of his heart is the main task" (N.A. Shereshevsky). Subjective and objective signs of cardiac disorders, which accompany diffuse toxic goiter, are united by the general term "thyrotoxic heart" (dystrophy, hypertrophy, cardiosclerosis, HNK).

Patients are disturbed by the feeling of frequent heartbeat, pain, suffocation. Tachycardia is the most persistent symptom. Pulse — more than 90 in 1 minute. High pulse pressure is characteristic. Tones are loud, strengthening of the II tone over the pulmonary artery, functional systolic murmur at the top of the heart. Atrial fibrillation is often detected - an indication for urgent radical measures. It occurs as a result of increased excitability of the atrial muscle. In the elderly, thyrotoxicosis can be manifested exclusively by attacks of atrial fibrillation, and between them the pulse rate can be normal, which creates difficulties for diagnosis. Sometimes there are angina attacks as a result of the increased demand of the myocardium for oxygen. Ultimately, a "thyrotoxic heart" occurs, which leads to heart failure.

Digestive organs. The frequency of defecation is noted. Sometimes a spasm of the pylorus is observed - an imitation of an ulcer, pancreatitis. Liver function is often impaired. As a result of thyrotoxicosis, there is an increased permeability of hepatocyte membranes with the development of serous hepatitis and liver cirrhosis.

Nervous System. One of the important manifestations of diffuse toxic goiter is a disturbance of the nervous system. Even Graves (1835) was one of the first to describe

this disease under the name "hysteria". Patients are capricious, conflicted, do not get along with others. Their mood often changes, tears easily appear. Marie's positive symptom (hand tremors), as well as the whole body, is a "telegraph pole" symptom. The tremor is constant, it interferes with work, writing, drawing. On the part of the central nervous system — thyrotoxic encephalopathy: headache, photophobia, double vision, sleep disturbances.

Muscular and endocrine systems. In severe forms, thyrotoxic myopathy develops — weakness of the proximal muscles of the limbs ("cell" symptom).

Disorders of the endocrine sphere are also observed - amenorrhea, hypocorticism, decreased potency and libido.

Definition of hormones. The level of thyroid hormones in the blood is increased, and the level of 0.2 mod/l, the level of T3≤thyroid-stimulating hormone of the pituitary gland is reduced (the level of TSH is increased more than 3 nmol/l, T4 - more than 150 nmol/l).

Thyrotoxic crisis is a life-threatening worsening of the condition of a patient with an accident, which develops mainly in persons with a severe form of the disease. Crises develop in the summer. Reasons - thyroidectomy, use of J131 for therapeutic purposes, intensive thyroid palpation, psychotrauma. It develops when the euthyroid state is not reached. With undiagnosed toxic goiter, in the absence of its treatment, thyrotoxic crisis can be provoked by infections, intoxications, surgical interventions, reactions to various medications.

The crisis develops quickly (hours), less often - gradually (days) due to a sharp increase in the tone of the sympatho-adrenal system, the release of a significant amount of T3 and T4 into the blood, and a decrease in the function of the adrenal cortex. Main symptoms: arrhythmia, temperature up to 40°C, sharp tachycardia (heart rate 200 1 or hypotension, increasing cardiovascular insufficiency. Gastrointestinal disorders rapidly increase (nausea, continuous vomiting, profuse diarrhea, abdominal pain), diffuse sweating, which leads to dehydration of the body. The patient's condition is aggravated by mental agitation, hallucinations, impaired consciousness. In some cases, jaundice develops. Blood analysis reveals leukocytosis, increased ESR, pathological liver tests (cytolytic and cholestatic syndromes). Ultrasound reveals hepatosis, hepatitis.

There are 3 variants of thyrotoxic crisis: cerebro-bulbar, cardiovascular, gastrointestinal.

Mortality without treatment of thyrotoxic crisis reaches 70-100%.

Treatment of diffuse toxic goiter involves suppression of thyroid function.

In case of medium and severe forms - inpatient treatment with provision of mental peace, adequate sleep and nutrition. There are three methods of treating diffuse toxic goiter:

1) medication;

2) surgical;

3) radioiodine therapy.

Drug treatment is used as an independent method for complete elimination of the disease or to prepare patients for subtotal thyroid resection or radioiodine therapy.

The drug Mercazolil has thyrostatic properties, which suppresses the activity of cytochrome oxidase and peroxidase, which are involved in the conversion of tyrosines into thyronins - T3 and T4. With a mild form, 15-20 mg per day is prescribed, with an average form - 20-30 mg, with a severe form - 30-40 mg (be careful, leukopenia!).

In preoperative preparation, iodine preparations are prescribed. Lugol's solution 5-10 drops 3 years. per day with gradual dose reduction.

If necessary, prednisolone, β-blockers, calcium antagonists, potassium preparations, vitamins, cardiac glycosides, sedatives, anabolic steroids. It must be remembered that non-steroidal drugs and sulfonamides cannot be prescribed together with Mercazolil (negative effect on the blood).

After subtotal resection of the thyroid gland (effect in 90% of cases), relapses of the disease, post-thyrotoxic encephalopathy, hypertension or tetany are possible.

Radioiodine therapy is prescribed to patients over the age of 50 with a severe course of the disease.

Treatment for thyrotoxic crisis is aimed at:

- a decrease in the level of thyroid hormones in the blood;
- elimination of adrenal insufficiency;
- elimination of cardiovascular disorders;
- elimination of neurovegetative disorders.

1. To reduce the secretion of T3 and T4, 1 ml of 1% Lugol's solution per 1 liter of 5% glucose solution, prepared in an isotonic solution of sodium chloride, is administered intravenously. Lugol's solution can be injected into the stomach through a thin probe or in

a microenema, or 30-40 drops can be given orally 3 times a day with milk (if necessary, the Lugol's dose can be increased to 40-50 drops every 4 hours). Iodine withdrawal is gradual.

2. Mercazolil is prescribed before the introduction of iodine, at first 60-80 mg, and then 40-50 mg every 4 hours.

3. Hydrocortisone 100 mg every 6-8 once a day or prednisolone 200-300 mg per day together with 50 ml of 5% ascorbic acid.

4. At low blood pressure, mezaton 1-2 ml is administered intramuscularly.

5. Beta-adrenoblockers (anaprilin, atenolol) intravenously, and then orally 25-50 mg every 4-8 hours (control of blood pressure, heart rate). Cancellation is gradual!

6. To reduce the activity of the kallikrein-kinin system, protease inhibitors (contrical, trasylol) are administered in an isotonic solution of sodium chloride.

Rehydration - isotonic solution of sodium chloride, glucose, albumin,

reopolyglukin up to 2-3-4 liters per day.

7. Symptomatic and heart remedies (vitamins of group B – B1, B6, B12, ascorbic acid, corglycon, phenobarbital, retabolil).

Hypothyroidism

Hypothyroidism is a syndrome, the development of which is caused by thyroid hypofunction, develops as a result of a decrease in the amount of functional thyroid tissue and is characterized by a reduced content of thyroid hormones and an increase in the level of thyrotropin in blood serum. In 95% of patients hypothyroidism is primary.

Clinical epidemiology. According to M.P.J. Vanderpump et al. (1995), the frequency of spontaneous hypothyroidism during a 20-year follow-up was 3.5 per 1000 female and 0.8 per 1000 male population. The average age of diagnosis is 60 years. It is worth noting that the frequency of hypothyroidism increases significantly in goiter endemic areas.

Etiology. By etiology, hypothyroidism can be acquired or congenital. Congenital hypothyroidism develops due to aplasia or hypoplasia of the thyroid gland. The most common cause of hypothyroidism is iodine deficiency. Thyroiditis, thyroidectomy, treatment with radioiodine, thyroid irradiation, thyrostatic therapy, long-term treatment with excess iodine, violation of the biosynthesis of thyroid hormones are among the

reasons for the development of the syndrome. It is also accepted to distinguish secondary and tertiary (hypothalamic) hypothyroidism.

Pathogenesis. The basis of the pathogenesis of all forms of hypothyroidism is a decrease in the level of thyroid hormones in the blood. The development of secondary hypothyroidism is caused by insufficient production of thyrotropin in the pituitary gland, and tertiary hypothyroidism is caused by a lack of thyroliberin. Hypothyroidism can also develop due to the resistance of peripheral tissues to the action of thyroid hormones. Deficiency of thyroid hormones, which is observed in all forms of hypothyroidism, leads to metabolic disorders and changes in the activity of all systems and organs.

Clinical picture. The main clinical symptoms of hypothyroidism are general weakness, fatigue, drowsiness; poor tolerance to cold, lowering of body temperature; decrease in memory and interest in others; dry skin, hair; weight gain with reduced appetite; low hoarse voice; constipation; disorders of the menstrual cycle; paresthesias, arthralgias, etc.

Classification and examples of formulation of the diagnosis:

Etiological classification of hypothyroidism (A. M. Okorokov, 2000)
I. Primary hypothyroidism (associated with damage to the thyroid itself)
1. Congenital:
• thyroid hypoplasia or aplasia;
• hereditary defects of biosynthesis of thyroid hormones (congenital defects of enzyme systems, defects of thyroglobulin biosynthesis).
2. Acquired:
• postoperative;
•post-radioiodine or as a result of damage by ionizing radiation;
• due to insufficient supply of iodine to the body;
• as a result of the action of drugs (thyrostatic drugs, cordarone);
• as a result of the transferred inflammatory process of the thyroid (autoimmune, viral thyroiditis);
• neoplastic processes of the thyroid gland.
II. Secondary (with damage to the pituitary gland and decreased secretion of thyrotropin):
• adenohypophysis ischemia after bleeding (trauma, childbirth);
• inflammatory processes of the pituitary gland;
• tumors of the brain, pituitary gland;
• autoimmune hypophysitis;
• medicinal effects (treatment with large doses of reserpine, bromocriptine, levodopa).

III. Tertiary hypothyroidism (with damage to the hypothalamus and decreased secretion of thyroliberin):
• inflammatory processes of the hypothalamic zone;
• craniocerebral injuries;
• brain tumors;
• long-term treatment with serotonin drugs.
IV. Peripheral hypothyroidism (in case of inactivation of thyroid hormones in the circulation or insensitivity of peripheral tissues to them:
• familial form of decreased sensitivity of receptors of peripheral target tissues to thyroid hormones;
• inactivation of thyroid hormones by antibodies in the circulation process;
• violation of the conversion of thyroxine into triiodothyronine in the liver and kidneys;
• selective resistance to thyroxine (defect of transport of thyroxine through the plasma membrane to the cytosol of the cell).

Clinic. There are subclinical and clinically obvious forms of the disease. Subclinical hypothyroidism is established in the absence of clinical symptoms, an increase in the level of blood thyrotropin and normal levels of thyroxine and triiodothyronine. Clinically obvious (manifest) hypothyroidism is diagnosed in the presence of typical clinical symptoms, an increase in the level of thyrotropin, a decrease in the content of thyroxine and triiodothyronine in the blood.

The clinical diagnosis indicates hypothyroidism, its etiological form, and degree of severity.

The mild form is characterized by the presence of drowsiness during the day, edema on the face, hypodynamia, weight gain, decreased memory and attention, a feeling of mild muscle weakness, bradycardia (< 60 bpm) and the presence of thickening of the mitral valve chords on ultrasound of the heart, as well as extrasystoles (up to 15 per hour), psychasthenia.

Moderate hypothyroidism - complaints and clinical signs of the disease indicate manifest hypothyroidism. Characteristic bradycardia (pulse - less than 50 bpm), arterial hypotension, on the ECG - a decrease in the amplitude of the T wave and depression of the ST segment, extrasystole of moderate intensity (15-20 per hour); an ultrasound of the heart reveals asymmetric septal hypertrophy, systolic and diastolic dysfunction of the heart, and a decrease in the ejection fraction. The presence of myopathies, slowing of reflexes, neuropathy is possible. Patients note a decrease in memory and intelligence.

A severe form of hypothyroidism - complaints and clinical manifestations of the disease are clearly defined. Characteristic are bradycardia (up to 40 heart contractions per minute) and arterial hypotension, extrasystole (over 30 per hour), a decrease in the

voltage of the waves on the ECG, the presence of asymmetric septal hypertrophy, systolic and diastolic dysfunction, a decrease in the ejection fraction, an increase in peripheral vascular resistance during ultrasound of the heart . Sharply reduced muscle tone and strength. Apathy, hypochondria, melancholy, depressive states, decreased memory and intelligence are characteristic.

Diagnosis of the disease is based on the registration of typical complaints of the patient. Physical examination reveals bradycardia, decreased pulse rate, changes in the cardiovascular system (enlargement of heart borders, hypotension or a slight increase in blood pressure due to activation of the renin-angiotensin-aldosterone system), paleness and dryness of the skin, brittle hair and nails; tendon reflexes are reduced, extremities are cold, swelling is found. One of the early symptoms is an increase in the tongue, a decrease in the tone of the voice. Quite early, patients note a decrease in physical activity, drowsiness. Constipation often occurs, appetite decreases, and body weight increases. During the objective examination of patients, bradycardia and changes in blood pressure are determined.

Laboratory and instrumental studies

On the ECG - bradycardia, low amplitude of R, P waves and the entire QRS complex. When studying biochemical indicators, hypercholesterolemia and hyperlipidemia are determined; increased blood concentration of creatine phosphokinase, lactate dehydrogenase. Decreased glomerular filtration rate of kidneys (in some cases up to 75% of normal). In the urine - moderate proteinuria. A general blood test can detect anemia due to a decrease in the blood content of vitamin B12, folic acid, and iron. In some cases, there is a relative lymphocytosis, an increase in ESR. During the hormonal examination of patients, the level of thyrotropin is increased (typical for primary hypothyroidism), and thyroxine and triiodothyronine are decreased. In secondary and tertiary hypothyroidism, the levels of thyrotropin, thyroxine and triiodothyronine are reduced. In women, there may be disturbances of the menstrual cycle due to changes in the secretion of follitropin, lutropin and an increase in the secretion of prolactin (this may be the reason for the detection of a specific syndrome of lactorrhea-amenorrhea - the Van-Wyck-Ross-Hennes syndrome, in children and adolescents - the Van-Wyck-Grombach syndrome). The time of the Achilles reflex decreases. The size of the thyroid gland on ultrasound can be normal, reduced (aplasia or hypoplasia) or increased in the hypertrophic form of autoimmune thyroiditis. For the diagnosis of the latter, it is important to determine the content of antibodies to thyroperoxidase, thyroglobulin in blood serum (there is an increase in these indicators). Hypothyroidism is characterized by a decrease in the level of ionized and total calcium in the blood, a violation of mineralization of the skeleton.

The diagnosis of primary hypothyroidism is clarified by a number of diagnostic laboratory tests. Functional insufficiency of the thyroid gland is characterized by a decrease in the level of thyroxine, triiodothyronine and an increase in the content of thyrotropin in the blood of patients. In autoimmune hypothyroidism, the level of antibodies to thyroid peroxidase and thyroglobulin may be elevated. Conducting a test with intravenous administration of 200 μg of tyroliberin allows diagnosing secondary and tertiary hypothyroidism. Thus, in the primary case, a hyperergic reaction to tyroliberin is observed, in the secondary case, the release of thyrotropin decreases, in the case of the tertiary case, the reaction to tyroliberin is preserved, but slowed down. Additional methods for diagnosing hypothyroidism include recording the time of the Achilles reflex, ECG, determining the level of HC, b-lipoproteins in the blood, ultrasound of the thyroid gland.

Differential diagnosis of hypothyroidism should be carried out with chronic glomerulonephritis, circulatory failure, anemia. This diagnosis is based on the detection of reduced levels of thyroxine and triiodothyronine in the blood; in primary hypothyroidism, the level of thyrotropin is increased, in secondary and tertiary hypothyroidism, it is decreased.

Treatment for hypothyroidism. The main method of treatment for hypothyroidism is replacement therapy (H03A - thyroid hormone preparations) using levothyroxine, triiodothyronine and combined preparations. The dosage of drugs is calculated depending on the initial levothyroxine dose of 1.5-1.8 μg/kg of the patient's body weight. The principle of treatment of hypothyroidism is based on a gradual increase in the dose of thyroid hormone preparations to an adequate level. Control of the effectiveness of treatment of primary hypothyroidism is carried out on the basis of determining the level of thyrotropin in the blood (only 1.5 months after the start of treatment). The rate and degree of reduction in the thyrotropin level indicate the rate of compensation of hypothyroidism and the adequacy of the dose. For the treatment of secondary hypothyroidism, it is necessary to remove the pituitary adenoma followed by replacement therapy according to general rules.

Polyclinic departments in which diagnosis, prevention and treatment of hypothyroidism are carried out under the supervision of an endocrinologist, in the case of significant decompensation of the disease, the presence of complications from the cardiovascular and nervous systems, osteoporosis, patients are referred to endocrinology departments, specialized centers, dispensaries for appropriate treatment. Diagnosis and treatment of hypothyroidism are carried out in institutions of the 2nd and 3rd levels of accreditation. A separate indication for hospitalization is an allergy to thyroid hormone preparations.

The list and volume of mandatory medical services. Examination by an endocrinologist, determination of the content of free or total thyroxine, free or total triiodothyronine, thyrotropin, antibodies to thyroid peroxidase, thyroglobulin, thyroid ultrasound, urine iodine excretion (in areas endemic for goiter). In the presence of secondary or tertiary hypothyroidism, an examination by a neurologist, X-ray of the skull or MRI of the brain, an examination by an ophthalmologist, and, if necessary, a neurosurgeon are required.

The list and volume of additional medical services. Ultrasound with thyroid dopplerography, determination of prolactin, follitropin, lutropin levels; consultation of a neurosurgeon, gynecologist; Ultrasound of ovaries, uterus, osteodensitometry.

Characteristics of algorithms and features of medical services. Detection of complaints typical for hypothyroidism in a patient involves referral to an endocrinologist for an examination, ultrasound of the thyroid gland, determination of the blood level of thyroxine (free or total), triiodothyronine (free or total), thyrotropin, antibodies to thyroid peroxidase, thyroglobulin, iodine excretion with urine Such procedures are safe for the patient's health, allow to determine the state of the thyroid gland, its functional activity, type of hypothyroidism, usually do not cause complications.

Depending on the form of the disease, timely diagnosis of hypothyroidism and adequate preventive and therapeutic measures in an outpatient setting or day hospital, specialized endocrinological departments, centers, dispensaries lead to a decrease in the manifestations of hypothyroidism, improvement of the patient's condition, prevent the development of complications and disability of patients.

Characteristics of the final expected result of treatment. As a result of the treatment, compensation for hypothyroidism is achieved, and the condition of patients improves. The working capacity of patients is restored and maintained under the conditions of adequate continuous treatment.

With full compensation of hypothyroidism, in the future, patients need careful dispensary supervision, a full endocrinological examination, correction of treatment 2-3 times a year. If there are complications from the cardiovascular, nervous, and skeletal systems, examinations are performed more often (preferably once every 3 months).

Requirements for dietary prescriptions and restrictions. Patients are provided with recommendations regarding a diet balanced by the content of proteins, fats, carbohydrates, trace elements and vitamins.

Requirements for the regime of work, rest, treatment, rehabilitation. Patients with hypothyroidism must adhere to the work regime (under the prohibition of night shift

work, in hot workshops, exposure to the sun), work must be alternated with rest. Patients need 8 hours of sleep a night. Preventive measures and treatment should be continuous, controlled and adequate in terms of quality and duration.

General material and mass-methodological support
lectures:

1. work program of the academic discipline
2. synopsis (plan-summary) of the lecture
3. multimedia presentation of the lecture

Questions for self-control:

1. Name the hormones synthesized by the thyroid gland and their functions.
2. Describe the concept of primary, secondary and tertiary hypothyroidism.
3. Etiology of hypothyroidism.
4. Thyroid disease syndromes.
5. Examination plan for a patient with hypothyroidism.
6. The main complaints of a patient with hypothyroidism.
7. Differential diagnosis of hypothyroidism.
8. Laboratory diagnosis of hypothyroidism.
9. Instrumental diagnosis of hypothyroidism.
10. Principles of treatment of hypothyroidism.
11. Describe the concept of primary, secondary and tertiary hyperthyroidism.
12. Etiology of hyperthyroidism.
13. Syndromes of hyperthyroidism.
14. Examination plan for a patient with hyperthyroidism.
15. The main complaints of a patient with hyperthyroidism.
16. Differential diagnosis of hyperthyroidism.
17. Laboratory diagnosis of hyperthyroidism.
18. Instrumental diagnosis of hyperthyroidism.
19. Principles of treatment of hyperthyroidism.
20. Describe changes in the oral cavity in patients with hyperthyroidism.
21. Explain the features of providing dental services to a patient with hyperthyroidism.
22. Specify the possible causes of hypoparathyroidism.
23. Describe the complaints of a patient with hypoparathyroidism.
24. Describe the symptoms of Trousseau and Chvostek.
25. What other symptoms can be detected on the upper and lower limbs, face with this disease?
26. To characterize an attack of tetany in hypoparathyroidism.

27. Indicate the principles of emergency care for an attack of tetany.
28. Describe the clinical manifestations of laryngospasm in hypoparathyroidism.
29. Describe emergency care for laryngospasm in a patient with hypoparathyroidism.
30. To characterize changes in tooth enamel in hypoparathyroidism.
31. To substantiate mandatory laboratory and instrumental studies in hypoparathyroidism.
32. Explain the significance of blood calcium determination as a screening marker for parathyroid diseases.
33. Indicate the principles of medical treatment of hypoparathyroidism.
34. Describe the difference between primary and secondary
35. hyperparathyroidism. Specify the reasons for their occurrence.
36. To describe the changes in bone tissue in hyperparathyroidism, in particular the condition of the lower and upper jaws (fibrocystic osteopathy, osteoblastoclastoma of the jaws, pathological fractures).
37. Describe the changes in the kidneys and gall bladder in patients with hyperparathyroidism.
38. To justify mandatory laboratory and instrumental studies in hyperparathyroidism.
39. Specify laboratory markers of bone tissue resorption.

references

1. Vlasenko M.V., Palamarchuk A.V., Prudius P.G. Diagnosis and treatment of patients with nodular goiter. Guidelines. - K. Medknyg Publishing House, 2019. - 72 p
2. American Diabetes Association (2022). 9. Pharmacologic Approaches to Glycemic Treatment: Standards of Medical Care in Diabetes—*2022*. Diabetes Care 2021;45 (Suppl.1): S125-S143 / https://doi.org/10.2337/dc22-S009
3. *Williams* Textbook of Endocrinology. Shlomo Melmed, Ronald Koenig, Clifford Rosen, Richard Auchus, Allison Goldfine. 14 edition, 2019. – 1792 P.
4. American Diabetes Association (2022). 6. Glycemic Targets**:** Standards of Medical Care in Diabetes—2022. Diabetes Care 2021;45, (Suppl.1): S83-S96 | https://doi.org/10.2337/dc22-S006
5. Endocrinology: a textbook (Y.I. Komisarenko, H.P. Mikhalchyshyn, P.M. Bodnar, etc.) Edited by Professor Yu.I. Komisarenko, - Ed. 5, processing. and additional – Vinnytsia: Nova Kniga, 2020. – 456 p.

Electronic information resources:

1. http://moz.gov.ua - Ministry of Health of Ukraine
2. www.ama-assn.org – American Medical Association /American Medical Association
3. www.who.int - World Health Organization
4. www.dec.gov.ua/mtd/home/ - State Expert Center of the Ministry of Health of

Ukraine

5. http://bma.org.uk – British Medical Association
6. www.gmc-uk.org - *General Medical Council* (*GMC*)
7. www.bundesaerztekammer.de – German Medical Association