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GENERAL INFORMATION ABOUT DIABETES MILETUS

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Annotation: *Occurs in the pathology of the endocrine system: 1. Disease or symptom of Itsenko - Cushing (a disease of chronic excess of cortisone). 2. Acromegaly (excess growth hormone). 3. Pheochromocytoma (a tumor that produces catecholamines in excess). 4. Cohn's symptom (primary hyperaldosteronism). Under the influence of aldosterone, the level of potassium decreases, and it is necessary for the utilization of glucose. 5. Glucogonoma (tumor from L-cells of the islets of Langerhans). The patients are emaciated, with ulcers on the extremities. Secondary pancreatic diabetes: after removal of the pancreas, with pancreatic cancer (body and tail). Iron storage disease (hemachromatosis). Normally, the level of iron in the blood is regulated by a feedback mechanism. Iron is absorbed more than necessary, and it enters the liver, pancreas, skin: Triad: dark skin, gray color, enlarged liver, diabetes mellitus.*

Keywords: *types of diabetes mellitus,primary ,idiopathic,symptomatic.*

Diabetes is a very common disease. It affects 2 to 4% of the population. According to American statistics, 50% of diabetic patients die from myocardial infarction, from blindness (2nd place), from atherosclerosis of the extremities, from pyelonephritis, from urolithiasis. Acute complications of diabetes mellitus 1. Diabetic ketoacidosis. Hyperosmolar coma. Hyperglycemia. Diabetes mellitus is a chronic polyetiological disease, which is characterized in terms of disorders by hyperglycemia, protein catabolism, fat, and regardless of the cause, these disorders are associated with a lack of insulin (absolute and relative). In diabetes mellitus, the fasting blood glucose level is more than 7.2 mmol / l at a two-time study (* 18 mg%). Types of diabetes mellitus 1. Primary (idiopathic). 2. Secondary (symptomatic).

Insulin - dependent diabetes mellitus - absolute insulin deficiency - type 1. 2. Insulin - independent diabetes mellitus. Occurs with relative insufficiency of insulin. In the blood of such patients, insulin is normal or elevated. May be obese or normal weight. Insulin dependent diabetes mellitus is an autoimmune disease. Its development is based on: 1. A defect in the 6th - 1st chromosome associated with the NLA system - D3, D4. This defect is hereditary. 2. Mumps, measles, coxsackie viruses, severe stressful

situations, some chemicals. Many viruses are similar to beta cells. A normal immune system resists viruses. With a defect, the islets are infiltrated by lymphocytes. B-lymphocytes produce cytotoxic antibodies. beta cells die, and insulin deficiency develops - diabetes mellitus. Insulin-dependent diabetes mellitus has a genetic defect, but manifests itself without the action of external factors. 1. Defect in the beta cells themselves and peripheral tissues. Insulin secretion can be basal and stimulated (at a blood glucose level of 6.5 mmol/l). 2. The sensitivity of peripheral tissues to the action of insulin decreases. 3. Changes in the structure of insulin. Insulin-independent diabetes is affected by obesity. At the same time, cells need more insulin, and there are not enough insulin receptors in the cells. Clinical manifestations 4 groups of disorders: 1. Metabolic disorders violation of carbohydrate metabolism - hyperglycemia, protein catabolism, fat catabolism. 2. Polyneuropathy, peripheral and autonomous. 3. Microangiopathy. 4. Macroangiopathy (atherosclerosis). Metabolic Disorders The function of insulin is the utilization of amino acids and glucose from human food. Tetraanabolic hormone lowers blood glucose levels. He is opposed by: 1. Glucagon. The stimulus for its secretion is a decrease in blood glucose levels. It works by glycogenolysis. An increase in blood glucose stimulates the breakdown of protein, glucose is formed from amino acids. 2. Cortisone - stimulates protein catabolism and gluconeogenesis. 3. Growth hormone - promotes protein synthesis, saves glucose for RNA synthesis. 4. Adrenaline - stimulates the breakdown of glycogen, inhibits the secretion of insulin. The normal concentration of glucose in the blood is less than 6.1 mmol / l. The maximum limit during the day is 8.9 mmol / l. The action of insulin with an increased amount of glucagon, little glucose is consumed in the cells, so the permeability decreases. The patient complains of: thirst, polyuria (with type 1 diabetes), weight loss, increased appetite. Polyuria is due to the fact that with an increase in glucose concentration of more than 9 - 10 mmol / l, glucose appears in the urine. Osmotic diuresis - a lot of urine with a high specific gravity. Thirst: blood osmolarity increases, the thirst center is stimulated. Weight loss: contrainsular factors have a lipolytic effect --> weight loss. Increased Appetite: Since the tissue does not efficiently utilize glucose, the hunger center is stimulated. In type 2 diabetes, obesity develops. since insulin is sufficient for the implementation of lipogenesis, however, in 5% of patients it is difficult to decide what type of diabetes they have.

Differential diagnosis 1. Diabetes insipidus. Thirst and polyuria are characteristic. This is a disease of lack of ADH produced by the hypothalamus. Functions of ADH - the reservation of fluid in the body. With diabetes, there is a lot of urine and its density is high. In diabetes insipidus, the specific gravity of urine is less than 1005. 2. Renal glucosuria is associated with a decrease in the threshold for glucose. She is moderate and unstable. 3. Glycosuria of pregnant women. Glucose in urine at normal blood levels. Fasting low blood glucose, but there is glucose in urine (hence low threshold). 4. Allocate diabetes of pregnant women. The placenta produces many anti-insular hormones. classification 1. Clinical classes: 1) Diabetes mellitus: (more than 7.2 mmol/l) primary (I

and II types), secondary. 2) Impaired glucose tolerance: obese, with normal body weight. 3) Diabetes of pregnant women. 2. Reliable risk classes, diabetic heredity, obesity, persons who had impaired glucose tolerance in the past; women who had glucose in their urine during pregnancy, etc. Acute complications of diabetes 1. Diabetic ketoacidosis. 2. Hyperosmolar coma. Diabetic ketoacidosis is an acute, very serious condition from which the patient will not come out on his own, death within 3-4 days. Mortality from DKA is 5-6%. DKA is a clinical and biochemical syndrome with high blood glucose levels, glucosuria, and hyperketonemia. Systemic acidosis --> dehydration --> collapse. The reason: a sharp lack of insulin and an excess of contrainsular hormones. TYPE I DIABETES 1. Undiagnosed type I diabetes. 2. Termination of insulin treatment. 3. Development of DKA during severe illness. DKA CLINIC. 1. Develops relatively gradually. The condition worsens within 1-2 days. 2. According to the course of DKA, there are: a) beginning DKA - ketoacidotic stupor. b) ketoacidotic coma. Beginning of DKA 1. The patient is conscious. 2. Complaints of weakness. 3. Thirst and polyuria are more pronounced. 4. Gastrointestinal syndrome (anorexia, nausea, vomiting can be repeated, frequent, 40-60% have abdominal pain due to dehydration. Objective data Skin and mucous membranes are dry; skin turgor is greatly reduced; smell of acetone in exhaled air; large noisy Kussmaul breathing due to irritation of arterial blood with acetone, pH> 7.2 tachycardia, increasing depression of the central nervous system (stupor), circulatory collapse may develop, deep loss of consciousness (coma) Acute renal failure develops, as renal filtration decreases sharply. pH < 7.0 without Kussmaul respiration is a poor prognostic sign 1. Glucose more than 300 mg% (18 mmol/l) 2. Glucosuria, severe aceturia ++++ 3. pH < 7.3 7.3-7 ,2 - mild acidosis, 7.2-7.0 - severe acidosis, 7.0 or less - severe acidosis, pH = 6.8 - incompatible with life In peripheral blood: hyperleukocytosis 13-35,000 with a shift to the left; increased creatinine (0.2-0.5).Causes: sharp protein catabolism and prerenal azotemia, the content of aliyah is decreasing.

HYPEROSMOLAR COMA 1. More severe than DKA. 2. It occurs much less frequently - 0.001%. Blood osmolarity sharply increases, hyperglycemia is more pronounced - up to 2000 mg%. These patients do not have ketoacidosis, only hyperglycemia. It develops in older people with type 2 diabetes. To suppress lipolysis, you need a little insulin. In hyperosmolar coma, insulin is sufficient to suppress lipolysis, hence there is no acidosis. Pronounced gluconeogenesis. a lot of sorbitol is formed. Clinical picture Characterized by the same complaints as in DKA. Gastrointestinal syndrome is less pronounced. Depression and loss of consciousness come on faster. No Kussmaul breath and no acetone breath. In the treatment of GOK, a good result is achieved faster. Laboratory data There is no acetone in the urine, or one +; blood pH is normal (7.35); increased creatine (protein catabolism occurs); hyperleukocytosis is less pronounced.

TREATMENT OF DIABETES MELLITUS Physician Objectives 1. Achieve adequate control of diabetes. The level of glucose in the blood on an empty stomach should be less than 6.5 mmol / l, after eating - less than 7-8 mmol / l. 2. Normalization of blood lipid spectrum = prevention of chronic complications. 3, Treatment of complications. 4. If it is a child, then treat it so that it has normal growth and development. Diet. general principles 1. Calorie content (you need to have a body weight close to ideal). For example, with a height of 100-105 cm, the number of calories in this case is 1600-2500 kcal, depending on physical activity. If the patient is obese, then calories should be less. The ideal weight is 60 kg, if the patient weighs 90 kg, then 20-15-10 kcal should fall on 1 kg of weight. 2. Qualitative composition of food. Protein makes up 20% of food. A healthy person needs 60-80 g per day, a sick person 50-40 g. Excess protein contributes to glomerulosclerosis. Fat is 30% (of which 10% is animal fat, 90% is vegetable fat + vitamin A). Carbohydrates - 50-60% - refined sugar and sugar-containing foods should be completely excluded from food. Instead of sugar, use saccharin 50 mg 3 times a day (however, there is evidence that it can cause bladder cancer). - simple carbohydrates - all vegetables (except potatoes) and some berries and fruits - cranberries, sour apples and fruits, lemons - intermediate carbohydrates - bread, bananas, grapes, potatoes, cereals, milk quantity. 3. At the same time, the same amount 3-4 times a day - intermediate carbohydrates. 4. Interchangeability. Type II diabetes begins to be treated with a diet. If the diet does not help, then sugar-lowering drugs are added to the diet: 1. Sulfamide-urea - in type 11 diabetes - increases the secretion of insulin into the blood. 2. Bishuanides do not act on beta cells. They inhibit gluconeogenesis, increase sensitivity in patients with normal body weight. Biguanides are given to obese persons (after meals). Contraindications: type 1 diabetes mellitus, major surgery, pregnancy, chronic renal failure, severe liver damage with impaired function, cardiopulmonary insufficiency. Preparations of short and long action: sulfanil - urea 0.5 hours before meals. Side effect - hypoglycemia, gastrointestinal discomfort, thrombocytopenia. Biguanide derivatives: reduce appetite, cause nausea. If there is no effect from the treatment, then the drugs can be combined, if this does not help, but it is necessary to switch to insulin. It is necessary to dose physical activity. Treatment of type 1 diabetes mellitus 1. Diet. 2. Insulin. 3. Physical activity. 30-40 units of insulin per 1 kg of the patient's weight. In the period between meals, short insulin is administered. Be sure to carry out control during the day on an empty stomach or 2 hours after eating. Complications of insulin treatment - Hypoglycemia Causes: 1. The patient is ignorant or undisciplined. Repeated, even mild, hypoglycemia leads to encephalopathy. 2. Overdose of insulin. 3. Excessive physical activity, since contra-insular hormones are produced, and they stimulate gluconeogenesis normally, but a patient with diabetes does not have it. There is more insulin in a patient than in a healthy one. Under these conditions, the clinical manifestations of hypoglycemia on the part of the brain are encephaloglucopenia, since the brain is insulin-independent tissue. Sympathoadrenal

stage - adrenaline glucagon is released, the tone of the sympathoadrenal system increases. The patient sweats, there is a feeling of anxiety, anxiety, hunger, palpitations, internal tremor. A person can independently exit this phase. Aggression, inadequacy, increased muscle tone, loss of contact with the patient, there may be loss of consciousness, convulsive seizure with defecation and urination. The duration of the coma is an indicator of the prognosis. Everything is developing rapidly. The patient should always have 6 lumps of sugar in his pocket and a syringe with glucagon, if the patient is unconscious, he is injected intravenously with a 40-50% glucose solution of 20-80 ml. Hypoglycemia gives rise to hyperglycemia.

THYROID DISEASES very diverse. 1. With an increase in the function of the thyroid gland - thyrotoxicosis, hyperthyroidism. 2. With a decrease in thyroid function - hypothyroidism. 3. With normal function - thyroiditis, endemic goiter, sporadic goiter, nodules.

HYPERTHYROISIS Thyroid hormones - triiodothyronine T3 and tetraiodothyronine (thyroxine) T4. Thyrotoxicosis syndrome - This is a polyetiological syndrome, the manifestations of which are associated with an increased amount of T3 and T4 in the blood. Causes 1. Diffuse toxic goiter, as one of the manifestations of Greevs' disease (Graves' disease). 1835 - the disease is described by Greevs. 1821 - the disease is described by Peri. 1840 - the disease is described by Bazedov. 2. Toxic thyroid adenoma - Plummer's disease. 3. Other nodular toxic goiter. Greevs' disease is a multisystem autoimmune disease that proceeds according to the type of delayed-type hypersensitivity. Greevs' disease: 1. Diffuse goiter --> thyrotoxicosis 90% 2. Infiltrative ophthalmopathy and ophthalmoplegia 50% 3. Infiltrative dermopathy. DIFFUSE TOXIC GOITER A defect in T-suppressors underlies the disease. Women between the ages of 20 and 50 are most often affected. Causes: infection, insolation, severe emotional stress. Helper T cells stimulate B cells and thyroid stimulating antibodies (TSaB) are produced. With an excess of cortisol, the control of T-suppressors is disrupted. TSaB sit on receptors, so the gland is under the control of TSaB, so the thyroid gland produces more T3 and T4. Stimulates the growth of the gland, sometimes to very large sizes. Normally, the weight of the thyroid gland is 20 g. On average, the weight can increase up to 600-800 g. 1. It is believed that the antigenic structure of retrobulbar fiber and thyroid antigens have a commonality. 2. There is a closed lymphoid circle between the thyroid gland and retrobulbar tissue. 3. There is an infiltration of the oculomotor muscles and dystrophic changes occur in them. INFILTRATIVE DERMOPATHY There is an increase in the volume of predibial fiber. Stimulation by T-lymphocytes. You can not collect the skin in a fold. Sometimes the skin is red and hot. Thyroid hormones have a wide spectrum of action: 1. on mitochondria - activation, an increase in the processes of oxidative phosphorylation, the Krebs cycle is activated, cellular respiration, the effect on catecholamines is enhanced. 2. The number of receptors

increases. In such patients, the utilization of energy substances decreases and lipolysis increases.

THYROID DISEASES (continued) The thyroid gland can be moderately enlarged and up to grade 3-4 goiter. On palpation, the thyroid gland is soft-elastic in consistency, but can also be dense. Cat's purring (= vascular murmur). Auscultation due to strong blood supply - vascular noise. Clinical manifestations of thyrotoxicosis 1. CNS manifestations. 2. Skin, 3. Muscular system. 4. Cardiovascular system. 5. Eye. 6. Gastrointestinal tract. 7. Lymphoid system. 8. Endocrine system. Manifestations from the central nervous system Patients are often fussy and restless, with rapid speech, irritable, touchy, quickly give vent to tears, anxious, often sleep poorly - this is due to the excessive effect of catecholamines on the brain. 1 Complaints of patients on: 1. To reduce tolerance to heat. They are always hot. This is because the body generates a lot of heat energy. Peripheral vasodilation - adaptation to heat - sweating. The skin is hot and moist. 2. Feeling of internal trembling and trembling of the hands (characterized by a small tremor of outstretched arms, eyelids with closed eyes), Manifestations from the muscular system. Severe weakness leads to thyrotoxic myopathy. From the CCC 1. Tachycardia in 99% of patients. Patients feel a heartbeat. Heart rate from 90 to 150 beats per minute. This tachycardia is constant and persists even during sleep. 2. Change in blood pressure: an increase in systolic and a decrease in diastolic - 160/60 and 140/50. 3. Hypertrophy of the left ventricle - manifested by increased apex beat. The size of the heart is normal. 4. During auscultation, loud 1 tone (clapping). Noise systolic intensive, rough. The reason is an increase in blood flow. Thyrotoxic heart leads to thyrotoxic cardiopathy. Lack of vitamin B1, cocarboxylase is not formed, in such patients the heart is similar to the heart in patients with beriberi syndrome. This is a secondary cardiopathy. More often right ventricular heart failure. In young people, thyrotoxicosis from the heart is more common. 1. Atrial fibrillation. 2. Left ventricular heart failure - shortness of breath at rest or during exercise, cough, hemoptysis, cyanosis, moist rales. A symptom of sinus node weakness in the elderly is atherosclerosis of the artery supplying the sinus node. This symptom may be delayed in thyrotoxicosis if there is no tachycardia, and even a tendency to bradycardia is noted. Thyrotoxicosis is the second reason that provokes the weakness of the sinus node. Manifestations from the side Gastrointestinal tract 1. Weight loss in 95 - 98% is associated with lipolysis and increased activity of catecholamines. 2. Appetite is good. 3. Frequent stools (from 2 to 10 - 15 times a day) without tenesmus and mucus. Eye manifestations Thyrotoxic symptoms are characteristic. Muscle retraction is Kocher's symptom. White stripe of sclera between iris and upper eyelid. Gref's symptom (see below). Wide palpebral fissure = Del Rimple's symptom. Increased tone of the retrobulbar muscles. Eye shine. Lymphatic system Lymphadenopathy as a manifestation of an autoimmune disease. Skin There may be areas of depigmentation. Endocrine system Increased need for cortisil. Relative insufficiency of the adrenal cortex develops, characterized by severe

weakness, fatigue, weight loss, skin pigmentation (increased ACTH). Infiltrative ophthalmopathy - it is judged by its appearance: swelling of the upper eyelid. Causes of thyrotoxicosis 1. Grevs' disease. 2. Toxic adenoma of the thyroid gland. Features: there will be no toxic goiter, a node in the thyroid gland is palpated, more often in the elderly. Hypotrophy and hypofunction of the surrounding tissue of the thyroid gland. The main manifestations of the cardiovascular system are angina attacks, left ventricular failure, there will never be infiltrative ophthalmopathy. Diagnosis: T3 and T4 are normal, TSH is low. The main test is a thyroid scan. A hyperfunctioning node is revealed. A test with T3 is not done for the elderly. 3. Multinodular toxic goiter. Munchausen's symptom (mental illness of the disease), the thyroid gland is not enlarged, there are no eye symptoms, low iodine uptake by the thyroid gland. Complications of thyrotoxicosis Thyrotoxic crisis is an acute condition with a sharp increase in catecholamines in the blood with a pronounced insufficiency of the adrenal cortex. This can be with severe untreated thyrotoxicosis, provoked by infection and stress. Clinic Dissociation of oxidative phosphorylation, increased activity of catecholamines, high body temperature (more than 39 degrees C), excessive sweating, rare tachycardia, atrial fibrillation with tachysystole, heart failure, vomiting, anorexia, frequent loose stools, collapse, dehydration. The patient himself will not come out of the thyrotoxic crisis. There are no laboratory data explaining the clinic.

TREATMENT OF THYROTOXICOSIS Treatment can be conservative and surgical. Use J131. Thyrostatic drugs: a) methylmazol derivatives: mercazolil, metatilin, methylmazol. b) thiuracil derivatives. Mechanism of action They inhibit the synthesis of thyr- and tetraiodothyronine in the thyroid gland. They bind and inactivate pyroxidase systems, the immunosuppressive effect is weak. Propylthiuracil at the periphery reduces the conversion of T3 to T4. I. Mercazolil Tablets 5 mg. Daily dose of 30 mg (2 tablets 3 times a day). The action occurs after 2-3 weeks, since there are iodine reserves in the thyroid gland and the binding of peroxidase systems does not occur immediately. Beta-blockers are prescribed until Mercazolil has begun its action. Mercazolil is used until the onset of euthyroidism. It does not act on iodine uptake. Then they switch to smaller maintenance doses. 2 tablets 3 times a day, then 10 mg/day. Side effects of Mercazolilum 1. Urticaria. 2. Gastrointestinal discomfort (bitterness in the mouth). 3. Agranulocytosis in 1% of patients (fever, sore throat - stop taking the drug). Necrotic tonsillitis, temperature 38-390C, peripheral blood leukocytopenia 1000-800, neutrophils 2-3%. With agranulocytosis: preparations of lithium salts. Lithium carbonate 0.3 3 times a day - inhibits the synthesis of thyroid hormones, but the effect is much weaker than mercazolil. Antibiotics, vitamins, prednisolone are also used, if lithium salt does not help. II. beta-blockers 1. Anaprilin 30 to 80 mg on average (can be from 20 to 160 mg / day - individual dose selection is used. 2. Trazikor. Apply until euthyroidism sets in. III. vitamin B1 Applied per os 50 mg 3 times a day Or 6% solution parenterally 1 ml IM for 2-3 days if the patient has frequent stools IV Sodium Perchlorate Blocks thyroid iodine

transport systems Not currently used Used at 200 mg doses 3-4 times a day, i.e. up to 1 g per day Depending on the condition, the dose is reduced V. Lithium preparations after agranulocytosis: lithium carbonate (see above) If thyrotoxicosis is not severe, then beta-blockers are used VI. Inorganic iodine preparations - Lugol's solution 1923 - Glumer: iodine blocks proteolytic enzymes in the (thyroid gland, the level of thyroid hormones rapidly decreases. Synthesis of thyroid hormones is inhibited, iodine uptake decreases. Inorganic iodine is not treated for a long time. cytosis, the patient is operated on. 2. Before preparing for surgery. Z. Thyrotoxic crisis. 4. In severe thyrotoxicosis, when there is a threat of a crisis. Indications for surgery 1. Very large gland. 2. Insufficient maintenance doses rkazolil. 3. If the treatment lasts 1-2 years without remission. Danger of operation 1. General anesthesia. 2. Damage to the parathyroid glands - transient hypoparathyroidism, chronic hypoparathyroidism. 3. Damage to the larynx (hoarseness of the nose). 4. Hypothyroidism. With toxic adenoma of the thyroid gland, they operate, therefore, thyrotoxicosis is eliminated. This operation is easier. Multinodular goiter is also operated on. Treatment with iodine-131 Contraindications: 1. After 35-40 years, they give radioactive iodine, at a young age they do not give it, since the risk of thyroid cancer is high. 2. Pregnancy. 3. Patients with diffuse toxic goiter without remission (large goiter). Indications 1. Patients over 35-40 years old with DTG. If iodine-131 is used, it is impossible to prescribe an inorganic iodine preparation, since the thyroid gland is blocked. 2. The most suitable option for the treatment of toxic adenoma of the thyroid gland - patients older than 35-40 years. Treatment of thyrotoxic crisis There is no diagnosis of thyrotoxic crisis. It happens only in patients with diffuse toxic goiter. 1. Inorganic iodine. 50 drops of Lugol's solution orally, when vomiting is administered through a probe 3 times a day, KI and NaI intravenously. 2. Mercazolil up to 60 mg/day. 3. Beta-blockers.

LITERATURE:

1. Fedele F., Mancone M., Chilian W.M., Severino P., Canali E., Logan S., De Marchis M.L., Volterrani M., Palmirotta R., Guadagni F. Role of genetic polymorphisms of ion channels in the pathophysiology of coronary microvascular dysfunction and ischemic heart disease.
2. Dick G.M., Bratz I.N., Borbouse L., Payne G.A., Dincer U.D., Knudson J.D., Rogers P.A., Tune J.D. Voltage dependent K+ channels regulate the duration of reactive hyperemia in the canine coronary circulation.
3. Saitoh S., Zhang C., Tune J.D., Potter B., Kiyooka T., Rogers P.A., Knudson J.D., Dick G.M., Swafford A., Chilian W.M. Hydrogen peroxide: A feed-forward dilator that couples myocardial metabolism to coronary blood flow.

4. Bender S.B., Tune J.D., Borbouse L., Long X., Sturek M., Laughlin M.H. Altered mechanism of adenosine-induced coronary arteriolar dilation in early-stage metabolic syndrome.
5. Berwick Z.C., Payne G.A., Lynch B., Dick G.M., Sturek M., Tune J.D. Contribution of adenosine A2A and A2B receptors to ischemic coronary dilation:
6. Zukhridinovna Z. D. MODERN ASPECTS OF NEUROPROTECTIVE TREATMENT IN HYPERTENSIVE RETINOPATHY //Web of Scientist: International Scientific Research Journal. – 2022. – Т. 3. – №. 02. – С. 949-952. https://wos.academiascience.org/index.php/wos/article/view/949/880 https://wos.academiascience.org/index.php/wos/article/view/949
7. Jalalova D. Z. (2022). Interrelation of Cardiological and Ophthalmological Parameters in Patients with Arterial Hypertension in Combination with Coronary Heart Disease. Eurasian Scientific Herald, 8, 135–141. Retrieved from https://www.geniusjournals.org/index.php/esh/article/view/1437 https://www.geniusjournals.org/index.php/esh/article/view/1437
8. Zukhriddinovna, Z. D. (2022). Development of Classification Criteria for Neuroretinal Ischemia in Arterial Hypertension. Central Asian Journal of Medical and Natural Science, 3(3), 59-65. https://doi.org/10.17605/OSF.IO/K76ZT
9. Жалалова, Д. З. Метод комбинированного лечения диабетической ретинопатии // Врач-аспирант, (2009).
10. Zukhriddinovna, Z. D. (2022). Development central asian journal of natural sciences, https://doi.org/10.17605/OSF.IO/K76ZT
11. Zuxridinovna, J. D. (2022). central asian journal of natural sciences, https://cajmns.centralasianstudies.org/index.php/CAJMNS/article/view/737
12. Z. , Z. D. (2022).. central asian journal of natural sciences, https://doi.org/10.17605/OSF.IO/SYA5K
13. Pirmanova, G. (2022). Extracurricular study of world monuments culture - historical and cultural architectural english heritage . Scientific Research https://inlibrary.uz/index.php/scientific-research-covid-19/article/view/8536
14. Najmiddinovna, K. D. (2022). Improvement of mental and inventive action of young people within the consider of verifiable and social landmarks, https://confrencea.org/index.php/confrenceas/article/view/176
15. Pirmanova, G. N., Yarkulovna, S. S., Najmiddinovna, K. D., Mustafakulovich, I. A., & Shavkatovna, J. N. (2021). https://doi.org/10.21744/lingcure.v5nS2.1716 https://media.neliti.com/media/publications/507798-extracurricular-classes-on-the-study-of-48af7bd6.pdf
16. Pulatov F A, Amanova N F, Amanova F F. (2022). cultural https://doi.org/10.5281/zenodo.6673999

17. Pirmanova G. N. et al. Extracurricularhttps://lingcure.org/index.php/journal/article/download/1716/614 https://lingcure.org/index.php/journal/article/view/1716
18. Amanova N.F Amanova F.F . innovative activity http://papers.euroasiaconference.com/index.php/eac/article/view/97
19. Amanova N.F Amanova F.F (2022) Malum bir maqsadga https://conf.iscience.uz/index.php/yumti/article/view/118/110
20. Amanova N, and Amanova F. "problems of quality of http://sciencebox.uz/index.php/ajed/article/view/1515/1403
21. Normuradova N, and Amanova N. "teaching https://eprajournals.com/jpanel/upload/1206am_41.EPRA%20JOURNALS-2147.pdf
22. Amanova N.F. "active teaching strategies https://doi.org/10.5958/2249-7137.2021.02068.1
23. Pulatov F A. (2022). the http://conferencezone.org/index.php/cz/article/view/147
24. Furkatovna, A. N., & Furkatovna, A. F. http://papers.euroasiaconference.com/index.php/eac/article/view/97
25. Furkatovna A. N., Furkatovna A. F. problems of quality of distance learning http://www.sciencebox.uz/index.php/ajed/article/view/1515/1403 http://www.sciencebox.uz/index.php/ajed/article/view/1515
26. .Н.Ф Аманова. О роли контекста при https://kpfu.ru/staff_files/F312709112/SBORNIK_MATERIALOV_NPK_2020__1___1_.pdf#page=43
27. Mavlyanova Z.F. NUTritional status of children with cerebral https://cyberleninka.ru/article/n/nutritivnyy-status-detey-s-tserebralnym-paralichom
28. Farangiz F. A. problems ofhttps://cyberleninka.ru/article/n/problems-of-quality-of-distance-learning-online http://scientificprogress.uz/storage/app/media/2-2.%20262.%201506-1508.pdf
29. Farangiz F A use of modern informhttps://cyberleninka.ru/article/n/use-of-modern-information-technologies-in-teaching-foreign-languages-1
30. Farangiz F A employees as an integral part of the tourism product // scientific progress. 2021. №2. URL: https://cyberleninka.ru/article/n/employees-as-an-integral-part-of-the-tourism-product http://scientificprogress.uz/storage/app/media/2-2.%20259.%201496-1498.pdf
31. Bakhritdinov B.R, Aliev M.A, & Mardieva G.M. (2022). MULTIVOXEL MAGNETIC https://scholarexpress.net/index.php/wbph/article/view/738
32. Mamadaliev A. M., Aliev M. A., Saidov K. D. The Research Of Different Methods https://ejmcm.com/article_4676_1feec211fea5848f81258ea221dae974.pdf

33. Yuldashev S. S., Mamadaliev A. M., Aliev M. A. To Questions Of Surgical Tactics In https://www.ejmcm.com/article_3419_20727dc3df7f4324cbcb048aa2f37c88.pdf
34. Z. F. Mavlyanova Nutritional status of children with cerebral palsy https://doi.org/10.31146/1682-8658-ecg-185-1-82-88
35. Khudoykulova F, Mavlyanova Z, Kamalova Y, Ibragimova M, & Abdullayeva N.(2022). Non-alcoholic fatty liver:, 2022,, https://doi.org/10.5281/zenodo.6651517
36. Abdusalomova M.A., Mavlyanova Z.F., Ravshanova M.Z. (2021). Children https://www.annalsofrscb.ro/index.php/journal/article/view/877
37. Payzullaeva U,Kan S, Z Mavlyanova https://www.ikprress.org/index.php/PCBMB/article/view/5455.
38. Z.F., Mavlyanova correlation https://ejmcm.com/article_2365_10c437813579ff2dfe2d43cda89cbc59.pdf
39. Z Mavlyanova ,G Burkhanova MRavshanova,S Makhmudov https://ejmcm.com/article_2366_b8ee379773f1c297c6f9892b076d2d7b.pdf