MINISTRY HEALTH OF THE REPUBLIC OF UZBEKISTAN

TASHKENT MEDICAL ACADEMY

Kurbonova Z.Ch., Babadjanova Sh.A., Sayfutdinova Z.A.

«LABORATORY WORK»

STUDY GUIDE

TOSHKENT 2023

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«LABORATORY WORK»
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Subject "Methods of general clinical and cytological examination of biological materials".

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TABLE OF CONTENTS

INTRODUCTION

Laboratory work is an integral part of clinical laboratory diagnosis and helps to monitor the effectiveness of clinical diagnosis and treatment.

“ Laboratory work ” manual provides up-to-date information on cell development, their activity, morphological structure and properties.. The first chapter provides information on clinical blood analysis, erythrocytes, hemoglobin, leukocytes, anemia, and leukemia. The second chapter provides information on laboratory diagnosis of kidney disease, urine analysis, methods of testing renal function. Chapter 3 provides laboratory diagnostics of liver disease, biochemistry blood analysis, Chapter 4 Laboratory diagnostics of heart and connective tissue diseases, and Chapter 5 provides information on the blood coagulate system, hemostasis.

At the same time, each chapter is an analytical section that provides pedagogical technology, situational issues, tests, graphic organizer, practical part, and control questions that you use in training.

”Laboratory work” manual medical higher education Institutions “Laboratory work” for master's and clinical ordinance students and clinical laboratory diagnostic specialization courses for cadets, general clinical laboratory staff.

CHAPTER 1. BLOOD CLINICAL ANALYSIS.

1.1. BLOOD CLINICAL ANALYSIS

Objectives of the training: methods of laboratory examination in hematology, morphological features of pathological erythrocytes, erythrocytometry, reticulocytes, introduction to myelogram.

Hematology- is the science that studies blood, blood-forming organs, and blood diseases. Hematology is a medical field that studies the etiology, diagnosis, treatment, prevention and prognosis of blood diseases, blood and its components (blood cells, hemoglobin, blood proteins, coagulation factors).

Hematological diagnostic methods are traditionally the most common tests. Many clinical diagnostic laboratories currently use hematologic analyzers with varying levels of complexity to calculate and analyze blood cells.

Cytological examinations are important in the diagnosis of hematological diseases. Their implementation is carried out in clinical diagnostic laboratories and special hematology laboratories.

The most important of the diseases diagnosed by hematologic methods are anemia, hematopoietic tissue tumors. Hematological tests are used to assess the body's response to many diseases, to determine the severity of the disease and the effectiveness of their treatment.

Blood is a complex fluid consisting of plasma and blood-forming elements: erythrocyte-red blood cells (RBC), leukocyte-white blood cells (WBC), and platelet-blood plates (PLT).

Methods of laboratory examination of blood in hematology:

– morphological examination of erythrocytes in blood smear;

– calculation of reticulocytes;

– osmotic resistance of erythrocytes;

– platelet examination in blood smear;

– morphological examination of leukocytes;

– cytochemical reactions;

– myelogram.

The most common clinical blood test from quantitative and qualitative study methods of blood cells: hemoglobin concentration, color index, erythrocyte count, leukocyte count, leukoformula, description of morphological appearance of blood cells, assessment of erythrocyte sedimentation rate. Determining the number of reticulocytes and platelets.

Technique of drawing blood from the finger.

The patient's unnamed finger is wiped with alcoholcotton and pierced with a scarifier, 1-drop blood is wiped. Subsequent drops are taken for inspection purposes.

To make the smear, one side of the piece window is covered in blood and pushed at an angle of 45^0. A well-prepared smear appears yellowish, clear, flat in the light.

BLOOD CLINICAL ANALYSIS.

Normal blood analytics rates (for adults).

Indicator	**Normative results**
Erythrocytes:	in men 4.0 - 5.1 x1012/l in women 3.7 - 4.7 x1012/l
Hemoglobin:	130-160 g / l in men 120-140 g / l in women
Color indicator:	0.86-1.05
Platelet count :	180-320 x 109/l
Reticulocyte count :	0.2-1.2% (2-12 %)
Leukocyte count:	4.0 - 9.0 x109/l
	Leukocyte formula
Mielotsit:	won't be
Metamielocyte:	:won't be
Beacon-core neutrophils:	: 1-6 % 0.04 - 0.30 x109/l
Segment core neutrophils:	47-72% 2.0 - 5.5 x109/l
Eosinophil:	0-5 % 0.02 - 0.3 x109/l
Basophilic:	0-1% 0 - 0.65 x109/l
Lymphocyte:	19-37% 1.2 - 3.0 x109/l
Monocyte:	3-11% 0.09 - 0.6 x109/l
Plasma cells:	do not exist
Erythrocyte sedimentation rate (ESR):	1-10 mm / hour in men 2-15 mm / hour in women
Erythrocyte diameter by Price-Jones	
normocyte	68.0±0.4 %
microcyte	15.3±0.42 %
macrocyte	16.9±0.47 %
The volume of erythrocytes is	31.8±3.5 ml / kg
Hematocrit	40-48% in men% 36-42% in women%
	Erythrocyte index
The erythrocyte content of hemoglobin is (MSH)	27.0-33.3 pg

erythrocyte concentration of hemoglobin (MCHC)	30-38 %
erythrocyte volume (MCV)	75-96 μm3 (fl)
Erythrocyte diameter	7 - 8 μm

Erythrocytes.

Erythrocytes were discovered by Anton van Levenguk in the early eighteenth century. Erythrocyte function:

1. It is the main oxygen carrier.

2. At the expense of carboangidraza in erythrocytes, it is bound to water with CO2 and transport H2CO3.

3. Erythrocyte also has an ion exchange feature with plasma. In particular, the Hamburg effect is due to the exchange of cations – H+ with Na+ - to keep the acid-alkaline balance and electrolyte balance in the body at the same time.

4. Erythrocytes are adsorbent for immune complexes. From the effects of immune complexes, the blood vessel wall is a physiological preservative that prevents the formation of vasculitis.

Erythrocytes are a metabolically active cell with bilateral botic form and strong deformation ability, despite being nuclear-free and macrum-free from many organoids.

Erythrocyte diameter 7 – 8 μm. In microcytitarian anemia, the erythrocyte diameter is less than 6.5 and in macrocytitic anemia it is more than 8.5 μm. Normochromia when MCH 26 – 34 pg and MCHC 31 – 37%, hypochromia when MCH 26 pg and MCHC 31%, MCH 34 pg and MCHC 37% hyperchromy is observed when more than.

Erythrocytosis is confirmed in the following cases:

- when the concentration of Hb in women is higher than 160 g / l, hematocrit is higher than 47%, erythrocytes are higher than 5.5 x1012 / l;

- when the concentration of Hb in men is higher than 180 g /l, hematocrit is higher than 54%, erythrocytes are higher than 6 x1012 / l.

Absolute erythrocytosis(true) is observed as a result of erythropoiesis enhancement. Physiological erythrocytosis is observed in people living in mountainous areas, in sportsmen, when taking erythropoiesis-enhancing doping.

Pathological erythrocytosis is divided into primary and secondary erythrocytes.

Primary erythrocyte includes erythremia or true polycythemia. It is a series of erythroid chronic leukemia, with an increase in erythroid, leukocytar, and thrombocytic cells. Patients are observed to increase blood volume, increase blood viscosity, increase arterial blood pressure.In peripheral blood, erythrocytes increase to 7-10 x1012 /l, leukocytes to 15 – 60 x109/l, platelets to 500 – 1000 x109/l, hematocrit to 60 – 80, ECHT

is reduced to 1 – 2 mm / h. An increase in all rows of cells is observed in the bone marrow. At the terminal stage, myelofibrosis and pancytopenia are observed.

Secondary erythocytosis is observed in the following cases:

- in chronic bronchoobstructive lung disease, night apnoeia syndrome, heart defects, erythrocytes that do not lead to a reduction in erythrocyte life, but in hereditary anomalies of Hb, leading to a decrease in oxygen transport capacity as a compensatory reaction against hypoxia;

- in renal diseases accompanied by an increase in erythropoietin levels: polycystosis, hydronephrosis, after kidney transplantation, renal cancer.

Patients have elevated erythropoietin levels in the blood, as well as the following changes in peripheral blood:

- Hb 171 – 290 g / l

- Er 6 – 9 x1012 / l

- Hematocrit increases sharply

- RK is relatively low or at the lower limit

- some patients have higher bilirubin levels.

Unlike erythema, platelets and leukocytes are in the norm. The erythroid series of cells also increase in bone marrow. Relative (false)erythrocytosis is observed as a result ofblood transfusion, decreased fluid part of the blood (vomiting, diarrhea, low water drinking, dehydration as a result of multiple sweating).

Differensial diagnosis of eritrotsitozis

Parameters	Truth polycythemia	Secondary polycythemia	Relative erythrocytosis
The actual number of erythrocytes is	rised	rised	normal
Leukocytosis	peculiar	Not observed	Not observed
thrombocytosis	peculiar	Not observed	Not observed
splenomegaliya	Observed in most cases	Not observed	Not observed
r_aO_2	normal	Decreased or normal	normal
Painting of leukocytes in IF	increased	Normal	normal
Serum erythropoietin	decreased	Increased or	normal

		normal	

Hematological analyzers.

The use of modern automated analyzers for blood testing provides sufficient clinical information about the state of the hemopoietic system and its effects on various external and internal factors. YUqari-tech hematologic analyzer has the ability to measure more than 20 parameters and more than 3 histograms.

The hematologic analyzer uses the following measurement methods: impedance method for the detection of erythrocytes and platelets; colorimetric method for the detection of hemoglobin; flow laser cytometry to detect leukocytes. The results for the remaining parameters are calculated.

For clinical analysis, blood is drawn from the vein of the patient on the hungry abdomen to the line marked on the K-EDTA anticoagulant test tube. Examination of blood samples can be checked from 5 minutes to 1 hour. When the analysis is performed 6-8 hours after taking a blood sample, the reliability of the results decreases.

For general blood analysis, the analyzer receives 15 μl or 11.7 μl of blood. The aspirated sample is diluted quickly and accurately with the solvent in the erythrocyte chamber. Dilution of blood samples is necessary to ensure a stable environment in order to calculate and determine the size of blood cells. The sample is then divided into two parts: one is diluted again and treated with different reagents.

In the leukocyte canal, after the stabilization of erythrocyte lysis and leukocytes, a cytochemical reaction occurs, after which the leukocytes differ by two characters: cell size, laser beam scattering, light absorption.

Differentiation of basophils from other granulocytes is done in the negotiation. Cytoplasm of all leukocytes except basophils is lysised after processing the sample with a specific lysate. The scattering of laser beams in the channel is then measured at a 2-degree angle, which allows the cells to be separated according to the shape of the nucleus.

Thus, the use of devices that work with a completely differentiated calculation of blood elements allows to increase the accuracy of the analysis, check the norm and pathology, and dynamically monitor blood changes.

Morphological properties of pathological erythrocytes.

Erythrocyte morphology is examined using augmentation of x1000 times in fatty immersion. This assesses the size, color, shape, color intensity, presence of impurities of erythrocytes. Morphologically normal erythrocyte is called normocyte and has a diameter of 7.2-7.5 μm, with the shape of a double-sided dipped disc; normochrome painted - intensively painted in the periphery of pink cytoplasm, light in the center, no insertions.

1. Anisocytosis is the formation of erythrocytes of various sizes. Typically, in peripheral blood, normocytes are 68–70%, microcytes(less than 6 μm in diameter) 15.5%, and macrocytes (8 μm in diameter) 16.5. When there are a lot of microcytes in

the blood, micrositosis is called macrositosis when there is a lot of macrocytes. Megalocytosis is called when erythrocytes larger than 12 μm in diameter are abundant.

2. Poykilositosis is the formation of erythrocytes in different forms. The shape of the poykylocytes may be different, for example:

Ovalocytes are formed at the expense of membrane defects and are characterized by hereditary ovalocytosis (hemolytic anemia), thalassemia, severe iron deficiency anemia, megaloblastic anemia.

Stomacites are erythrocytes that have an oral-like light zone located in the center of the cell. After blood transfusion, liver disease, infectious mononucleosis, hereditary dentistry (hemolytic anemia) occurs.

Spherocytes are spherical erythrocytes that have lost their double-edged form of boron, with no hungry zone at the center. Sferocytes are called microsferocytes if the diameter is less than 6 μm. Sferocytes hereditary microspherositosis (hemolytic anemia), burns, incompatible blood transfusions, when artificial heart valves are placed, DTII – syndrome (dissiminized intravenous coagulation syndrome) occurs.

Acantocytes are star-shaped erythrocytes. Acantocytes appear in the blood during hereditary acanthositis (hemolytic anemia), lipoproteinemia, liver disease (cirrhosis), during treatment with heparin, after splenectomy.

Exinosites are erythrocytes that have the same tumors in the cytoplasm. Exinosites occur in severe anemia, gastric cancer, ulcers, renal failure, uremia.

Dacriocytesare drop-shaped erythrocytes that are found in myelofibrosis, severe anemia, and toxic liver damage.

N-erythrocytes accumulate hemoglobin in the center and are erythrocytes similar to the target form, which are detected after thalassemia (hereditary hemolytic anemia), severe iron deficiency anemia, liver disease, splenoectomy.

Anulocytesare hollow, ring erythrocytes, and are prone to severe iron deficiency anemia.

Drepanositesare sickle-shaped erythrocytes that occur in hereditary hemolytic anemia in the sickle cell.

Schizophrenic small pieces appear in hemolytic anemia, hemolytic uremic syndrome, DVS syndrome, vasculitis after burns, kidney transplantation.

Degmasitesare helmets of erythrocytes that cause hereditary hemolytic anemia.

3. Anisochromia is the formation of dyed erythrocytes of different intensities. The color of red blood cells depends on the concentration of hemoglobin, the hemoglobin concentration is 32-36% in the norm. Normochromic erythrocytes, usually saturated with hemoglobin, have a pink color. Erythrocyte discoloration:

Hypochromiaare light-painted erythrocytes. Hypochromia of erythrocytes is caused by a low hemoglobin level in erythrocytes and is characterized by iron deficiency anemia, lead poisoning, sideroblastic anemia, thalassemia. Hypochromia is usually accompanied by micrositosis.

Hyperchromy is a dark staining of erythrocytes due to an increase in hemoglobin in erythrocytes. Hyperchromy vitamin B12 deficiency anemia, folic acid deficiency anemia, hereditary spherositosis (hemolytic anemia) specific.

Polychromasia(polychromatophilia) - Occurrence of erythrocytes of different colors: gray-purple, dark gray. These erythrocytes are specific to vitamin B12 deficiency anemia, folic acid deficiency anemia, hemolytic anemia, posthemorrhagic anemia.

4. Additions in the cytoplasm of erythrocytes. Typically, erythrocytes do not contain cytoplasm.

Heints-Erlix bodies are located at the edge of erythrocytes, 1–2 μm of incineration, and consist of denatured hemoglobin. Heints-Erlix bodies are detected in fermopathy.

Bazophilic punctuation is a remnant of mitochondria and RNA in the form of dark blue grain, which is diffuse in erythrocytes. Toxic damage to basophilic punctuation bone burial, such as poisoning by heavy metal salts, radiation therapy, treatment with cytotoxic drug, erythropoiesis activation, megaloblastic anemia, can occur in thalcemia.

Jolly-Gowell bodies are 1-2 μm, reddish-purple, round-shaped DNA residues in the erythrocyte cytoplasm. Jolly-Gowell bodies appear in megaloblastic anemia, hemolytic toxins, after splenoctomy, against the background of erythropoiesis activation.

Kebot rings are the remnants of a red-purple, ring-shaped nuclear shell located in the cytoplasm of erythrocytes. They are detected in poisoning by heavy metal salts, megaloblastic anemia, and leukemia.

Schuffner grain is 20-30 small red-purple point compounds in erythrocytes, detected in three-day malaria. Damaged erythrocytes increase in size and color.

Maurer's spots are large, pinkish-red spots of various sizes, 10–15 points, in erythrocytes in patients with tropical malaria. Erythrocytes do not increase in size and do not change color.

Siderotic grains are granules of non-hemoglobin iron (ferritin, hemosiderin) blue, small (0.5–1.5 micron). Determined by cytochemicaltests.Usually, 0.8-1.0% of siderocytes can be detected in peripheral blood. Increased siderocytes are observed after sideroblastic anemia, myelodysplastic syndrome, against the background of hemolysis of erythrocytes, splenoctomy.

Erythrocytometry.

Erythrocytometry is the measurement of the diameter of painted erythrocytes using a micrometer. Erythrocytometry is performed using an increase of x1000, with maximum illumination of the area. The diameter of 100-200 erythrocytes located in the visible area is measured. The diameter of the erythrocytes obtained in the measurement results is expressed as a percentage. Typically, in peripheral blood, 6-8 μm of normalocytes are 68-70% in diameter, microcytes smaller than 6 μm in diameter are 15.5%, and macrocytes larger than 8 μm in diameter are 16.5.

Reticulocytes

Reticulositosis reflects the level of regenerative activity and erythropoiesis activity of bone coal. Detection of reticulocytes is used in the following cases:

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The amount of reticulocytes in newborns is much higher than in adults. 4 – There is a decrease by the month, but in breast-age children the amount of reticulocytes is slightly higher than in adults. Reticulocytes make up 1% of erythrocytes in the blood. The production of reticulocytes is assessed by a reticulocyte index. The reticulocyte index is the amount of reticuts in erythrocytes in 1000 peripheral blood and it is calculated in the promilli. Norma 2 – 10 ‰ or 0.2 – 1.

RsI (%) = (Number of detoxites / number of erythrocytes) x 100 = 0.2 – 1%

Reticulocytes show the regenerative ability of erythropoiesis. In hemolytic anemia, mainly during the chrysanthemum, the amount of reticulocytes increases dramatically. Reticulocytosis is also observed in polycythemia, malaria, against the background of treatment. The number of reticulocytes increases when some drugs are taken, for example, the amount of reticulocytes increases when taking iron drugs, vitamin B12.

A significant decrease or absence of reticulocytes is a bad sign of anemia. This indicates bone coma injury and loss of regenerator ability (aplastic anemia).

Osmotic resistance of erythrocytes (EOR)

Resistence refers to the resistance of erythrocyte to decomposing agents (osmotic, chemical, mechanical). More osmotic resistance is used in the clinic. In hypotonic solution, the diameter of erythrocytes changes (swollen), the hypotonic solution for erythrocytes is a solution of uqf sodium chloride (NaCI) 0.85% concentration. The minimum osmotic resistance of erythrocytes is checked at the highest concentration of the hypotonic solution, i.e. the most intolerant erythrocyte is equal to 0.55 – 0.46% sodium chloride in the norm in the solution at which it begins to crack. Maximum osmotic resistance is the rupture of all erythrocytes at low hypoosmolar concentrations of sodium chloride. It is 0.34 – 0.28% in adults.

In microspherositar hemolytic anemia, the osmotic resistance of erythrocytes is significantly reduced. Decreased EOR is also observed in hemolytic disease in infants, toxicoses, bronchopneumonia, tuberculosis, malaria, leukemia, liver cirrhosis. EOR increase is observed in drepanositar anemia, mechanical jaundice.

Determination of osmotic resistance of erythrocytes.

To do this, 6 probes are numbered and placed in the tripod, and a reduced concentration of NaCI solutions are prepared.

Переводчикбот, [19/01/2023 17:09]

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In microspherositar hemolytic anemia, the osmotic resistance of erythrocytes is significantly reduced. Decreased EOR is also observed in hemolytic disease in infants, toxicoses, bronchopneumonia, tuberculosis, malaria, leukemia, liver cirrhosis. EOR increase is observed in drepanositar anemia, mechanical jaundice.

Determination of osmotic resistance of erythrocytes.

To do this, 6 probes are numbered and placed in the tripod, and a reduced concentration of NaCI solutions are prepared.

Probation numbers	1	2	3	4	5	6
1% NaCI, ml	1,8	1,5	1,35	1,2	1,05	0,9
Distilled water, ml	1,2	1,5	1,65	1,8	1,95	2,1

Solution concentration %	0,6	0,5	0,45	0,4	0,35	0,3

Each test tube is filled with 40 μl and the mixture is left in the strain for one hour or centrifuged at a speed of 3000 revolutions for 5 minutes. The result is determined by the color of the solution and the erythrocytes that fall into the precipitate.

Hemoglobin

The main protein in erythrocytes – is hemoglobin, which is 98. It consists of gem, i.e. protoporphyrin attached to an iron atom, and globin, i.e., a protein holding four polypeptide chains. Of the four polypeptide chains in a normal human body, two are species a – and the other two are of the other species (β ,γ, or δ). a – chain on 16 chromosomes, the remaining chains are coded on 11 chromosomes. The high solubility of hemoglobin depends on the fact that the tetramer in the globin consists of different pairs of chains. If the tetramer consists of the same type of chain, it undergoes rapid denaturation, which leads to a reduction in the life of erythrocytes (hemoglobinopathy). In the blood, hemoglobin is in the form of oxyhemoglobin(the oxygen compound of hemoglobin) and reduceratedcarbgemoglobin (, which gives oxygen to tissues and binds carbon dioxide to hemoglobin). Oxigemoglobin occurs in arterial blood and gives it a light red color. Venous blood contains carbhemoglobin and gives it a dark red color. 1gr Hb contains 1.34 ml of oxygen – Geftnercoefficenti. Each tetramer uzi can attach 4 malukula O2 with a turtta iron atom.

Normal Hb fractions:

- Hb A – 97%
- Hb A2 - around 2
- Hb F – around 1

The newborn has 140 – 190 g / l Hb in umbilical cord blood. After a few milks, the amount of Hb rises to 165 – 225 g / l. 15 – decreases to normal by 30 days. 2 – Decreased to 100 – 130g/l by 3 months. 1 year old is 120 g / l. In children born to premature, the amount of Hb decreased to 80 -100g/l in 2 – 3 months. But this is not pathologically calculated, as the amount of Hb normalizes when you are 1 year old.High hemoglobin levels in the norm are observed in people living in mountainous areas, pilots after flight, climbers, after severe physical stress. A significant increase in Hb concentration is observed when erythrocyte levels increase (truthpolycythemia).

Decreased Hb levels are called oligochromia and are the main symptom of anemia. A sharp decrease in hemoglobin levels is observed after aplastic anemia and massive blood loss.

The amount of Hb in the blood plasma. Norma has traces of Hb in plasma and does not exceed 10 mg. Because plasma concentrations of Hb increase when intravenous hemolysis is observed. This condition is observed in immune hemolytic anemia, drepanocytosis, a sharp increase in hemoglobinuria. In microspherocytosis, the concentration of Hb does not change because hemolysis occurs within the cell.

Other forms of hemoglobin can also be formed in the body: methemoglobin, carboxyhemoglobin.

Methemoglobin – Fe++ atom becomes Fe++ +. Erythrocytes are always produced in small amounts of methemoglobin during the exchange process, which is (0.03 – 0.3 g /%), with a total content of 2. However, it is regenerated under the influence of the enzyme methemoglobinreductase.

Methemoglobinemia is observed in the following cases:

1. congenitalmethemoglobinemia:
   - enzymopenic congenital methemoglobinemia (Djibon) – methemoglobinreductase enzyme deficiency
   - congenital hemoglobin M methemoglobinemia (Herleyn, Veber) – hemoglobin M occurs with the appearance. Methemoglobin levels increase to 0.7 – 7.5g/%, and it accounts for 5 – 60% of total Hb.

2. Stockvis – Talm syndrome is an enterogenousmethemoglobinemia caused by endogenous and bacterial toxins/

3. poisoning (nitrites, nitrates, nitrobenzene, aniline derivatives, salicylates, PASK). Methemoglobin levels rise to 6 g. Methemoglobin is determined spectroscopically. The use of freshly obtained blood for analysis is necessary.

Determination of hemoglobin levels.

The hemoglobinometer is heated. 5 ml of hemoglobin cyanide solution is poured into the test tube and 20 μl of patient blood is added. 3 – Stir for 5 minutes. The resulting mixture is poured into a special cuvette of a hemoglobinometer and is looked at using photometry.Color indicator

This is a relative indicator, indicating the amount of hemoglobin in one erythrocyte. Norma 0.86 – 1.05. The color indicator is calculated by the following formula:

Color indicator = Hb X 0.03 / Number of places (mln/mm3)

When the color index is 1.0, the amount of Hb in one erythrocyte is 33.34 pg. (MCH (mean corpuscular hemoglobin – Average amount of Hb) =33.34 pg/cell). When the color is high, the volume of erythrocytes also increases. Therefore, in hyperchromy, the creation of a megaloblastic type of blood is observed, i.e., an increase in the average diameter of red cells is observed. If the hemoglobin level in erythrocytes exceeds 100 pg, erythrocyte ruptures.

There are two main causes of hypochromia:

1. Erythrocytes shrink in average diameter.
2. Insolvency of erythrocyte cytoplasm with hemoglobin.

Erythrocyte sedimentation rate

3 phases differ during erythrocyte deposition:

1. Under the influence of the gravitational force of erythrocytes, erythrocytes become a cell in aloxia and begin to sink slowly.

2. After a certain time, the drowning accelerates and the erythrocytes start to sink in the form of agglomerates. The larger the agglomerates, the faster the erythrocytes will sink.

3. By this phase, the drowning slows down again. The agglomerates thicken and then their drowning becomes difficult and the drowning stops.

Erythrocyte sedimentation rates are affected by erythrocyte volume, hemoglobin concentration within erythrocytes, blood pigments and salts, CO2 concentration, plasma viscosity, and most importantly, changes in the amount and quality of proteins in the blood. Albumins protect erythrocytes from agglomeration. In inflammatory and tumor diseases, the amount of globulins increases and erythrocyte subsidence accelerates.

In infants, the rate of erythrocyte drowning is slowed because they have low levels of globulins in the blood. After 4 weeks, the rate of erythrocyte drowning is relatively rapid, and this condition lasts until the age of 4 years. This condition is associated with physiological anemia because a decrease in the number of erythrocytes increases the rate of erythrocytes sinking and a decrease in proliferation.

ESR mm/ h = 42 – 7.5 x erythrocyte count

In addition to the increase in leukocyte count in infectious and inflammatory diseases, the ESR also increases, indicating the severity of the disease. The ESR increases a few days after the leukocyte count increases or after the devastation rises. Because it will take some time for the protein spectator to change. In some infectious diseases, an increase in (influenza, tonsilitis) ESR is observed after improved patient follow-up. In acute rheumatism, the ESR is a clear indicator of the activity of the inflammatory process. Increased ESR is also observed in kidney disease, mainly nephritis.

In paroproteinemia, a sharp increase in ESR is also observed in (mieloma, macroglobulinemia, atypical leukemia, cryoglobulinemia). Although dysproteinemia is observed in heart disease, a slowdown in ESR is observed, which is associated with polycythemia and increased CO2 concentrations in the blood. In anemia, the ESR will be slightly accelerated. This condition is associated with a decrease in erythrocyte levels. But microsferocytes and drepanocytes are the opposite in anemia because such pathological shakils of erythrocytes prevent their agglomeration.

Myelogram

Bone fat is taken by a doctor under aseptic conditions using a puncture. The cell composition of bone coal depends on its peripheral blood, the condition of the bone coma, the age of the patient.Myelogram is the ratio of bone burial cells. 500-1000 cells are analyzed to calculate the myelogram. All cells in the field of view are counted. In the myelogram, the granulositar series cells make up 60-70%, the erythroid series cells 20-25%, the lymphocytes 7-10%, and the monocytes about 2. The myelogram also includes plasmatic cells, megacariocytes, fat cells, macrophages, osteoblasts, and osteoclasts.

In evaluating the myelogram, the cell level of the bone coma is first assessed as (multicellular, low-cell, normal cell), followed by the cells of each row of bone coal.

Control questions:

1. Cytological diagnostic methods in hematology
2. Methods of quantitative and qualitative study of blood cells
3. Morphology of erythrocytes
4. Anisocytosis
5. Poykilositosis
6. Anisochromia
7. Additions in the cytoplasm of erythrocytes
8. Erythrocytometry

9. Reticulosites

1.2. NORMAL HEMATOPOIESIS. HEMATOPOIETIC FACTORS.

The purpose of the training: to introduce hemopoiesis, bone burial, red bone coma blood-forming barriers, hemopoietic cell classes, blast-specific symptoms, hemopoeia regulation, factors necessary for normal erythropoes.

Hemopoeis is a system that ensures that blood-creating cells are constantly renewed. During the hyemopoeia, blood cells - leukocytes, erythrocytes and platelets - are formed, fed and broken down non-stop. Currently, the hemopoiesis hierarchical model has been approved, according to which blood cells are formed from hemopoieticmultipotent stem cells. The main member of hemopoiesis is bone coal, which has the following types:

1. Red bone coal (consists of hemopoietic cells).
2. Yellow bone coal (adipose tissue).

Red bone coal serves as the only member of blood formation from 20 weeks of fetal activity. It is located in the epiphyses of skeletal flat bones and larynx bones. There are several blood-forming barriers to red bone coal:

1. Erythrocytes - produces erythrocytes.
2. Myelocitar produces series - eosinophil, neutrophil and basophils.
3. Lymphocytar series - produces lymphocytes.
4. Monocitar series - produces monocytes.
5. Myegakariocitar series - produces platelets.

Hemopoieticpolypotent oral cells are class I cells from which all series of cells of the hemopoeia are formed under the influence of cytokines. Under the influence of cytokines, the stem cells become class II cells - colonies of polypotent cells - granulocytar - erythrocyte -macrophagal - megacariocytar colony-forming units and lymphocytic colony-forming units. Under the influence of cytokines, polypotent cells are class III oligopotent reached to the cells. Granulocytar - erythrocyte -macrophagal - megacariocytar colony-forming units are converted into 3 different types of cells: granulocyte and monocyte colony-forming unit, erythrocyte colony-forming unit, and megacariocyte colony-forming unit. These processes are controlled by leukopoietin, erythropoietin, and thrombopoietin.

Class IV cells are blastes in which only one row of cells is formed: lymphoblast, monoblast, myeloblast, erythroblast, megacarioblast. During the lymphoblast differentiation process, the prolymphocyte (V class) and the lymphocyte (VI class) stages are passed. Monoblast forms promonocyte (V class) and monocyte (VI class). Myeloblast ingestion results in eosinophil, basophil or neutrophil promyelocyte, myelocyte, metamielocyte, rod-core (V class) and segment-core (VI class) leukocyte stages.Erythroblast pronormocyte, basophil, polychromatophilic and oxyphilicnormoblast, reticulocyte (V class) and erythrocyte (VI class) are differentiated. Myegakarioblast is converted to megacariocyte (V class), and platelets are separated from the cytoplasm of the megacariocyte (VI class).

The cell composition of the bone marrow is evaluated by puncturing the skull or lateral bone and counting the myelogram.

Blast-specific features: nucleus large, nucleus-cytoplasmic ratio 1:4-1:8. cell cytoplasm occurs from light aphids to dark basophils, there is no perinuclear hungry area

around the nucleus, there is no graininess in the cytoplasm. The chromatin structure of the blast nucleus can be thin-cross, 1-2 nuclei.

Based on the myelogram, diseases such as acute and chronic leukemia, anemia, thrombocytopenia, lymphogranulematosis, tuberculosis, Goshe disease, Niman-Pik disease, tumor metastases, visceralleishmaniosis can be diagnosed. However, it is also important in evaluating the effectiveness of the therapy.

Hemopoeic regulation

Blood creation is governed by the following factors:

□ growth factors - erythropoietin, leukopoietin, thrombopoietin;

《cron trace elements, vitamins, hormones (eritropoietin, thyroxine, androgen, corticosteroids, growth hormones).

Growth factors include colony stimulants, interleukins, and inhibitory factors. Almost all growth factors affect the cells that make up the Cossack and the colony. Necessary for normal erythropoiesis:

1. Hormones that control protein metabolism (pituitary somatotropic hormone, thyroxine, and b.)
2. Calcium metabolism-controlled hormones (paratgormone, tireocalcitonin)
3. Androgens stimulate erythropoiesis, while estrogens brake.
4. Erythropoietin. Most erythropoietin is formed in the kidneys. Its formation is associated with renal circulation and oxygen deficiency. A decrease in erythrocyte count and a decrease in oxygen parsial pressure are the basis for an increase in erythropoietin production. Erythropoietin production is reduced in chronic kidney disease.

Thrombopoietin is synthesized in the liver, enhances the proliferation and differentiation of the megacariocytar series cells that form the colony, and enhances platelet formation.

5. Micronutrients (iron, copper, zinc, selenium and b.). Iron is necessary for the synthesis of hemoglobin by entering the gem. Iron deficiency in the body develops in alimentary causes, gastrointestinal disorders, increased iron demand (pregnancy, sportsmen), bleeding. Copper erythrocytes play an important role in the diet.
6. Vitamins. Folic acid, vitamins B12, B6, B2 and C are necessary for blood formation. Vitamin B12 and folic acid are involved in the synthesis of nucleic acids in erythroblasts and enhance their proliferation.
7. Cytokines (interleukin 1, 3, 6, 11 and 12, tumor necrosis factor)polypotent is involved in the differentiation of stem cells. Inhibiting factors weaken the production of hemopoietic cells. As a result of a lack of these factors, leukemia, an increase in leukocytes in the blood, develops. Lyceum inhibitor monocyte - brakes the proliferation and differentiation of macrophages

Control questions:

1. Gyemopoez.
2. Bone coal.
3. Cells of hyemopoietic classes.
4. Gyemopoez barriers.
5. Development of gyemopoez barriers.
6. Gyemopoeous regulation
7. Necessary substances for hyemopoiesis

1.3. LABORATOR DIAGNOSTICS OF ANEMIES.

Objectives of the exercise: anemia, classification of anemia, changes in blood and bone burial in iron deficiency anemia, changes in blood and bone burial in megaloblast anemia, changes in blood and bone coma in acute posthemorrhagic anemia, introduction to changes in blood and bone burial in hemolytic anemia, changes in blood and bone burial in hypo-, aplastic anemia.

Anemia is a group of clinical-hematological syndromes, the common symptom of which is characterized by a decrease in the amount of hemoglobin and erythrocytes in the blood. Hb for men is less than 130 g /l, land is less than 4x1012 /l, Hb is less than 120 g /l for women and Earth is less than 3.5x1012/l.

 False anemia oligocythemic hypervolemia;

 Actual anemia oligocythemic hypo – or normovolemia. This reduces hematocrit.

Hemoglobin in erythrocytes transports oxygen from the lungs to the tissues and carbon dioxide from the tissues to the lungs. Patients with anemia develop symptoms of oxygen deficiency - hypoxia in the tissues. In mild anemia, patients are disturbed by general weakness, rapid fatigue, attention deficit. In severe anemia, a slight physical strain is added to the sigh, palpitations, headache, dizziness, ear noise, appetite disorders. Heart failure is added in severe anemia, especially when the patient is pathologically. Increased anemia is characterized by an increase in skin and mucous membrane palate.

Classification of anemia:

1. Erythrocyte size:

□ micrositar anemia (iron deficiency anemia)

□ macrocytar anemia (vitamin B12tan deficiency, follic acid deficiency anemia)

《Normositar anemia (hemolytic anemia, aplastic anemia, metaplasticanemia)

2. By color indicator.

Color index indicates that erythrocyte is saturated with hemoglobin. In Norma, RK is 0.85-1.05. Types of anemia depending on it:

1. Hypochrome anemia (color index 0.85 and less):

□ iron deficiency anemia;

□ thalassemia.

2.Normochrome anemia (color index0.85-1.05):

□ hemolytic anemia (erythrocytes due to multiple breakdown);

□ posthemorrhagic anemia ( ) due to excessive bleeding;

□ acute and chronic leukemia, lymphomas;

□ aplastic anemia;

Tape metastasis in bone fat;

□ Anemia developed due to decreased erythropoietin production.

3.Hyperchrome anemia (color index more than 1.1):

□ vitamin B12-deficiency anemia;

□ follic acid deficiency anemia;

Refractory anemia in □odysplastic syndrome.

3. By weight:

– Mild anemia - hemoglobin 90-120 g / l.

– Moderate to severe anemia - hemoglobin 90-70 g / l.

– Severe anemia - hemoglobin less than 70 g / l.

4. According to the regeneration function of bone coal:

The main sign of regeneration of bone burial is an increase in reticulocytes in peripheral blood. Reticulosites in Norma - 1-10‰.

《generator (aplastic anemia) - sharply reduced reticulocytes;□ hyporegenerator (vitamin B12 deficiency anemia, iron deficiency anemia) - decreased reticulocytes;

《normoregenerator or regenerator (posthemorrhagicanemia) - reticulocyte levels are normal.

□ Hyperregenerator (hemolytic anemia) - The number of reticulocytes increases sharply.

5. Etiopathogenetic classification.

□ Chronic anemia: tuberculosis, bacterial endocarditis, bronchoectatic disease, pulmonary abscess, brucellosis, pyelonephritis, osteomyelitis, collagenoses(systemic red boricha, rheumatoid arthritis, and b.).

□ Iron deficiency anemia. The amount of iron consumed or lost in iron deficiency anemia is greater than the amount of iron received.

□ Megaloblast anemia: vitamin B12 deficiency anemia, follic acid deficiency anemia. Lack of vit B12 and folatekislata results in megaloblastic anemia as a result of DNA synthesis disorders.

□ Hypo-, aplastic anemia.

□ Metaplastic anemia: leukoses, metastases of malignant tumors.

□ Posthemorrhagic anemia - as a result of bleeding:

?? sharp - 5 ml/kg of volume and more of blood is observed in case of loss;

?? develops after chronic – long-lasting and recurrent blood losses.

1. Hemolytic anemia is the predominance of the erythrocyte decomposition process from a hemopoeia. Erythrocyte lives in the norm for 120 days. In hemolytic anemia, erythrocyte occurs when the life is 18 days and shorter. In this case, normal bone coal boosts erythropoiesis up to 7 times. According to the localization of hemolysis:

?? vein:

mechanical or mechano – chemical thrombosis damage under the influence of microbial thrombus in the microcircular flow (Sunni heart valves and vascular prostheses)

complimentarylysis (non-compliant blood transfusion). But the compliment can break down the Earth even without antieritrocytarantitanas. Because SR1 is a receptor on Earth and in the physiological state a compliment is attached to it, which protects it from its litic effects. When this connection is broken, the Earth becomes unstable to the compliment (paroxysmal nocturnal hemaglobinuria).

?? intracellular – in which the Earth becomes anomalous or damaged from the beginning and loses its ability to deform.

Iron deficiency anemia

Iron deficiency anemia is the most common, accounting for 80% of anemia. Cytological features of iron deficiency anemia:

1. In peripheral blood:

□ Decreased erythrocyte and hemoglobin;

□ Erythrocyte microsytosis - (6 μm and shrinkage);

《crythrocytehypochromia - pale color;

□ Erythrocyte raccalocytosis - a change in shape.

2. In the myelogram, the creation of a normoblastic type of blood, erythroid series hyperplasia is observed.

Megaloblast anemia

Megaloblast anemia includes vitamin B12 and folic acid deficiency anemia, and their cytological features are:

1. In peripheral blood:

Decreased □ erythrocyte and hemoglobin;

□ erythrocytemacrocytosis (9-12 μm), megalosytosis (12 μm magnification);

《crythrocytehyperchromy - discoloration of color;

Change of form;

《bodies (core residues);

《cabolt rings (core membrane);

Hypersensitivity of nuclei neutrophils in the □ segment - 5 and more of the segments;

Decreased regression.

In severe anemia:

The emergence of megaloblasts;

Decreased platelets, increased macroplastins;

《cromaphilia - the appearance of polychromatophyll-painted erythrocytes;

Occurrence of □ myelocytes and metamielocytes;

《galocytes increase in reticulocytes as a result of the addition of hemolysis in spleen sinuses as the number of megalocytes increases.

2. In the myelogram, the creation of a megaloblastic type of blood, erythroid series hyperplasia is observed.

Acute posthemorrhagic anemia

Acute posthemorrhagic anemia is characterized by excessive bleeding in a short time. The results of objective examination and instrumental examination are of great importance in the diagnosis of acute posthemorrhagic anemia.In the cytological diagnosis of this type of anemia, the time elapsed after bleeding is of great importance:

1. **In peripheral blood:**

《Normochrome, normositar anemia is observed after bleeding;

《After 4-5 days the number of reticulocytes increases, polychromaphilia - polychromatophilic-painted erythrocytes, nuclear erythrocytes - normocytes appear;

《 After 10 days, symptoms of iron deficiency anemia develop (erythrocyte microsytosis, hypochromia, zucilocytosis).

2. Normoblastic type blood formation in the myelogram, erythroid series hyperplasia is observed after 4-5 days.

Hemolytic anemia

Hemolytic anemia is congenital and acquired. Cytological features specific to hemolytic anemia:

1. **In peripheral blood:**

Decreased □ erythrocyte and hemoglobin;

□ erythrocytenormochromia (erythrocyte hypochromia and hyperchromy in microspherositosis are observed only in thalassemia);

□ erythrocytenormocytosis (erythrocyte diameter decreases only in microspherositosis);

□ reticulocytes increase;

In congenital hemolytic anemia, the shape of erythrocytes changes:

□ small 5–6 μm, hyperchrome erythrocytes appear in microspherosytoz;

Oval eryrocytes appear in ovalosytoz;

《creatic erythrocytes appear in the agene acanthositis;

《citrocytes of oral hypochrome zone appear in dentistry;

In , the form of erythrocytes does not change normally, only in the case of strong hypoxia is hemolytic chrysanthemums, and sickle erythrocytes - dacriocytes appear;

《plate, hypochrome erythrocytes - codocytes appear in thalassemia.

In hemolytic crisis:

《s are formed by large amounts of unripe nuclear normalocytes;

□ reticulocyte content exceeds 30.

2. In the myelogram, the creation of a normoblastic type of blood, erythroid series hyperplasia is observed.

Abnormal shapes of erythrocytes by shape, size, structure, color and their diagnostic significance (Rouz, Berliner, 2000)

Ername	***Clinicalsignificance***
According to shape,size,color	
1. normocyte (erythrocyte)	In normal and aplastic anemia
2. polychromatophilic cell	Normally up to 1 %,in polychromatophilia-reticulocytosis
3. macrospherocyte	In the megaloblastic state
4. microspherocyte	In the abnormality of plasma membrane lipids
5. microsite	Inhypochromic anemias
6. microspherocyte	In hemolytic anemia wih hereditary microspherocytosis and intracellular hemolysis
7. echinocyte	Uremia,hypophosphatemia,hypomagnesemia,pyruvate kinase deficiency,gastric cancer and ulcer,bloodtransfusion,artifact during blood smear preparation
8.acanthocyte	HypovitaminosisE,hyposplenism

9. degmasites	Unstable hemoglobins,glucose 6-phosphate dehydrogenase deficiency
10.vesicularcells	Immunehemolytic anemia
11. elliptocyte	Hereditary elliptocytosis,thalassemia,megaloblastic condition
12. stomatocyte	Hereditary stomatocytosis and spherocytosis,liver pathology
13.codocyte (target cell)	Thalassemia,hyposplenism,hemoglobinopathy,liverdiseases,iron deficiency
14.schistocyte(squamous cells)	Hemolytic anemia with intravascular hemolysis
15.sickle (sickle cells)	Sickle cell anemia,HbS gene carriers,thalassemia
16.anulocyte (ring cell)	Whenthere is hypochromia
17.dacryocyte (droplet cell)	Myelofibrosis,thalassemia
Inclusionsfound inside erythrocytes	
18.nucleus(erythrokaryocyte)	Indicative of a hyperregenerative process
19. Jollybody	Characteristic of nuclear,remnant,hyposlenism,megaloblastic anemia
20.basophilic punctation	Thalassemia,megaloblastic and sideroachrasticanemia,poisoning
21. Pappengame corpuscle	Fe^{+3} granules,hyposplenism,sideroacrestic,hemolyticanemias
22.Cabot ring	Characteristicof megaloblasticanemias
23. HbScrystals	S – hemoglobinopathy
24.Schiffner pellets (earworm,banana-shaped parasites)	Malaria

25. Gaines' corpuscle	Normally it is up to 4.Characteristic of enzymopathies

Poyctlocytosis – erythrocytes given at different levels. Occurs in severe anemia. However, razylocytosis is not observed in aplastic anemia.

Anisocytosis – zritrocytes of various forms.

1.4. TROMBOSITOPOEZ. TROMBOSIT QATOR PATHOLOGY LABORATOR DIAGNOSTICS.

Objectives of the training: to get acquainted with thrombocytopoiesis, method of counting platelets, thrombocytosis, thrombocytopenia, cytological examination of the thrombocytic series pathology of blood and bone coma.

The process of platelet formation in the body is called thrombocytopoiesis. The mother cell of platelets is a megacariositar cell.

Megacariositar cell elements are formed, differentiated, and mature from the myeloid front cells in the bone burial. Megacariocytopoiesis main stimulants: IL-1, IL-3, IL-4, IL-6, IL-11, collon stimulants, erythropoietin, thrombopoietin.

Thrombocytopoiesis is based on the reverse garden 'princip: an increase in platelets in the blood stops thrombocytopoiesis, while thrombocytopenia stimulates platelet formation. In bone burial, the megacariocyte cell undergoes several stages of morphological differentiation: megacarioblasts, promegacariosites, and megacariosites. 75-85% of megacariositar series cells are megacariocytes, 10% are megacarioblasts, and 15% are promegacariosites.

Megacariocyte is a giant polyploid cell with a diameter of 60-120 μm. Megacariocyte is a large cell with a polymorphic nucleus, broad, pink cytoplasm, which preserves platelets.

The main function of megacities is to form platelets and keep their number constant. Up to 5,000 platelets are separated from a single megacariocyte. In the norm, 60-70% of megacariocytes are active, i.e. they form platelets. About 80% of platelets are in the blood and 20% in the spleen. Platelets live 7-8 days.

Without thrombocystidro, it is a cell with a diameter of 2-4 μm, involved in hemostasis and blood anxiety. The number of platelets in a healthy person is 180-320x109/l.

Platelets are round and oval in shape, the cytoplasm is composed of light purple painted gialomer and central pink - purple granulomer parts.

Thrombocyte functions:

1. Angiotrophic: nourishes the vascular wall and ensures its strength.
2. Adgesia: A thrombocytic formed in primary hemostasis attaches to a damaged vascular wall.
3. Aggregation platelets stick together.
4. Lacta retraction: The platelets are attached to each other, resulting in a shortening of the blood clot and a thrombus.
5. Produces vasoconstrictors to reduce bleeding.

Thrombocytosis is an increase in the number of platelets in the blood, and thrombocytopenia is a decrease in the number of platelets.

Thrombocytosis and thrombocytopenia species:

1. Primary (absolyut) thrombocyte count exceeds 400x109/l, and the activity of megacariositar series cells in bone burial increases. Primary (absolyut) thrombocytosis occurs in the following cases:

a. Megacariositar leukemia (essential thrombocythemia)
b. In Eritrea
c. In chronic myeloley
d. Inmyelofibroz

2. Secondary (absolyut) thrombocytosis may occur:

a. When eating
b. In the morning
c. After bleeding
d. Asphyxia
e. In hemolysis
f. On fire
g. Insarcoidozda
h. After the practice of cirrhosis
i. Aftersplenectomy
j. After treatment with corticosteroids
k. In chronic inflammatory diseases (rheumatoid arthritis, nospesific ulcerative colitis, tuberculosis, osteomyelitis)
l. Poor quality tumors

3. Causes of relative thrombocytosis:

a. Dehydration
b. Bleeding

Dangerous clinical signs of thrombocytosis are formed at the level of platelet concentrations 700-900x109/l. Thrombocytosis can occur in thrombocytoses, thromboembolism can occur.

Absolute thrombocytopenia is the fact that platelet count is less than 150x109/l. Clinical manifestations of thrombocytopenia are observed when reduced from 70x109/l. Absolute thrombocytopenia occurs in the following cases:

1. Thrombocytopoese hereditary pathology
2. Immune thrombocytopenia (autoimmun)
3. Blood diseases (aplastic, megaloblast anemia, leukemia, paroxysmal nocturnal hemoglobinuria)
4. Severe bleeding
5. Injury to bone coal (in metastases, tuberculosis, radias)
6. hemalitic - uremic syndrome
7. renal insufficiency
8. liver diseases
9. stroke, spleen, tumors
10. eclampsia
11. hyperteriosis, hypothyroidism

12. Infectious diseases (virus, bacteria, ricketsiosis, malaria, toxoplasmosis, human immunodeficiency syndrome)
13. Eclampsia in pregnancy
14. Take a look
15. Effects of drugs (cytostatics, analgetics, antihistamines, antibiotics, etc.)
16. Alcohol, heavy metals poisoning
17. Hypersplenism, dissiminized intravenous coagulation syndrome, after hemodialysis.

Morphology of platelets

When dyed in the blood of a healthy person in the Romanovsky-Gimza method, mainly 4 different platelets are distinguished:

1. Edited platelets are 90-95%, round or oval in shape, 3-4 μm in diameter, gialomer and granulomer are clearly distinguished.
2. Young unripe platelets are 0-1%, measuring 4-6 μm.
3. Old platelets are 2-6%, 2-3 μm in size, with a thin cytoplasm in the form of dumol, oval, tooth.
4. Damaged, degenerative platelets are 0-1%, large-sized, long-tailed, blue or pink cytoplasmic, azurophil-grained, vacuolysated cell.

Methods of counting platelets

1. Phonio method
2. Counting in Goryayev's cell
3. Counting on an electronic automatic hematological analyzer

Determination of platelet count by the phonio method

1. Panchenkov capillary “25 to ” 14% magnesium sulfate solution or 6% ethylenediamintetraacetate (EDTA) is obtained and poured into the test tube.
2. The blood from the finger is taken to the K line of the Panchenkovcapillium and placed in the test tube.
3. The test tube is mixed and greased from it, fixed and painted in Romanovsky-Gimza method.
4. The platelet count in an area enlarged by 1000 times is calculated relative to 1000 erythrocytes (‰).
5. Knowing the number of erythrocytes in the blood at 1 μl and the number of erythrocytes in the blood is 1 μl based on the formula.

Thrombocyte (x109/l) =Erythrocyte x thrombocyte ( )

The platelet count by the Fonio method in Norma is 45-70 ‰ compared to a thousand erythrocytes‰.

Determination of platelet count in Goryayev's cell

1. Probiraka is filled with 1% - 4 ml of ammonium axolate solution
2. The test tube is 20 μl of blood, mixed gently and placed for 25-30 minutes for erythrocyte hemolysis
3. After re-mixing, the solution is poured into Goryayev's chamber
4. The number of thrombotsites is counted in 25 large squares
5. The number of platelets is calculated by formula

calculated platelet count x 2000

Count the platelet count on the automatic analyzer

In modern hematologic analyzers, platelets are seen in sizes in the range of 2-30 fl. Automatic analyzers cell size,evaluates structures, cytochemicals, and other sensations, analyzes approximately 10,000 cells in a single sample.

Control questions

1. Thrombocytopie
2. Megocariocite sensation
3. Platelets
4. Platelet functions
5. Changes in platelet count
6. Plate morphology
7. Methods of counting platelets
8. Determination of platelet count by the phonio method
9. Determination of platelet count in Goryayev's cell
10. Count the platelet count on the automatic analyzer

1.5. Leukopoez. Leukopoez regulation. Leukopoietic factors.

Laboratory diagnostics of leukopenia, leukocytosis and leukemoid reaction.

The purpose of the training: introduction to leukocytes, granulocytes, agranulosites, granulositopoiesis, monocytopoiesis, lymphocytopoiesis.

Leukocytes are nuclear-bearing blood cells that vary drastically in appearance and function. Leukocytes protect the body from external and internal pathogenic factors. The total amount of leukocytes is 4-9x109/l.

According to Romanovsky – Gimza method, 2 types of leukocytes differ in the dye of granules:

1. Granulosites. There are special granules in the cell cytoplasm that include neutrophils, eosinophils, and basophils.
2. Agranulosites. There are no special granules in the cytoplasm. These include monocytes and lymphocytes.

Leukopoez consists of granulositopoiesis, lymphocytopoiesis, and monocytopoiesis.

Granulositopoez

Granulositopoiesis cells, which form a colony from the anterior cells of myelopoiesis, appear in the bone burial and become basophils, eosinophils, and neutrophils granulocytes as a result of maturation. Types of granulocytes in bone coal:

1. Proliferative cells - myeloblast, promielositis, myelocyte.
2. Solvent cells are myelocytes, rod-core and segment-core neutrophils.

In the granulopoez regulation, the colony stimulant granulositar factor (GM-KSF) and the granulomonositar factors (G-KSF) are involved.

The diameter of the beak core neutrophil is 12-16 μm. Nuclear-cytoplasma ratio 1:1. The core is pinkish-purple in color, the shape is rod-shaped, the chromatin structure is large-piece, dense, the core is non-existent. Cell cytoplasm has a pink color, neutrophil grain.

The diameter of the neutrophil nucleus with segmental nucleus is 12-16 μm, the cell nucleus is red-purple, the nucleus is cytoplasm ratio is 1:6-1:8. The chromatin structure is large. Cell cytoplasm has a pink color, neutrophil grain.

The main functions of neutrophils are:

1. Phagocytosis

2. Dezintoxication
3. Calling for an inflammatory reaction.
4. Participation in the breeding of leukocytes.
5. Participate in blood clotting

In the norm, rod-core neutrophils in peripheral blood are 0-6%, segment-core neutrophils are 47-72%.

Eisinophils are round cells with a diameter of 12–16 μm, with a cytoplasmic ratio of 1:1. The core is dark purple, usually consisting of two segments, the structure of the chromatin is uneven, large-breed. Cytoplasma has oxyphilic, large yellow-pink-colored special granules. Eisinophils are present in the blood for 6-12 hours, then pass into the tissues. Eisinophils live 4 to 30 hours. In Norma, 0-5% of leukoformula are eisinophils.

Functions of eosinophils:

1. Limitation of allergic reactions.
2. Anthelment is the formation of immunity.
3. Phagocytosis.
4. Participate in the inflammatory process.
5. Participate in blood clotting.

Basophils occur in the norm of leukocytar formula 0-1. Their function:

1. Improving vascular permeability.
2. Limitation of allergic reactions.
3. Participate in the anti-assum process.
4. Anti-inflammatory effect.
5. Participate in blood clotting.
6. Participation in triglyceride metabolism.

Monocytopoez

Monocytes and macrophage cells in bone coal, blood, and tissues are integrated into a system of mononuclear phagocytes. Immature cells of the mononuclear phagocyte system are formed from polypotent stem cells. As a result of maturation, these cells are converted into macrophages colony-forming cells and monoblasts. Monocitar series cell stimulants(IL-3, GM-KSF, M-KSF) and inhibitors (interferon alpha, betta, prostoglandins, IL-10) are present.

Monocytes are a round-shaped cell with a diameter of 18-20 μm. Nuclear - cytoplasmic ratio 1:1. The monocyte nucleus is bean-shaped, renal, segmented, or rod-shaped, eccentric in location. Chromatin structure is sparse. Monocyte cytoplasm is broad, airgray.

Functions:

1. Phagocytosis.
2. Creating special immunity.
3. Participation in reparative processes.
4. Hemopoeic regulation.
5. Participation in metal metabolism (iron, copper, zinc).

The number of monocytes in leukoformula in Norma is 3-11%. Absolute number 0.09-0.60x109/l.

Lymphositopoeia

Lymphocytes are formed from the cell in front of the lymphocytopoiesis in the bone burial. B-lymphocytes are completely removed from the bone burial and have antigen-dependent differentiations. T-lymphocytes migrate to the thymus and are given there. Inoculated T-lymphocytes accumulate in peripheral lymph nodes.

Lymphocyte is a round cell with a diameter of 9-15 μm. Cell nucleus - cytoplasmic ratio 4:1-8:1, nucleus round, chromatin structure rough, fragmented, cytoplasm basophilic, thin. In morphology, lymphocytes are small, medium, and large.

In the norm, the amount of lymphocytes in the leukositar formula is 19-37%, the absolute number is 1.2-3.0x109/l.

Lymphocyte function:

1. It becomes a plasma cell and produces antitelo
2. Cytotoxic effects against meat, cancer cells, viruses, simple animals
3. Stores information about antigens

Control questions:

1. Leukocytes
2. Granulositopoez
3. Neutrophil granulocytes
4. Eosinophilic granulocytes
5. Basophilic granulocytes
6. Monositopoez
7. Monocyte
8. Lymphositopoeia
9. Lymphocyte

1.6. A SERIES OF CHANGES IN LEUKOCYTES.

Leukocytes have 4000 – 9000 in 1 mm3 of blood in the norm, or 4 – 9 x 109 in 1 liter of blood. Relative leukocytosis in infants is 9 – 30 109 / l.

An increase in leukocyte content of 9 x 109/l is called leukocytosis.

1. Physiological leukocytosis is observed in infants in the first days of xayotinig, in the second, third trimester of pregnancy, in women at 2 – 3 weeks after delivery. This is explained by the redistribution of leukocytes circulating in the blood and in the depot. In addition, physiologically redistributed leukocytosis is observed as a result of any stress. After physical exertion " myogen", physiologically redistributed leukocytosis is observed "aclimation " 3 hours after food intake. Physiologically redistributed leukocytoses are always at the expense of neutrophils.

2. Pathological leukocytosis is neoplastic and reactive.

?? Neoplastic is typical of the leukemic type of leukemia.

?? Reactive – is associated with increased cytokine exposure and is observed in the following cases:

a. In injection processes. Therefore, infectious diseases, especially purulent processes, are accompanied by true leukocytosis.

b. Inasseptic inflammation. Allergic reactions, autoimmune diseases, burns, freezing, trauma, poor quality tumor rupture, necrosis in myocardial infarction are accompanied by asseptic inflammation.

v. Poisoning and radiation. Compensatory exacerbation of leukopoiesis is observed as a result of cell death in the acute phase.

Leukotsitar formula

The ratio of leukocyte species to% when the total number of leukocytes is taken as 100. The absolutleukocytar formula is an absolute amount of each type of leukocyte in the blood of 1 μl. The amount of leukocytes is not constant. Its amount increases in the second half of the day, decreases in the morning, increases in the horizontal plane, decreases in the vertical state.

Granulocytes are leukocytes that store granules in the cytoplasm –. Depending on the color of the granules, they are divided into neutrophils, eosinophils, basophils. Granules consist of lysosoma and peroxisoma. Granulocytes are produced only in bone burial.

Agranulocytes include lymphocytes, monocytes, and, unlike granulocytes, are formed in the thymus, spleen, and lymph nodes in addition to bone burial. Agranulocytes do not mean leukocytes without lysosoma and peroxisoma, only their granules are as small as they cannot be seen under a microscope.

According to changes in leukoformula, leukocytosis has the following manifestations:

I. Neutrophil leukocytosis – Increased number of neutrophils with rod core and segment nucleus. Specific to purulent bacterial infections.

II. Eosinophilic or basophilic leukocytosis occurs with an increase in eosinophilic or basophilic granulocytes. Specific to allergic diseases and parasites.

III. Lymphocyte leukocytosis occurs with an increase in lymphocytes. Viral diseases, including abdominal typhoid, are typical of paratyphoid diseases.

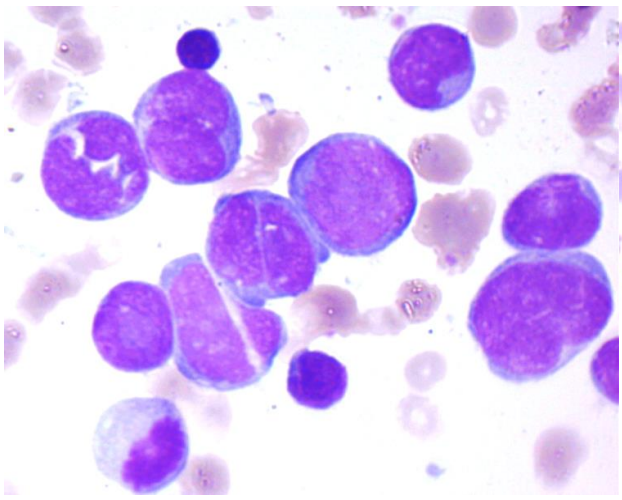
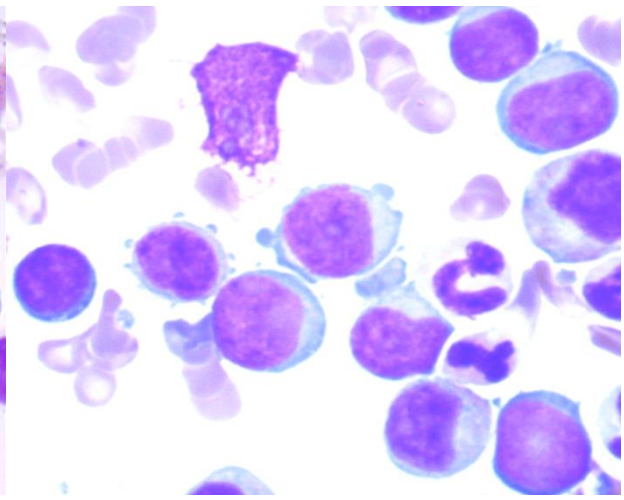
IV. Monocytar leukocytosis occurs with an increase in monocytes. Autoimmun, a long-lasting lick, is prone to disease.

Leukopenia is a decrease in leukocytes and is pathological and physiological. Physiological leukopenia is hom to European.

The etiology of pathological leukopenia varies according to pathogenesis. Pathological leukopenia is observed in aplastic anemia, megaloblastic anemia. Autoimmune in leukopenia is due to autoimmune cytolysis.

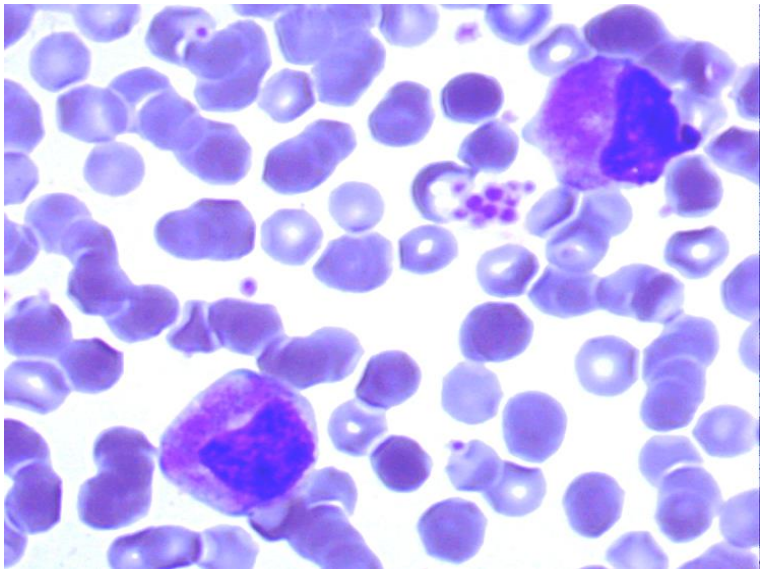
Neutrophils

Neutrophils are 1.5 – 2 times larger cells than erythrocytes. Myeloblast, promielocytes, and myelocytes in the neutrophil series are cells with active mitotic division. In Miellgshramma they occur in a ratio of 1:3:12. The remaining neutrophils – are young metamielositis, rod-core, segment-core neutrophils do not have splitting properties.1.Mieloblast is found in the norm only in bone marrow, 0.5 – 2% of all nuclear cells, round or oval nucleus cells with a diameter of 16 -20 μm. Cytoplasm of the air and granules have not yet appeared. The myeloblast core stores 1 to 5 cores.

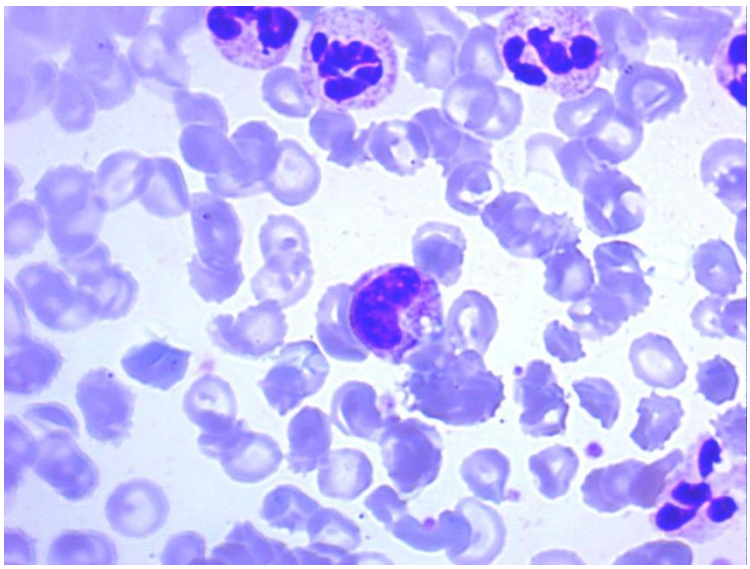



2. Azurophil primary granules are detected in promielocytes. These primary granules retain the chromium-preserving myeloperoxidase enzymis. Granules also store lysosomal enzymes, which are – acidic phosphotase, esterase, b – glucuronidase.

3. The nucleus is not visible under the myelocyte light microscope. Myelocyte granules contain alkaline phosphotase, unlike the above neutrophils.



4.Metamielocyte (young neutrophil) does not have granular formation and division. They are moving cells that can sometimes be detected in peripheral blood

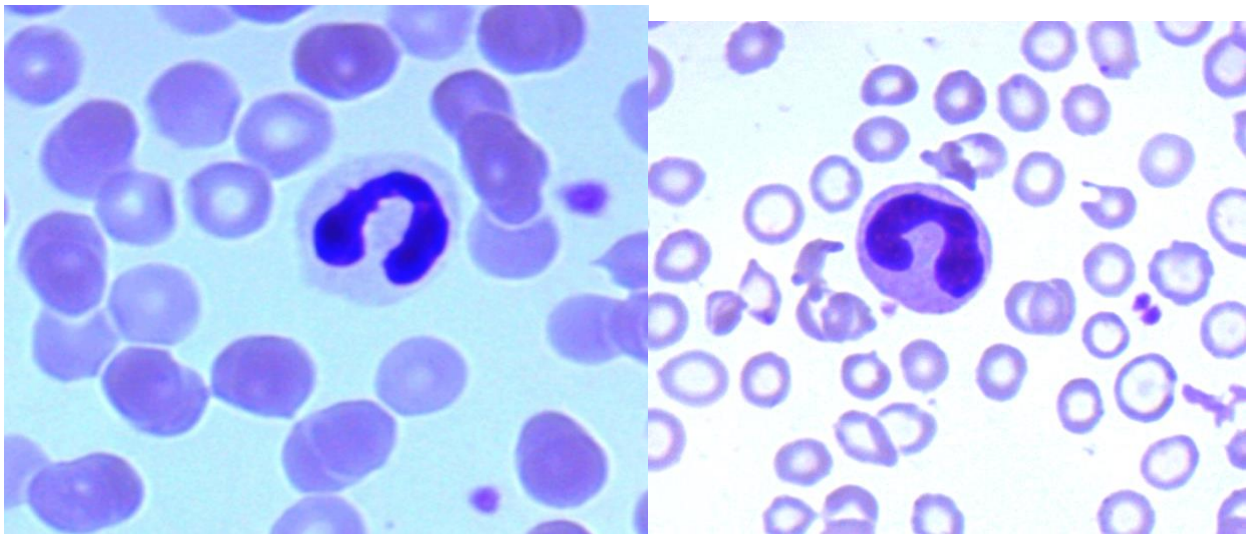


5.The beak nucleus is 3 – 5% of leukocytes in the neutrophil peripheral blood. By this period of maturation, it retains 3 different granules in the neutrophil cytoplasm:

• Specific granules (secondary) store alkaline phosphotase, peroxidase, aminopeptidase, lysocime, collagenase, lactoferrin. Secondary granules are rich in NADF-dependent oxidase and transcobolamine.

• Azurofil granules (primary) contain acidic phosphotase, esterase, myeloperoxidase, sulfated glycosaminoglycans, lysocyme. Serprocidine, a defense that determines the bacteriocidal ability of neutrophils.

• • Tertiary granules store the enzyme gelatinase, which can be detected by an electron microscope. The components of neutrophil granules are determined as follows: lipids are stained with black water, peroxidase with toluidine or benzidine.



6. The segment is a neutrophilated cell with a nucleus, the nucleus of which is broken into pieces. The core is dark ink, the cytoplasm is pink, and the primary, secondary, and tertiary granules are preserved. The core of the mature neutrophils is 4-5% of the 3-segment neutrophils consisting of 2 – 5 segments. Neutrophils from more than 5 segments are not found in the norm. More than 5 of the segments are larynx from the disruption of nucleic acid synthesis in the granulocyte nucleus. This condition is typical of vit B12 deficiency and folate acid deficiency.

Neutrophil hypersegmentation Hame is observed when drugs are taken to disrupt DNA synthesis, such as hydroxymochevina.

There is also hypersegmentation of the nucleus of good quality hereditary granulocytes. This disease is transmitted from the offspring – to the offspring in an autosom dominant shape. The opposite variation of the granulocyte nucleus to the opposite side of the shakilla leads to – Pelger – Hewitt – granulocyte nucleus not segmented (homozygous) or bisegmen-tlane (. This kuasal is accompanied by neutrophil leukocytosis and is harmless to the body. There is also a pseudopelger anomaly of the neocopia of this disease – granulocyte nucleus. It is an acquired disease that is similar to myeloproliferative and myelodysplastic syndrome. In the norm, the nucleus of 5 – 10% granulocytes occurs in the form of pockets, mostik, fibrillation bodies. These manifestations are not specific to any pathology, but vary significantly in Down syndrome and leukemia. Neutrophils and other granulocytes are the ring nucleus of chronic alcoholism.

“ drum sticks ” which can be detected in the neutrophil nucleus are a segment of chromatin on the chromosome. Norma is detected in 3-10% of women with 46XX cariotipes. 46XXU in Kleinfelter syndrome is also detected when there is more than 1 X – chromosome in somatic cells. In women with 45XO cariotipes, the Pelger – Hewitt anomaly is not found at all in carriers.

Neutrophils are evaluated according to their structure and core and are referred to as the core index. The YAdro index is the ratio of the amount of % of myelocytes, metamielocytes, rod-core neutrophils to the amount of % of segmented nuclear neutrophils.

YaI = (M+Yu+T/Ya) / S/Ya

The core index ranges from 0.06 to 0.1 in the norm.

An increase in the amount of young neutrophils and a core index greater than 0.1 is typical of a shift to the left, rather than a decrease in the amount of young neutrophils, an increase in segmented neutrophils, and a core index less than 0.06. The shift to the right is a slowing of myelopoeis and is observed in aplastic anemia, megaloblastic anemia.

Neutrophil function:

1. Phagocytosis. Neutrophils produce lipoxine. Lipoxins derived from arachidonic acid belong to the group of anti-inflammatory lipid mediators.
2. Neutrophils synthesize a special enzyme, lipid-degrading acid – oxyacyl-hydrolase. It is this enzyme that provides the disintoxication role of granulocytes in gram – negative microflora infections because lipid A is an active component of gram-negative microflora endotoxin.
3. Neutrophils and promielocytes are involved in hemostasis. They are part of the white thrombus.

4. Neutrophils produce vitamin B12-binding protein – transcobolamine 3.

Neutrophilia

Neutrophilia – Increased neutrophil granulocyte count in μl of blood 7500 – is mainly observed in acute infections called by pyogenic bacteria, including Pneumococcus, Streptococcus, Staphylococcus, some fungi.

Neutrophilia and leucocytar formula left shift are typical of the following infectious diseases:

1. Bacterial diseases (scarlatine peak period, staphylococcal, gonorrhea, meningococcal infection (Toxic grain is often detected in neutrophils).2. Pseudo-TB and iersiniosis, leptospirosis, Layma's disease.

3. Many viral diseases and some bacterial infections (whooping cough, salmonellosis, and b.), sometimes the normal number of neutrophils is even lower.

4. Asseptic neutrophilia is observed in acute blood loss, hemolysis, burns, myocardial infarction, intestinal obstruction, gout arthritis, immunocomplex diseases – this does not mean that there is infection.

5. Intoxication (uremia, diabetic lactatacidosis and ketoacidosis, pregnancy toxicosis, thyreotoxicosis, jaundice resulting from alcoholic hepatopathy, snake venom, lead, poisoning with various chemists, some medications may experience side effects) still aseptic neutrophilia.

6. In patients with myeloley, Vakez's disease, and idiopathic myelofibrosis, neoplastic netrophilia is always accompanied by a shift to the left. In addition, reactive neutrophilia is observed under the influence of cytokines produced as a result of lymphogranulematosis, lymphoma, and various cancers, especially when there is necrosis and inflammation in the tumor.

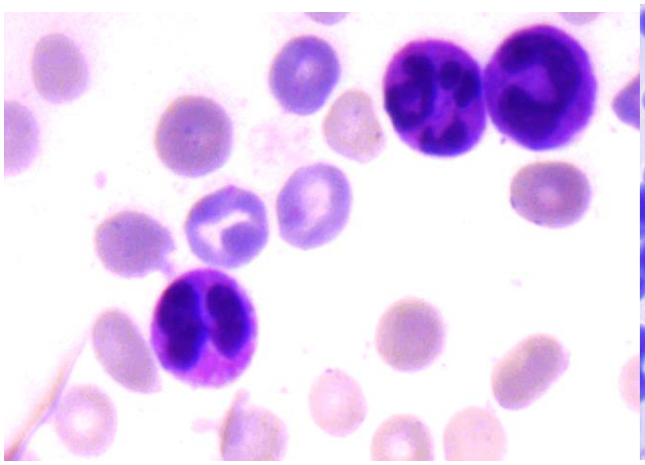
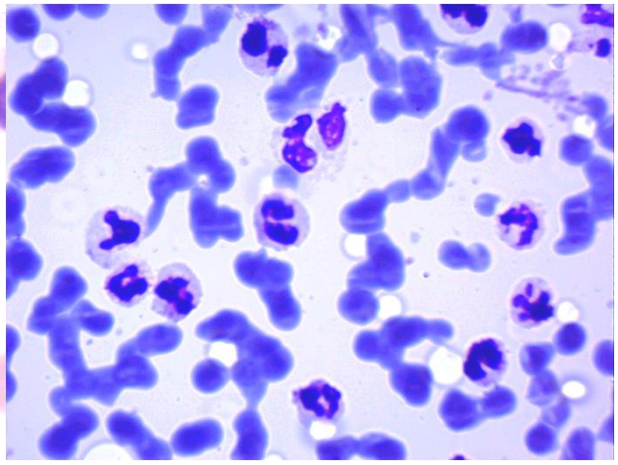
7. Ham neutrophilia is observed after splenectomy.

1.7. LEYKEMOID REAKTIONS.

A strong neutrophil is accompanied by leukocytosis and leukoformula with hyperregenerator left shift. In this case, the number of granulocytes exceeds 30-50 x109/l, sometimes it may be less.

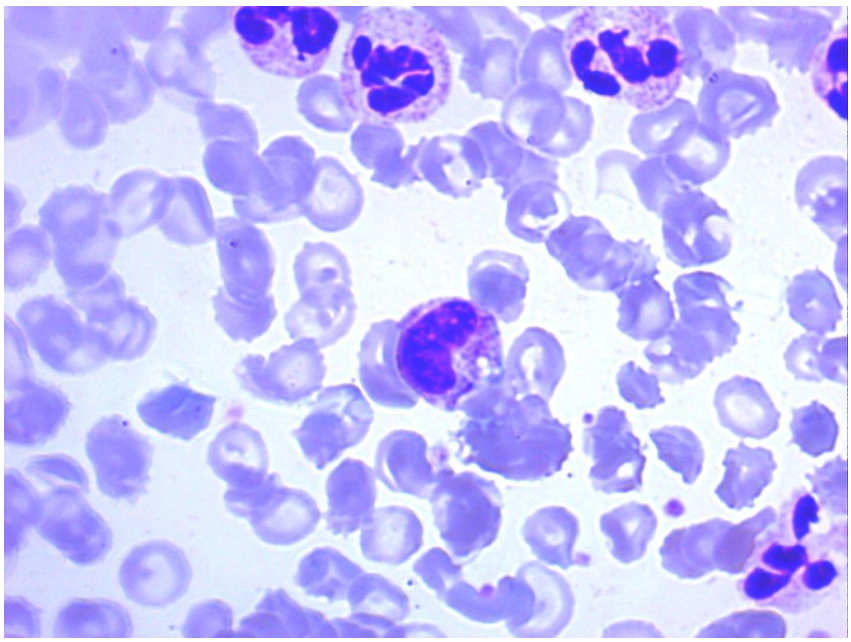
Reasons:

- Severe septic processes
- Exogenous poisoning
- acute phase of radiation
- acute hemolysis
- shock
- metabolic changes (uremia, endocrinopathy)
- Disintegration of malignant tumors, production of bone coma metastasis or granulopoiesis stimulants (hypernephroma).

Specific characterization of leukemoid reactions:

- Blast cells in leukemia reactions do not retain antigen markers in leukemia.
- No chromosomal changes in leukemoid reactions.
- Increased or normal alkaline phosphotase in myeloid leukemia reactions, low activity in leukoids.
- Myeloblast leukemia is high in myeloblast leukemia, and myeloid leukemia may be present in reactions. Leukemic gap is not observed.
- No signs of acute leukemia, i.e., hemorrhagic syndrome and splenomegaly, are observed in myeloid leukemia reactions.
- Bazophilia and eosinophilia are not observed in myeloid leukemia reactions. Mieloleucosis has basophilic and eosinophilic assosia.
- Toxic grain in the cytoplasm of neutrophils in myeloid leukemia reactions, changes in the nucleus are observed, which is not specific to leukemia.
- The Auyer body is detected in the leukemia, which is not typical of leukemoid reactions.
- Dynamics is rapidly observed in leukemoid reactions. Once the main patholactic process is eliminated, the hemogram is returned to normal.
- Anemia and thrombocytopenia are not observed in leukemoid reactions.



Neutropenia.

Decreased neutrophil counts from 1.5 x 109/l are indicative of functional or organic deficiency of bone fat or by exposure to antithesis against circulating leukocytes in the blood, immune complexes or toxic factors (autoimmune diseases, tumors, aleicemic forms of leukemia, effects of certain drugs, hypersplenism) Increased neutrophil breakdown. Neutropenia usually occurs with leukopenia.

Reasons:

1. Severe acute and chronic infectious diseases:
- viral (influ, measles, rubella, chickenpox, infectious hepatitis, AIDS);
- bacterial (bel typhoid, paratyphoid, brucellosis);
- ricketsiosis (rash typhoid);
- protozole (malyaria, toxoplasmosis).

2. Side effects of some drugs (sitostatics, sulfanilamides, analgetics, anti-seizure drugs, antitireoid drugs);
3. light therapy and radiation;
4. hypersplenism (anemia and thrombocytopenia are raw);
5. aplastic anemia;
6. agranulocytosis;
7. anaphylactic shock.

Neutropenia occurs mainly as neutrophils move to the left and develop against the background of purulent inflammation. When the resistance of the organism decreases, it is characteristic of older organisms.

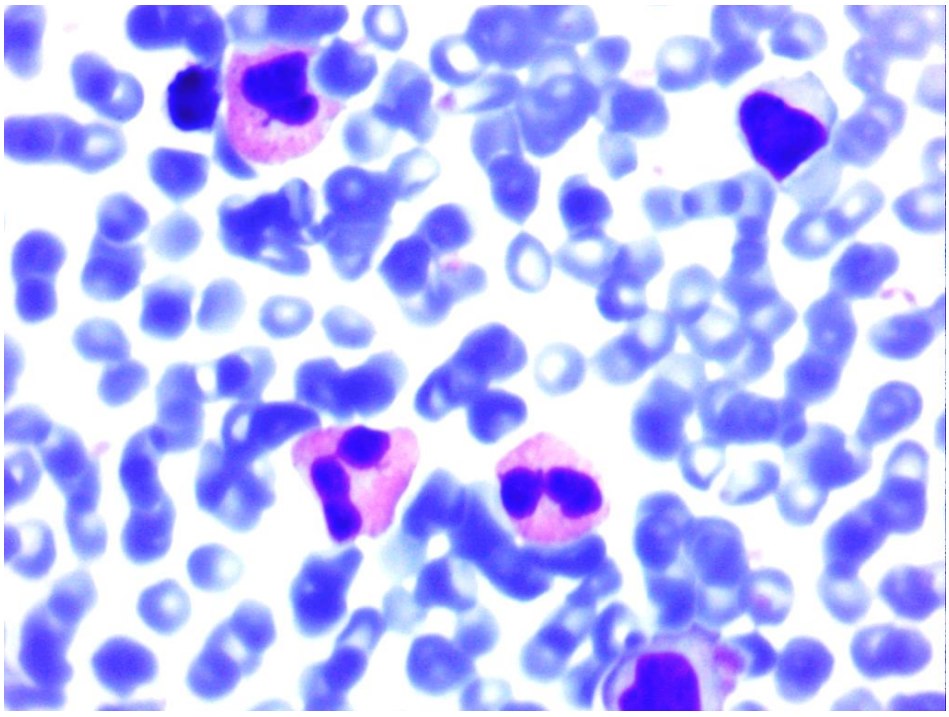
Eosinophils.

Eosinophils are 12–15 μm in diameter, a cell with a larger nucleus than neutrophils and a lower number of segments, and the grain is more strongly expressed. Antigen - phagocytosis of the antitana complex. They respond to

chemotaxic factors separated from fat cells and basophils. During the day, the amount of eosinophils varies, the amount increases at night, decreases during the day. Based on the reduction of eosinophils in peripheral blood from 0.4 x 109 /l, the formation of an antigen-antitana complex, autoimmune diseases lie.

Prolification of the eosinophilic line in bone burial:

1. Allergic diseases (bronchial asthma, echkemia, angioneurotic edema, serum sickness, drug disease).
2. Parasitic invasion (trichinellosis, exinococcal, opistorchosis, ascaridosis, difillobotriosis, lyambliosis, malaria).
3. Adhesive tissue diseases and systemic vasculitis (periarthritis, rheumatoid arthritis, sclerodermia, systemic red wolf).
4. Nospecific ulcer colitis.
5. Skin diseases (dermatitis, eczema, lishay skin shape).
6. Blood diseases (lymphogranulematosis, erythremia, chronic myeloleycosis).
7. Eosinophilic infiltrate in the lungs.
8. Phyoplastic endocarditis of lyoffler.



Eosinopenia

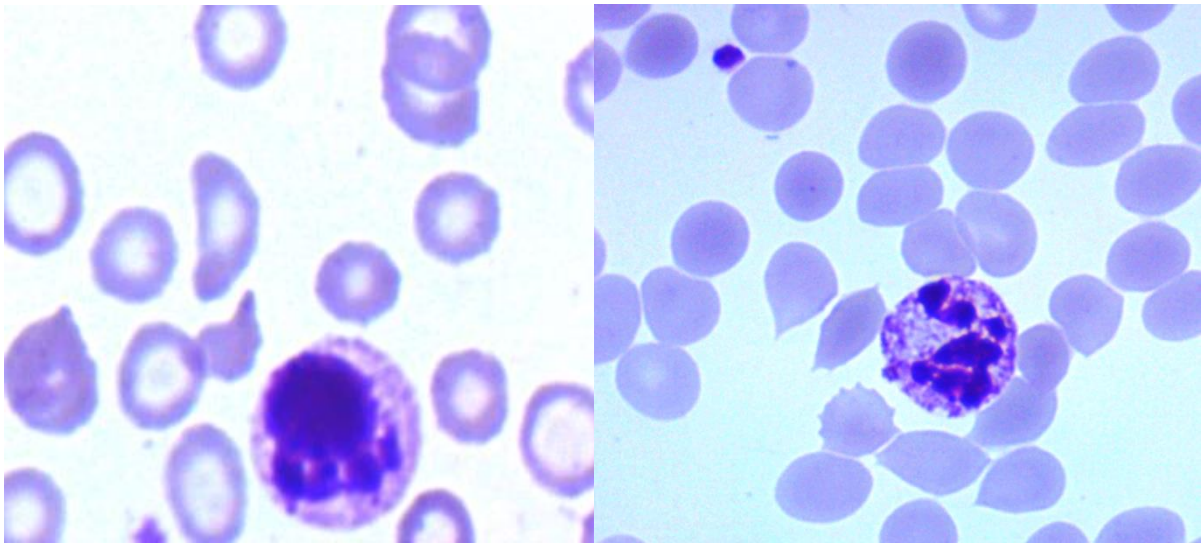
Eosinopenia: Decreased or lost eosinophils. In infectious and purulent diseases, leukocytosis is accompanied by neutrophilia and indicates the activity of the process and the normal reaction of bone coma to inflexmia. If eosinopenia comes with neutropenia and leukopenia, it indicates a decrease in organism resistance and is a bad prognosis. Eosinopenia is also observed in aplastic and vit.B12 – deficient anemia.

Basophilia

Basophilic size 8-12 μm,. produces heparin, histamine granules. The main task is to participate in a rapid allergic reaction. Increased basophils are a rare condition in the clinic:

1. Myeloproliferative diseases (chronic myeloleucosis, myelofibrosis with myeloid metaplasia, Vakez disease).
2. Hypothyroidism.
3. Lymphogranulematosis
4. Chronic hemolytic anemia.

Bazopenia has no diagnostic significance.

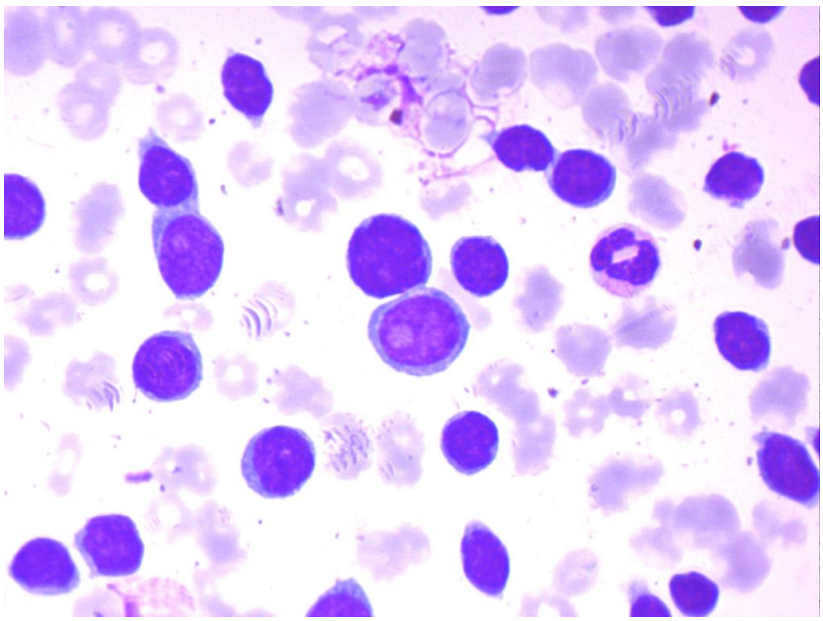


Lymphocytosis.

The main function of lymphocytes is to be familiar with the antigen and to participate in the immune response of the organism. T- lymphocytes are involved in cellular immunity, B-lymphocytes are involved in humoral immunity. Relative lymphocytosis is often observed in the clinic, i.e., the amount of lymphocytes increases by% even if the absolute amount is normal. Relative lymphocytosis is observed in all diseases transmitted by absolut neutropenia and leukopenia. Lymphocytes are observed to exceed the absolute amount of 3.5 x 109/l:

1. Acute infections (including pediatric infections: whooping cough, measles, chickenpox, scarlet, infectious mononucleosis, parotitis, lympholeucosis, acute viral hepatitis, cytomegalovirus).
2. Miliar tuberculosis, bronchial gland tuberculosis.
3. Hyperthyreosis.
4. Acute and chronic lympholeukemia
5. Lymphoma (lymphogranulematosis), lymphosarcoma.

Lymphocytosis should not be considered as a laboratory sign indicating the response of the immune system in purulent – inflammatory diseases.



Lymphocytopenia.

Decreased lymphocytopenia in peripheral blood. Relative lymphocytopenia is observed in patients with neutrophilia, so it has little diagnostic and prognostic significance. Absolut lymphocytopenia less than 1.2 x 109/l T is a sign of systemic immunodeficiency (immune deficiency) and in this case immunological examinations should be performed.

Monocytosis.

Monocytosis is relative and absolute. The body is cleansed of iodine bodies and the immune system is involved in the reaction along with lymphocytes. Some of the monocytes move along the vascular wall and some along the bloodstream. Monocytes moving along the wall emerge from the veins into the tissue, and tissue macrophages are formed from them. The diameter of monocytes is 15-25 μm.

Relative monocytosis occurs when neutropenia and leukopenia are observed, and independent diagnostic significance is negligible.

• Absolute monocytosis is important in some infections and purulent – inflammatory processes, and their functions include:

1. Some classes protect against microorganisms.
2. Participates in immune reaction stages with antigens and lymphocytes.
3. Destroys damaged and obsolete cells.

Absolute monocytosis is observed:

1. Some infections (infection mononucleosis, acute endocarditis, viral, fungal, ricketsiosis, and protozoic infection).
2. Inflammatory diseases of long-acting pus –.
3. Granulematous diseases (active tuberculosis, lame disease, sarcoidosis, nospecific ulcer colitis).

4. Blood diseases: acute monocitar leukemia, chronic myeloicosis, myeloma, aplastic anemia.

Monocytopenia.

Decreased or complete loss of monocytes in peripheral blood is observed in severe infection and purulent – inflammatory diseases.

1.8. 1.8. MAIN CLINIC -LABORATOR MARKERS OF ACUTE LEYCOS.

Objectives of the training: leukoses, major cytological features of leukemia, acute leukemia, acute leukemia species, cytomorphological features specific to the blast cell, changes in peripheral blood in acute leukemia, bone in acute leukemia changes, introduction to cytoxymic reactions.

Leukemia is a group of malignant tumors that develop from hemopoietic cells. Tumor cells are proliferated in bone coal, blood, lymphoid, and other tissues, and have systemic characterization from the onset of the disease.

The main cytological features of leukemia:

1. Uncontrolled, non-stop proliferating.
2. Apoptosis disorder.
3. Cells lose their sensations of differentiation and maturation.
4. Cell morphological and metabolic atyipism.
5. Metaplasia in blood-forming organs.
6. Atypical cells are released into the peripheral blood.
7. Occurrence of blood-forming furnaces in organs and tissues not involved in hemopoeia (liver, kidney, subcutaneous fiber, intestine, and b.).

Leukemia is divided into the following types according to the characteristics of tumor cell differentiation and maturation:

1. Acute leukemia (tumor substrate immature blast cells).
2. Chronic leukemia (asthma substrate ingested and sown cells).

Acute leukemia is a heterogeneous, cloned-risk tumor disease in the blood system consisting of unripe cells.

According to the cytomorphological and cytoxymic sensations of leukemic cells, acute leukemia is divided into three groups:

1. Acute myeloblast leukemia;
2. Acute lymphoblast leukemia;
3. Non-differentiated leukemia.

According to the who, acute leukemia contains 20% or more of the peripheral blood and bone burial blast cells.

Blast cell-specific cytomorphological features:

1. The structure of the nuclear chromatin is thin mesh;
2. Nuclear - the presence of nuclei;
3. Basophilic cytoplasm;

4. Nuclear-cytoplasmic ratio 4:1-8:1.

Changes in peripheral blood in acute leukemia:

1. Normositar anemia;
2. Leukocyte count from severe leukopenia to severe leukocytosis (1 to 300 x109/l):

a) aleukemic form - leukocyte content 1-3 x109/l, no blast cells or 1 -2%, relative lymphocytosis;

b) subleukemic form - leukocyte content 4-14 x109/l, blast cells 5-10%;

c) leukemic form - leukocyte content greater than 15 x109/l, blast cells more than 10.

3. Thrombocytopenia;
4. In the leukositar formula «leukemic cavity » - the presence of blast and crushed cells in the blood, the absence of intermediate series cells.
5. ESR increase.

Changes in bone burial in acute leukemia:

1. Bone remover blast transformation (blast cells more than 30%);
2. Decreased blood-forming myeloid, lymphoid, erythroid barriers;
3. A sharp decrease in megacities.

Cytochymic reactions.

Blood cytoxymic reactions are based on a color reaction with metabolically active enzymes and substrates of blast cells to determine the type of acute leukemia. Detection of myeloperoxidase, acidic and alkaline phosphatase, nospecific esterase, glycogen and lipids is of great diagnostic importance. Cytochymic reactions allow the identification of blastes, the degree of cell absorption, and treatment tactics to be determined.

Control questions:

1. The concept of leukemia.
2. Classification of leukemia.
3. The main cytological signs of leukemia
4. Blast cell-specific cytomorphological features
5. Acute leukemia.
6. Changes in peripheral blood in acute leukemia.
7. Changes in the myelogram in acute leukemia.
8. Cytochymic reactions in acute leukemia.

1.9. CONCEPT OF CHRONIC LEUKEMIA AND LABORATORY DIAGNOSIS.

Objectives of training: chronic leukemia, chronic myeloleucosis, chronic myeloleicosis, cytological diagnosis of acceleration and terminal stage, chronic

myeloleicosis cytological diagnostic criteria, chronic lympholeucosis, introduction of cytological diagnosis of chronic lympholeucosis and terminal stage.

Chronic leukemia is a tumor disease in which the blood-creating organs are differentiated to the insulated cells while maintaining the absorption sensations of the tumor cells. Chronic myeloleucosis and chronic lympholeucosis are the most common among chronic leukemia.

Chronic myeloleucosis is a tumor disease in which the blood-forming system developed from the pre-myelopeez cell. Chronic myeloleucosis is the main cytomorphological substrate of mature and yyet granulocytes - promyelocyte, myelocyte, metamielocyte, rod-core and segment-core neutrophils.

Chronic myeloley is mainly found in 30-60 years. Clinical delay consists of 3 stages:

1) chronic (good quality);
2) acceleration phase;
3) terminal stage (polyclone, dangerous).

Cytological diagnosis of chronic myeloleicosis

In peripheral blood:

1. Mild-grade normochrome anemia.
2. Leukocytosis 50-1000x109/l.
3. Increased beacons of neutrophils.
4. Occurrence of metamyelocyte, myelocyte, promielocytes in the blood.
5. Granulositis anisositosis, nuclear and cytoplasma vacuolization, nuclear polymorphism, absence of neutrophill granules (gipo- and agranulation).
6. A small amount of blast can come out.
7. Eosinophil-bazophilic association (eosinophil and basophilic increase).
8. Decreased lymphocytes.
9. In 40% of cases, thrombocytosis is up to 600-1000x109/l.

In the myelogram:

1. Bone coal is multicellular.
2. A sharp increase in granulositar series cells.
3. Eosinophil-bazophilic association.
4. Blasts up to 10.
5. Megacariocytes abound.
6. Decreased erythrocariosites.

Cytological diagnosis of chronic myeloleic acidation stage

In peripheral blood:

1. Moderately severe and severe normochrome anemia.
2. Leukocytosis 50-1000x109/l.
3. Increased beacons of neutrophils.

4. Occurrence of metamyelocyte, myelocyte, promyelocytes in the blood.
5. Blasts in the blood up to 15.
6. Eosinophil is a basophilic association.
7. Platelet levels are reduced.

In the myelogram:

1. Bone coal is multicellular.
2. A sharp increase in granulositar series cells.
3. Eosinophil-bazofill association.
4. Blasts up to 15.
5. Megacariosites are reduced.
6. Erythrocariocytes decrease sharply.

Cytological diagnosis of the terminal stage of chronic myeloleucosis

In peripheral blood:

1. Severe normochrome anemia.
2. Leukocytosis 50-1000x109/l.
3. Decreased segmented neutrophils.
4. Occurrence of metamyelocytes, myelocytes, promielocytes in the blood.
5. Blasts in the blood are more than 15.
6. At some point eosinofill is a bazofill association.
7. Platelet levels decrease sharply.

In the myelogram:

1. Decreased edible granulocytes.
2. Decreased erythrocyte and megacariosyrate series cells.
3. Blast cell proliferation.

Cytological diagnostic criteria for chronic myeloleucosis:

1. Normochrome anemia.
2. Leukocytosis 50-1000x109/l.
3. Increased beacons of neutrophils.
4. Occurrence of metamyelocyte, myelocyte, promyelocytes in the blood.
5. Blasts can appear in the blood.
6. Decreased segmented neutrophils.
7. Eosinophil is a basophilic association.
8. At 40% the platelet content increases, decreases during the terminal period.
9. Myeloperoxidase positive in tumor cells in cytoxymic examination.

Chronic lympholeucosis is a lymphoid tissue tumor, a cytomorphological substrate of yelted lymphocytes. People over the age of 50 are diagnosed with chronic lympholeucosis.

Cytological diagnosis of chronic lympholeucosis

In peripheral blood:

1. Normochrome anemia.
2. Leukocytosis 50-600x109/l.
3. Absolute lymphocytosis.
4. Ridel cells (are divided or renal-core lymphocytes).
5.Gumprext shadows (traces of lymphocytes broken down during grease preparation).
6.Granulositar cells are a decrease in rod-core and segment-core neutrophils.
7. Thrombocytopenia.
8. Cytochymic examination: glycogen-positive in tumor lymphocyte cells.

In the myelogram:

1. Bone fat total lymphoid infiltration.
2. Decreased cells in granulositar, erythrositar, megacariositar.

Cytological diagnosis of the terminal stage of chronic lympholeucosis

In peripheral blood:

1. Normochrome anemia.
2. Leukocytosis 50-600x109/l.
3. Absolute lymphocytosis.
4. The number of blasts exceeds 15.
5. Ridel cells (Divided or renal-nuclear lymphocytes).
6. Gumprex shadows (traces of lymphocytes broken down during grease preparation).
7. Granulositar cells are a decrease in rod-core and segment-core neutrophils.
8. Thrombocytopenia.

In the myelogram:

1. Bone fat total lymphoid infiltration.
2. Decreased cells in granulositar, erythrositar, megacariositar.
3. Blast cells exceed 15.

Control questions:

1. The concept of leukemia. Acute leukemia.
2. Classification of leukemia.
3. Changes in peripheral blood in acute leukemia.
4. Changes in the myelogram in acute leukemia.
5. Cytochymic reactions in acute leukemia.
6. Cytological diagnosis of chronic myeloleycosis.
7. Cytological diagnosis of chronic myeloleicosis accelerator stage.
8. Cytological diagnosis of the terminal stage of chronic myeloleucosis.
9. Cytological diagnosis of chronic lympholeucosis
10. Cytological diagnosis of the terminal stage of chronic lympholeucosis.

1.10. LEUKOCYTOSIS, LEUKEMOID REACTIONS AND LEUKOS LABORATOR DIAGNOSTICS.

Objectives of the training: to provide information on leukocytosis, neutrophil,eosinophil, basophil, lymphositar and monositar leukocytosis, leukemoid reactions, myeloid, lymphositar, eosinophil leukemoid reaction, secondary erythrocytosis, reactive thrombocytoz.

Leukositar and leukemoid reaction is a common clinical-hematological syndrome. The development of leukositar and leukemoid reactions affects the progression and outcome of the disease.

Leukocytosis – is a clinical laboratory syndrome characterized by an increase in the number of leukocytes in the blood above 10x109/l. Leukocytosis has neutrophil,eosinophil,bazophil, lymphositar, and monositar species. The most common leukocytosis is neutrophil leukocytosis.

Neutrophil leukocytosis

Functional neutrophil leukocytosis is observed for a short time and is not associated with symptoms of the disease (a few minutes to several hours). Eating is observed after stress.

True neutrophil leukocytosis is a long-term increase in neutrophil leukocytes (from a few hours to several weeks).

True neutrophil leukocytosis occurs in the following cases:

1. Inflammatory diseases of bacterial etiology.
2. Severe exo and endogenous intoxications.
3. Severe hemolysis.

Simple animal invasions (toxoplasmosis, malaria)

2. Acute lymphocytosis

□ Cardiovascular insufficiency (Acute cardiac arrest, myocardial infarction, septic shock)

□ Lymphositosis under the influence of drugs

□ Allergic reactions

《 After major surgery

□ After epileptic seizures

□ Severe injuries

3. Chronic lymphocytosis

□ Adhesive Tissue Systemic Diseases (Revmotoid Arthritis)

l Osmas

□ Chronic jaundice diseases

□ Smoking

Chronic lympholeucous differentiated diagnosis is performed with lymphocytic myeloid reaction (4-id).

Eosinophil leukemia reaction

Eosinophilic leukemia is characterized by an increase in the amount of eosinophils in the blood by more than 20% in the reaction and the appearance of eosinophilic metamielocytes, myelocytes, promielocytes.

Eosinophilic leukemia reaction occurs in the following pathologies:

1. In allergic reactions
2. Gijja invasions
3. Immunopathological diseases (revmatoid arthritis, Crohn's disease, nospesive ulcer colitis and b.
4. Hemoblastosis and other neoplasia (Chronic myeloicosis, lymphogranulematosis, lymphomas, and b.
5. Pulmonary eosinophil infiltrates, bronchial asthma
6. Quinke angioneurotic tumor
7. Dermatoses
8. After vaccination and b.

Eosinophilic leukemia reaction is differentiated by chronic myeloleycosis. For this, the eosinophilic series counts as 100 cells. If the eosinophilogram is dominated by rod and segment-core eosinophils, eosinophil is considered a leukemoid reaction. In chronic myeloleucosis, eosinophil metamielocytes, myelocytes, promielocytes predominate in the eosinophilogram.

Monositar leukemia reaction

The etiological factors of monositar leukemoid reaction and monocytosis are the same. Monositar-type leukemia reaction is differentiated by chronic monositar leukemia (5-application).

Secondary erythrocytes

Secondary erythrocytes are an increase in the absolute amount of erythrocytes.

Secondary erythrocytes develop in the following cases:

1. Increased erythropoietin in renal disease
2. Inflammatory diseases of the pulmonary pus
3. Pulmonary edema
4. Congenital heart defects
5. Randyu-Osler syndrome
6. Vascular and hepatic tumors

Secondary erythrocytes should be diagnosed with erythremia (true polysitemia) (6-application).

Reactive thrombocytes

Reactive thrombocytes are an absolute increase in platelet count.

Reactive thrombocytes are observed in the following cases:

1. Chronic inflammatory diseases
2. Hemolytic anemia
3. After splenectomy
4. In oncopatology
5. When strong bleeding
6. After burning
7. After surgery
8. When treated with corticosteroids
9. Immunopathological diseases (revmatoid arthritis, Crohn's disease, nospesive ulcer colitis and b.

Differential comparison of reactive thrombocytes with eccentric thrombocythemia is required (7-ilova).

Control questions:

1. Neutrophil leukocytosis
2. Eosinophil leukocytosis
3. Bazophilic leukocytosis
4. Lymphositar leukocytosis
5. Monositar leukocytosis
6. Myeloid type leukemoid reaction
7. Lymphositar type leukemoid reaction
8. Eosinophilic type leukemoid reaction
9. Monositar type leukemoid reaction
10. Secondary erythrocytes and reactive thrombocytozes

1.2. ANALYTIC PART

New pedagogical technology used in training “BUM”

All participants stand in a rotating shape, they have to count the departure and whoever sets the number to three must say BUM. A participant who does not say BUM will be asked a pre-prepared question.

CONTROL QUESTIONS:

1. Erythrocyte structure?
2. What potological erythrocytes do you know?
3. Normal erythrocytes?
4. Hemoglobin structure?
5. Changes in hemoglobin?
6. Types of leukocytes?
7. Which pathological entries occur in neutrophils?
8..Leukoformula normal cadets?

9. What is the left shift of the leukofurmula?
10. Is this poykilacitosis?
11. Is this anisacitosis?
12. A formula for determining a color indicator?
13. Platelet information?
14. What factors influence the change in the ECHT?
15. When will the ECHT increase?
16. When will the ECHT decrease?

SITUATIVE ISSUES.

Situational issue №1.

Hb 66 g /l, er.3.2 x1012 /l, RK 0.6, ret.3‰, trom.179x109/l, leyk.5x109/l, t / ya 2%, s/ya 69%, eoz.2%, mon.5%, lim.22%, ECHT 10 mm / h. Morphology of erythrocytes, microcytosis, hypochromia, zucilocytosis.

Questions: 1. What disease is a hemogram specific to?
2. Hose changes to the disease in the hemogram?
3. Morphology of erythrocytes?

Answers:1. Iron deficiency anemia.
2. Hb 66 g /l, er.3.2 x1012/l, RK decreased by 0.6.
3. Microcytosis, hypochromia, zucchylocytosis.

Situational issue №2.

HB. 80 g /l; Eritrea. 3.2x1012/l. Reticulocyte 0.2‰, Color indicator. — 1.1, Leukocytes 3.9x109/l, Platelets 170x109/l, Leukoformula: t/ya-1%, s/ya-74%, eoz.-1%, base.0%, lymph.-21%, monocyte-3%, ECHT—10 mm / s. Morphology of erythrocytes: macrocytosis, megalocytosis, Jolly body, Kebot ring. Leukocyte morphology nuclear hypersegmentation.

Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. Morphology of erythrocytes.

Answers: 1. Vitamin B12 deficiency anemia.
2. Pansytopenia, displacement of the leukoformula to the right.
3. Erythrocyte morphology: macrocytosis, megalocytosis, Jolly body, Kebot ring, hypersegmentation of leukocyte nucleus.

Situational issue №3

HB. 60 g /l; Eritrea. 2.2x1012/l. Reticulocyte 40%, Color index 0.9. Leukocytes 9x109/l, platelets 190x109/l. Leukoformula:t/ya-8%, s/ya-66%, eoz.-1%, baz.0%, lymph.-21%, monocyte-4%, ECHT—10 mm/s. Morphology of erythrocytes: microspherocytosis

Questions: 1. What disease is a hemogram?

2. Hose changes to the disease in the hemogram?
3. Morphology of erythrocytes.
Answers: 1. Microsferocytary hemolytic anemia.
2. Decreased hemoglobin and erythrocyte, reticulocytosis.
3. Microsferocytosis.
Situational issue №4
Hb 70 g / l, er. 2,1x1012/l, RK 0.9, reticulocyte 40‰, leukocyte 5,2x109/l, t/ya 4%, s/ya 61%, eosinophill 6%, lymphocyte 32%, monocyte 7%, ECHT 20 mm / hour. Blood bioximic analysis: a sharp increase in the unbound fraction of bilirubin. Kumbs reaction is positive.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. What does the Kumbs reaction determine?
Answers: 1. Autoimmune hemolytic anemia.
2. Anemia, reticulocytosis.
3. Antiteloses against erythrocytes.
Situational issue №5.
Hb 60 g /l, er. 2,2x1012/l, RK 0.7, ret.0,1‰, trom. 40.x109/l, leyk. 2.4x109/l, t/ya 5%, s/ya 35%, lim.52%, mon.8%, ECHT 25 mm / h.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. Clinical syndromes?
Answers: 1. Aplastic anemia.
3. Pansitopenia.
4. Anemia, hemorrhagic syndrome, infection syndrome.
Situational issue №6
Hb 80 g /l; Eritrea. 3.2x1012/l. Reticulocyte 0.2‰, Color index 0.9. Leukocytes 3.9x109/l, platelets 50x109/l. Leukoformula: blast-45%, t/ya-1%, s/ya-37%, eoz.-0%, baz.0%, lymph.-14 %, monocyte-3% ECHT—2-10 mm / s.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. Blood blasts in the norm?
Answers: 1. Acute leukemia.
2. Pansytopenia, blastemia.
3. It can't be.
Situational issue №7
Hb 80 g /l; Eritrea. 3,2x1012/l.Reticulocyte 0.2‰ Color indicator.— 0.9. Leukocytes 399x109/l, Platelets 470x109/l, Leukoformula: myelocyte 5%,

metamielocyte 9%, t/ya-12%, s/ya-45%, eoz.-9%, baz.4%, lymph.-13%, monocyte-3%, ECHT—25 mm\s.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. What is the increase in eosinophyll and basophill?
Answers: 1. Chronic myeloley.
2. Hyperleukocytosis, myelocyte, metamielocyte, peripheral bleeding of promielocytes, eosinophil, basophilic increase, thrombocytosis.
3. Eosinophilic basophilic association
Situational issue №8.
Hb 50 g /l, er.2,1x1012 /l, RK 0.8, platelet 50x109/l, leukocyte 130x109/l, blast 1%, t /ya 1%, s /ya 14%, eosinophyll 4%, lymphocyte 84%, monocyte 3%, ECHT 4 mm / hour.
Questions: 1. What disease is a hemogram?
2. Disease-specific changes in the hemogram?
3. What is the cause of anemia?
Answers: 1. Chronic lympholeucosis.
2. Hb 50 g /l, er.2,1x1012/l, platelet 50x109/l, leukocyte 130x109/l, blast 1%, lymphocyte 84%.
3. Tumor cells squeeze the normal erythroid series of cells into the bone coma.
Situational issue №9.
Hb 190 g / l, er. 6.3x1012/l, RK 0.9, platelet 550 x109/l, leukocyte 11 x109/l, t 6%, s/ya 60%, eosinofill 4%, lymphocyte 24%, monocyte 55, ECHT 1 mm / hour.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. What is detected in the myelogram?
Answers: 1. Eritrea.
2. Hb 190 g /l, er. 6.3x1012/l, platelet 550x109/l, leukocyte 11x109/l, ECHT 1 mm / hour.
3. Myelogram: three rows of hyperplasia, erythropoiesis sharply increased.
Situational issue №10.
Hb 90 g /l, er.3.4 x1012 /l, RK 0.8, platelet 5x109/l, t 3%, s /ya 62%, eosinophill 3%, basophil l0%, lymphocyte 25%, monocyte 5%, ECHT 21 mm / hour.
Questions: 1. What disease is a hemogram?
2. Hose changes to the disease in the hemogram?
3. What is detected in the myelogram?
Answers: 1. idiopathic thrombocytopenic purpura
2. platelet 5x109/l
3. Myelogram: high number of megacariocytes, broken plate separation.

TESTS.

1. What changes are observed in the overall blood test in hemophilia:
A. thrombocytopenia
B. no obvious changes*
C. leukocytosis
D. pancytopenia
E. lymphopenia
2. In Verlgof's disease, the following is reduced:
A. megacariocytes
B. leukocytes
C. platelets*
D. monocytes
E. normocytes
3. What is the main root of the disease in acute leukemia:
A. erythrocytes
B. monocytes
C. blasts*
D. s/ya neutrophils
E. t/ya neutrophils
4. In acute leukemia is not typical for blood analysis:
A. anemia
B. thrombocytopenia
C. leukopenia
D. thrombocytosis*
E. leukemic rupture
5. In which type of leukemia, blast cells appear in the blood:
A. erythremia
B. in acute leukemia*
V. myeloma
G. hemophilia
D. aplastic anemia
6. In which type of chronic leukemia ESR is equal to 1-2mm/ hour:
A. erythremia*
B. chronic myeloley
V. chronic lympholeucosis
G. hypoplastic anemia
D. myeloma
7. Chronic myeloleucosis is the main root of the disease
A. blast cells

B. young myeloid cells*
C. lymphocytes
D. plasmodites
E. basophils
8. Characteristic for blood analgesia in myeloma:
A. ESR acceleration*
B. Decrease in ESR
C. thrombocytosis
D. eosinophil-bazophilic association
E. erythrocytosis
9. The diffuse stage of erythema is not in the blood analgesia
A. pancytosis
B. leukocytosis
C. Slowing down the ESR
D. leukopenia*
E. increase in hemoglobin,erythrocytes
10. Mielom cannot be a substrate for disease:
A. plasmodites
B. T-lymphocytes*
V. myelocytes*
Gm. basophils*
D. plasmoblasts
11. Chronic myeloleucosis is characteristic for changes in the terminal stage:
A. thrombocytopenia*
B. thrombocytosis
V. finding blast cells in the blood*
G. leukopenia
D. erythrocytosis
12. Not specific to hypoaplastic anemia:
A. leukocytosis*
B. erythrocytosis*
V. thrombocytopenia
G. leukopenia
D. reticulocytopenia
13. The most informative for the diagnosis of hypoaplastic anemia:
A. hemogram*
B. myelogram *
V. proteinogram
G. hemoglobin electrophoresis

D. coagulogram
14. Not typical for chronic myeloicosis:
A. leukopenia *
B. hyperleukocytosis
V. left shift of the leukoformula
Gm. eosinophil-basophilic association
D. blastoz*
15. Pregnant anemia is different from hemolytic anemia:
A. erythrocyte viability norma*
B. erythrocyte viability decreased
V. reticulocytosis yok*
G. erythrocyte hypochromia is present*
D. intravenous hemolysis
16. What is aniked in erythrocytes typical of vit B12 and folate deficiency deficiency:
A. Jolie's body*
B. Kebot ring*
V. hyperchromia*
G. membrane pathology
D. enzyme pathology
17. The propensity for nospecific infection in hypoplastic capillary is related:
A. neutropenia*
B. anemia
V. thrombocytopenia
G. lymphopenia*
D. leukopenia*
18. Unlike hemolytic anemia, hypoplastic anemia does not occur:
A. jaundice*
B. bilurubinemia*
V. general weakness, dizziness
G. rapid heartbeat
D. urobilinuria*
19. Pathology in paroxysmal nocturnal hemoglobinuria:
A. erythrocyte menbrane*
B. in the enzyme erythrocytes
V. erythrocytes in hemoglobin
Gm. menbranopathy*
D. in the erythrocyte nucleus
20. Detected in acute leukemia leukogram (Find false):

A. neutrophillosis*
B. blastose
V. lymphocytosis
Gm. it's all right*
D. ESR decline*

Graphic organizer. Cluster scheme.

Guidelines for general blood analysis in hematological diseases.

PRACTICAL PART

Interpretation of total blood test in various blood diseases

Purpose: Interpretation of total blood analgesia, according to laboratory data.

Stages:

№ Action Procedure Failure to complete the step

(0 points) Complete the step correctly

1. In iron-deficient anemia, hemoglobin and erythrocyte are reduced, color is reduced, and microcytosis, hypochromia, and zychylocytosis are observed in erythrocyte morphology. 0 10
2. In vitro B12 deficiency anemia, hemoglobin and erythrocyte are reduced, color levels are increased, and in severe cases, leukocyte and platelet counts are reduced. In erythrocyte morphology, macrocytosis, megaloblasts, hyperchromy, zucchylocytosis, Jolly bodies, Kebot rings, neutrophil hypersegmentation are observed. 0 12
3. In aplastic anemia, the number of hemoglobin and erythrocytes, platelets, leukocytes decreases sharply, relative lymphocytosis is observed in leukoformula, ECHT increases. 0 10
4. In hemolytic anemia, hemoglobin and erythrocytes are reduced in a short time, acute reticulocytosis is observed, leukocytes increase, ECHT increases. Erythrocyte morphology identifies microsferocytosis, ovalocytosis, acanthocytosis, dentocytosis, treaded cells, sickle cells. 0 13
5. Hemoglobin and erythrocytes in acute leukemia, a sharp decrease in platelet count, increased leukocyte count, normal or decreased, blastes are detected in leukoformula, ESR increases. 0 10
6. Chronic myeloleucosis reduces hemoglobin and erythrocytes, platelets initially increase, then a sharp decrease in number is observed, leukocyte levels increase to 600-1000x109/l, myelocyte, metamielocyte, promielocytes are detected in leukoformula, ESR increases. 0 15
7. Decreased hemoglobin and erythrocytes in chronic lympholeucosis, decreased platelets, increased leukocyte levels to 200-600x109/l (absolut lymphocytosis is observed), prolymphocytes are detected in leukoformula, ESR increases. 0 15

8. In erythema, the number of hemoglobin and erythrocytes, leukocytes, platelets increased sharply, ESR decreased to 1 mm / hour 0 5

9. Decreased hemoglobin and erythrocytes in myeloma, a sharp increase in ESR (60-90 mm / hour) was observed. 0 5

10. Decreased hemoglobin and erythrocytes in idiopathic thrombocytopenic purpura, a sharp decrease in platelet count 0 5

Control questions

1. Normal hemogram rates?
2. Erythrocyte structure?
3. What pathological erythrocytes do you know?
4. Morphology of erythrocytes in various diseases?
5. Normal erythrocytes?
6. Is this poykilocytosis?
7. Is this anisositosis?
8. A formula for color rendering an indicator?
9. Hemoglobin structure?
10. Changes in hemoglobin?
11. Types of leukocytes?
12. Which potological impurities occur in neutrophils?
13. Normal values of leukoformula?
14. What is the left shift of the leukoformula and when is it observed?
15. What is the right shift of the leukoformula and when is it observed?
16. Changes in hemogram in anemia?
17. Changes in the hemogram in leukemia?
18. Changes in hemogram in hemorrhagic diathesis?
19. Leukocyte morphology?
20. Platelet information?
21. Platelet, reticulocyte count rule?
22. What factors influence the change in the ECHT.
23. When will the ECHT increase?
24. When will the ECHT decrease?

CHAPTER 2. GREAT DISEASES LABORATOR DIAGNOSTICS.

2.1.URINE ANALYSIS.

1. Objectives of the training:

- Normal urine analysis;
- Laboratory diagnostics of renal diseases.
- Methods of checking renal function.

Clinical analysis of the urine

Urine – is a fluid formed in the kidneys. The final products of substance exchange from the body through urine, excess water, various salts, some hormones, enzymes, vitamins are released.

Urine analysis not only provides information about kidney function, but also about other organs, including the liver, heart, and gastrointestinal system.

The outcome of a urine analysis often depends on its proper collection (collection time, storage conditions, cleanliness of the container, adherence to hygienic rules, amount of liquid drunk, the nature of the food product, and b.). For analysis, a morning urine is taken, the shelf life of which does not exceed 1.5 hours, stored in a cold place as much as possible. When collecting a milky urine, it should be borne in mind that the amount of liquid increases when it is drunk too much and decreases when too much sweating is observed.

Urine analysis

Milk content:

infants (1-2 days) 30-60 ml

400-500 ml under 1 year

1-3 years 500-600 ml

3-5 years 600-700 ml

5-8 years 650-1000 ml

8-14 years 800-1400 ml

women 600-1600 ml

men 800-1800 ml

250-2400 ml in the elderly

The relative density of the urine in the morning port

babies 1012

1002-1006 before the age of 1

adults 1008-1026

The maximum relative density of the Zimnitsky test is higher than 1020

Concentration index 3.0

color Simon - yellow

clarity

no protein or traces (25-75 mg/sut)

sugar (0.02% less)

There will be no acetone

No ketene bodies (50 mg / milk less)

No urobiline bodies (6 mg / milk less)

There will be no bilirubin

No ammonia (0.6-1.3 g /cut)

Profobilinogen 2 mg/l

There will be no hemoglobin
Reaction Neutral, or weakly acidic
General features

Milk content. A healthy person spends an average of 1,500 ml of urine per day. Yangi has a small amount of urine in the bladder of the newborn and it is separated by birth. Over the next 1 – 2 days, urine separation is sharply reduced due to low fluid intake (1-2 times a day) this condition is called physiological anuria. 4 – Urine levels increase by day. Children's daily urine is determined by the following formula.

600 + 100 (x – 1) = ml is separated in the daily urine norm.

But this amount varies depending on the amount of fluid you drink per day. The morning por-sentence of the urine is 150-200 ml. Decreased or increased lactic acidity is a common clinical indication.

Increased daily urine volume – more than (2000ml) is observed in physiological patients, in the third trimester of fetal-density, when the animal is finished, when protein products are under-consumed.

Polyuria, in some kidney diseases, diabetes, epilepsy, hysteria, heart disease-reversal period, alimentary dystrophy, and sugar-free diabetes (up to 4-6 l per day). Polyuria should be distinguished from the occasional urinary tract, such as the wind-blown of the urine sac.

Oliguria – Decreased daily urine (500-300 ml and less). In a healthy person, oliguria is observed when working in food limits, sweating a lot, physical stress, hot sex. Glomerulonephritis from kidney disease in pathological regions, chronic renal failure, pyelonephritis, nephrotic syndrome, as well as systemic red fever, DVS-syndrome, infectious diseases (plague, dysentery, abdominal typhoid, malaria), severe trauma, The result of the effects of drugs is observed in poisoning with lead, sulema, arsenic, skipidar.

Anuria – urinary incontinence was not observed. It is a very frightening symptom, the cause of which is severe kidney damage (acute nephritis, incompatible blood group transfusion, acute renal failure), abdominal trauma, acute peritonitis, renal piercing , catheterization of the urinary tract, can be shocking.

Isureya – independent urine separation disorder natixa holding urine in a bladder. The reason is prostate damage in men (inflammation, adenoma, cancer), urinary tract strictu-rasa (narration), spinal cord injury or injury when the urinary sac shortening function is impaired, unpleasant.

Pollakiuria – fast – fast urine separation. A healthy person urinates 4-7 times a day, and all this is done in a day, and at night the urine is observed to be separated once. In Pollakiuria, frequent peshob detachments are observed only during the day and night.

Nicutia – Abundance of nocturnal diuresis compared to daytime diuresis. Increased night diuresis is observed in diabetes, some kidney diseases, prostate hypertrophy.

Urine color. The normal urine is straw-yellow. Substances formed from pigments in the blood to the urine (urobilin, urochromes, hematoporphyrin, and b.) gives color. The color of urine varies depending on its relative density, daily volume, body-to-eat products, peripherals, drugs, vitamins. For example, when taking red-amydopyrine, taking pink-aspirin, carrots, beets, green-blue-methylene blue, brown - sulfa-nilamds, active charcoal, green-yellow-rave, alexandr leaf, orange-riboflavin, 5-NOK, observed when taking furagin. The more orange the color of the urine in the norm, the higher the relative density, and vice versa. The color of the concentrated urine is clear. But a urine of normal color does not mean a healthy human urine. Peshob discoloration is a common diagnostic sign in many diseases. For example, dark-colored heart failure (-suckling kidneys, tumors), light-colored and sugar-free diabetes, yellow disease in green-yellow drugs, pivasimon-parenchymatous jaundice, red-kidney panchigida, in renal infarction (blood or Hb), “ meat wash ”- in acute renal cold (nephritis), dark color (almost black)- acute hemolytic anemia, melanoma observed. Only changes in the color of urine residue are observed when large amounts of salts, pus, mucous erythrocytes are twisted. For example, if there are a lot of urates in the urine, it will turn into a liver-reddish, when uric acid is dark, when there are phosphates.

Clarity. The normal newly separated urine will be clear. Even in normal cholera, small amounts of opaque can be observed at the expense of epithelial cells and mucus. Significant leaching of urine is observed when erythrocytes, leukocytes, fat ‘, epithelium, bacteria, salts (urates, phosphates, oxalates). The cause of the blur was determined by microscopic and chemic analysis of urine residue. From which part of the urinary tract (urinary tract, urinary sac, renal gums) through a three-cup test. Blurring of the urine in the cold is isoxed by the presence of urates in it and phosphates in the heat.

Smell. The new urine will not have a sharp unpleasant odor. An amiak smell comes from him at the expense of alkaline ferment from a standing urine. The arrival of amiac chidi from the new urine can be observed when there is a cold in the urine bag. Atsectal odor comes from the urine during severe diabetes. A rotten smell comes in the urine bag when there is a gangrenous process. Unpleasant smell chandeliers from the urine when consuming large amounts of garlic onion or xren along with food.

Relative density. The relative density of urine (comparative weight) is determined by the concentration of dissolved substances (mochevina, uric acid, creatinine, various salts). The relative density depends on the amount of fluid

released daily — the higher the daily urine, the lower the relative density, and vice versa. In Norma, the specific weight of urine varies from 1010 to 1030. A healthy human urine is a food product consumed during the day, the amount of fluid consumed, sweating, and so on. relative density varies depending on the larvae. If the morning urine density is 1018 or higher, the renal concentration function is considered intact. When a urine contains glucose and protein, its density increases. High relative density is observed in low-grade diabetes, when tumors are increased, vomiting, diarrhea, low urine is high, (is observed when urine returns, and when urine is taken by the diocesan, in sugar-free diabetes).

Low relative density urine separation is observed in some kidney diseases, which is associated with disruption of the separation of mochevina and sodium chloride, which make up 3/4 of the solids that dissolve in urine. The relative density of the urine determines the concentration property of the kidneys. Cryoscopy method (Determination of the oscillation point by determining the osmotic concentration of the urine via) provides accurate information on the condition of the renal concentration feature. If chronic renal failure develops, the ability to concentrate decreases (this condition is called hyposthenuria; if the concentration index is below 1.8, the specific weight is less than 1018). A complete loss of osmotic concetation capacity leads to an equalization of urine and yon osmotic pressure (isosthenuria), with a concentration index of 1.0 relative weights of 1010 bsh. The concentration and water separation function of the kidneys is determined by the Folgard symbol under Zimnitsky or anhydrous product consumption conditions.

Chemical inspection of the urine.

Urine reaction. In a healthy person who feeds a mixture, the urine reaction is neutral or weakly acidic (rN 5.0-7.0). In physiological disorders, the schrelease of a urine reaction depends on the nature of the diet. Meat foods are acidic, plant products are alkaline. Heavy physical cocktails increase urine acidity, alkaline solutions (mineral water, soda) – alkaline side shch. Ideally, the urine reaction is equal to the blood rN i. Increased urine acidity (rN less than 7.0) in diabetes, chronic heart failure, gout, acute renal failure, elevated cholera, renal tuberculosis, increased potassium in the lungs, acute nephritis, alkaline reaction in cysts, After vomiting, when the amount of potassium in the blood is reduced, when the gastric acid is high at the time of digestion, the urine reaction is almost unchanged in low-acid (.

Detection of urine reaction Dissolves insignificant acidic reaction phosphates, alkaline reaction urates in urinary stone disease. Depending on this, the urine reaction can be changed if appropriate parchment and medications are selected. However, it should be borne in mind that an alkaline reaction is considered a

favorable mule for the proliferation of microorganisms that call for the inflammatory process in the kidneys and urinary tract.

Salting the urine in the room ruins increases the signage in it and leads to the disintegration of the cellular elements, which affects the result of the analysis.

Protein. There is no protein in the urine in the norm (0,002g/l). However, small amounts of protein can be observed in healthy people in the following cases: after consuming large amounts of protein-rich products, after a cold hardening, in emotional stress, after a long physical test. The formation of a significant amount of protein in the urine is a pathological condition - called proteinuria. Weak proteinuria (1g/per day), mean (1-3g / day), strong (3g / more than a day). Which fractional protein predominates by dividing the proteins in the urine into fractions on paper by the method of electropharez. If a low-molecular, small-dispersed protein dol – albumin, microglobulin proteinuria is selective, if all fractional types are identified, it is called nonselective proteinuria.

Reasons for proteinuria:

• light chain of immunoglobulins, hemoglobin, myoglobin when the formation of small dispersed proteins in prerenal prteinuria is accelerated. Bo‘ proteins are filtered more than normal, and ducts cannot reabsorb them. Mielom disease is an example of this. In this case, V – lymphocytes are differentiated to plasmatic cells and produce paroprotein from themselves. Bens – Jones protein is called easy to pass through the kidney filter and separate through the urine. As a result, myeloma develops nephropathy, followed by SBE.

• The renal prteinuria is glomerular, channel, and functional. Glomerular proteinuria is observed in acute and chronic glomerulonephritis, amyloidosis, renal disease with recurrent tissue disease, renal vein thrombosis, hypertension, atherosclerotic nephrosclerosis, renal dimming. The cause of proteinuria is increased protein filtration as a result of damage to the basal membrane of the balls. It is necessary to think about the medium and strong proteinuria and the renal proteinuria when it comes with cylinder, hematuria. When proteinuria itself comes, it should be distinguished by myelo disease and amyloidosis by the nephrotic form of chronic glomerulonephritis. The arrival of proteinuria with hematuria is typical of a mixed type of acute glomerulonephritis, acute glomerulonephritis. Channel proteinuria is observed when the proximal ducts cannot reabsorb low molecular proteins that are profiltrated by balls. It is characterized by weak proteinuria and is specific to pyelonephritis, interstitial nephritis, Fanconi syndrome

• postrenal prteinuria, urinary excretion pathways are observed in inflammatory diseases and is associated with inflammatory exudate (urine sac urinary exudate canal, genital diseases).

Functional renal proteinuria

• In young people with asthenic tanatus, it is observed when there is a lordosis in the spine for a long time in the vertical aunt, which disappears after switching to the horizontal aunt.
• After severe physical stress
• Proteinuria observed when temperature rises.
• When alimentary proteinuria consumes large amounts of protein-rich products
• Palpator proteinuria re-– after palpation of the kidneys
• Emotional proteinuria

Functional proteinuria does not exceed 1.0 g / l.

Glucose. Glucose in a normal urine is not anized in normal ways. The appearance of glucose in the blood '- was bshch in pathological and physiological disorders. When the blood glucose level exceeds 8.8 – 9.9 mmol/l, the urine begins to separate glucose – glucose. Typically, the renal threshold does not exceed 9.9 mmol/l for glucose. Physiological glucosuria is observed when taking large amounts of carbohydrates (alimentar), (emotional) after emotional stress, (caffeine, corticosteroids) when taking certain drugs. Persistent glucosuria is observed in diabetes, tireotoxicosis, cerebral tumors, Itsenko – Kushinga syndrome, liver cirrhosis. A daily urine is collected to determine the glucose in the urine.

Keton bodies. In the urine, ketone bodies (acetone, acetosirca, and b-oxymoy kislot) are clearly called ketonuria. Norma can be detected in urine up to 20 – 50 mg/sut per day. Excessive separation of ketone bodies through urine can lead to increased destruction, cold hardening, physical stress. Ketonuria is observed in the decompensation phase of diabetes, in severe toxicoses, in dysentery, in prolonged starvation, in severe tireotoxicosis when taking normal amounts of fat ' products and limiting carbohydrates

Bilirubin. Norma is not detected in the urine. The appearance of bilirubin in the urine is a pathological situation. This does not pass through the connected bilirubin kidney filter, which is connected to the connected bilirubin through the kidney filter. Separation of bilirubin by urine is observed in parenchymatous inflammation of the liver (vi-Russian hepatitis), mechanical jaundice (of the liver), cirrhosis, cholestasis. In hemolytic jaundice, bilirubin is usually not detected in the urine. To determine the presence of bilirubin in the urine, a 3 – 4 ml urine is taken into the test tube and added to it 1 – 2ml 1% alcohol solution or Lugol solution along the solution wall. If bilirubin, a green bag is formed where the two solutions are mixed.

Urobilinogen. Traces of urobilinogen can be detected in normal urine. A sharp increase in the amount of hemolytic sari (eritrocytes are broken down intravenously), toxic liver damage and inflammation are observed in intestinal

diseases (enterites, constipation) are observed. There is no urobilinogen in the urine when it is observed that the bile ducts are completely closed.

Other indicators. Herbic acids are found in urine in viral hepatitis, liver cirrhosis, diseases that cause bile ducts to close (Tumor, grass – stone disease). Amylase in the urine is primarily used in pancreatic diseases (when the path of recovery is closed, the tissue of the gland is inflamed, and necrosis is). Porfirin is detected in urinary tract in hereditary diseases (primary porphyria) and impaired liver or blood-forming organs. Norma has 67 mcg coproporphyrin, 4.4mkmol/l porphobilinogen, 14nmol uroporphyrin, 6 mg urobilinogen in the daily urine.

Check the urine residue

flat epithelium In small amounts

transient epithelium In small amounts

there will be no renal epithelium

Leukocyte 0-3 (er.) i 0-6 (female.) in the field of view

Erythrocyte in 0-2 preparations

There will be no cylinder

Low amount

Bacteria will not

In an inorganic residual acid reaction- uric acid crystals, urethra; alkaline reaction- amorphous phosphates, mochexis ammonium, trypelfosphates; oxalates - all types of salts are detected in small amounts in any reaction.

Examination of urine by Nicheporenko method leukocyte- 4000, erythrocyte- 1000 1 ml; cylinder - 0-1 4 when counting the camera

Amburge method

Erythrocyte 1.5-102 / min

Leukocyte 2.5-102 / min

Zimnitsky's test is 65-75% of the fluid in the dairy urine. Kunuzgi diuresis make up 2/3- 3/4 of the daily urine. Relative 1004-1024.

For microscopic examination of urine residue, the first port of the morning urine is taken, which is brought to the laboratory in an aunt no later than 1 – 1.5 hours. Because 50% of the cells in the urine, especially if there is an alkaline muhit or a low-density urine, decomposes after 2 to 3 hours.

Organic residue.

Erythrocytes. There will be no erythrocytes in 1 field of view under the microscope or 1 - 2 cells may appear in the norm. Detection of erythrocytes in the norm is observed in those with heavy physical cocktails or sports. Significant amounts of erythrocytes are visually identified in the urine. If the urine reaction is acidic, the brown color will turn red if the reaction is alkaline or neutral. A small number of erythrocytes are visible under the microscope. Erythrocyte separation by

urine is specific to renal and urinary tract diseases (acute and chronic glomerulo and pyelonephritis, urinary stone disease, tumor, infection, prostate adenoma). In addition, erythrocytes are observed in malaria, smallpox, hemorrhagic lixoradca, intestinal tumors, infectious mononucleosis, impaired blood clotting, heart failure, and increased dose of (sulfanilamides, urotropin, anticoagulants. If the number of erythrocytes increases, the number of cylinders also increases, the process does not increase from the intra-renal hematuria to the daloate, cylinders, and protein, the process is non-renal (mn., Urinary tract). Glomerular and noglomerular hematuria are different. Finding more than 80% of altered erythrocytes in urine is glomerular hematuria (The main reason is glomerulonephritis), 80% of the discovery of unchanged erythrocytes is called noglomerular hematuria.

Leukocytes have a large bundle difference of 2-4 times that of erythrocytes, erythrocytes do not have grains, and there are two contours. Leukocyte grain and cytoplasm are clearly visible in the succulent acid urine, leukocyte grain loss in the alkaline rush, and the swollen contour becomes indistinct. Norma men have a field of vision of 0 – up to 0 – 5 in women. But heavy physical cocktails can be 1.5 – 2 times higher than the norm. Urinary excretion of leukocytes in renal and urinary tract diseases (acute and chronic pyelonephritis, cystitis, urethritis, acute and chronic glomerulonephritis), when the destruction rises. The fact that the amount of leukocytes is more than 50 – 60 in one area of vision indicates the acuteness of the inflammatory process. In such a case, the bacterial bath is detected in the urine. Absence of bacteria (Absence when sown) tuberculous or lupus – nephritis. A 3-cup test is performed to determine exactly where the allergy process is. The discovery of leuko-cytes in the first glass indicates that the inflammatory process is urethrade, the discovery in the second, third cup is inflamed in the prostate gland, and the discovery in the whole glass is an inflamed urine bag, loxanka. The presence of cylinders and protein in the urine other than leukocytes indicates that the process is renal. Increased leukocytes in the urine are observed after taking ampicillin, aspirin, heroin.

Epithelial cells are always present in the urine residue.

Flat epithelial cell. The urine falls from the vaginal, external genitalia, urinary tract. It is a single-core, fine-grained cytoplasm cell in a broad, oval, or round shape. In leukocytes, 3 – 4 times larger, the drug is placed alone or in the ball.

Transient epithelial cells. Urine sac,pelvis of the kidney, urine tubes fall into the urine as a result of desquomation of the mucous layer. Usually these cells are painted in different shapes, sizes and yellows.

Renal epithelial cells. Urine ducts are formed from the epithelium. These are small round or cube-shaped, large-core, granular, yellow cells with vacuoles in the cytoplasm. There is always a flat and transient epithelium in the normal urine, and

their proliferation is of little diagnostic importance, and one or two kidney epithelium can also occur. Renal epithelium has diagnostic significance when accompanied by leukocyturia, hematuria, or cylinderuria. This condition is observed in pyelonephritis, acute encrosis of the ducts, poor quality nephrosclerosis, toxic effects of salicylates, in heavy metals, in poisoning with ethylene glycol.

Cylinders. They are formed in plasma proteins that pass through a glomerular filter. The simplest cylinder – is a gialin – protein cylinder. If a channel cell is located in a hyaline cylinder, an epithelial cylinder is formed, if only the nucleus is located – a granular cylinder, the erythrocyte is located, the erythrocyte cylinder is called a leukocyte cylinder.

Gialin cylinder. Reversing with proteinuria is found in all kidney diseases. The appearance of hyaline cylinders in the urine indicates an increase in the permeability of the ball capillaries.

Epithelial cylinders. Acute necrosis of the renal ducts, poisoning with heavy metals, ethylene glycol, intoxication of salicylates, nephrotic syndrome. The appearance of epithelial cylinders in the urine indicates damage to the tubular apparatus of the kidneys.

Separately cylinders. The appearance in urine indicates a degenerative dystrophic change in the epithelium of the proximal ducts of the kidneys. Glomerulonephritis, pyelonephritis, diabetic nephropathy, renal amyloidosis, poor quality hypertension are detected in urine.

Wax cylinders. It is formed when strong atrophy is observed at the expanded part of the distal part of the canals. Acute subterranean malignant glomerulonephritis, renal amyloidosis, renal failure are detected in urine. The appearance of a waxy cylinder in the urine is a sign of the severity of a severe pathological process in the kidneys.

Erythrocyte cylinders. It is detected in hematuria belonging to the kidneys and indicates pathololgy of the balls. Glomerulonephritis, renal tumor and infarction, renal vein thrombosis, acute bacterial endocarditis.

Leukotsitar cylinders. Pyuria is formed in the renal ducts when observed, and purulent diseases of the kidneys are specific to pyelonephritis.

Inorganic residue. uric acid, urates, oxolate crystals, amorphous phosphates, tripelphosphate, mochexisli ammonium are found in the acidic urine.

Uric acid crystals. It is yellow in color and easily soluble in alkali, insoluble in leic acid.

Urats uric acid salts, small grains of amorphous brown or pink color. Soluble in alkali and heated.

Oxalates. Calcium shaveluxus salts are soluble in hydrogen chloride and insoluble in alkali and acetic acid.

Crystalline derivatives urine reaction Clinical significance

Pathologically acidic reaction of uric acid urine

1. High concentration of urine in hypovolemia (vomiting, diarrhea, fluid restriction, oliguria)

2. Increased tissue breakdown (decomposing tumor, leukemia, pneumonia)

Urates – uric acid salts (ammonium, sodium, potassium, magnesium, calcium) acidic reaction of urine 1. High concentration of urine in hypovolemia (vomiting, diarrhea, fluid restriction, oliguria)

2. Increased tissue breakdown (decomposing tumor, leukemia, pneumonia)

Calcium oxalate is often an acidic reaction, sometimes alkaline 1. when foods rich in Shavel acid (tomatoes, spinach, shavel, apples, grapes, oranges) are consumed.

2. (shavel acid diathesis) when substance exchange is impaired

3. poisoning with ethylene glycol (antifreeze, brake fluid)

Tripelfosphates Only alkaline reaction

1. when large amounts of plant products are taken.

2. cystitis, pielolnephritis, when accompanied by an alkaline reaction.

amorphous phosphates (calcium and magnesium phosphate,) Alkaline reaction 1. multiple vomiting, loss of (NSI), when administered with alcohol.

2. when the intestinal function is impaired.

3. (raxit, phosphate - diabetes) as peshob separation of phosphates increases.

Alkaline reaction. Normal or acidic reaction in breast age 1. Inflammation of the urinary tract.

2. Alkaline exaggeration of the urine.

Mucus is not present in normal urine. Urinary tract can be detected in inflammatory diseases (cystitis, urethritis, urolithiasis, prostatitis). Bacteria are found in inflammatory diseases of the urinary tract and kidneys when there are more than 100,000 bacteria in 1 ml of urine.

METHODS OF TESTING KIDNEY FUNCTION.

Nechiporenko test.

Counting shaped elements in 1 ml of urine. For examination, urine can be taken at any time of the day (preferably in the morning). A medium portion of urine is taken. 5 10 ml of urine will be enough. The urine is centrifuged and 0.5 ml (500 μl) of urine is left with the residue. The residue is mixed and placed in a Goryaev chamber with a volume of 0.9. Under the microscope, the number of leukocytes, erythrocytes, cylinders is counted separately. First, the shaped elements in 1 μl of urine are calculated using the formula. and then in 1 ml of urine:

N Shaped elements in 1 ml of urine, X Shaped elements in 1 µl, volume of 500 urine (in µl), left in the tube with residue, V amount of urine taken for centrifugation. Normally, 1 ml of urine contains 2000 leukocytes, up to 1000 erythrocytes, cylinders are almost undetectable. If leukocytes are increased, it is characteristic of urinary tract inflammatory diseases and pyelonephritis, increase of erythrocytes, glomerulonephritis, urinary stone disease, kidney tuberculosis, kidney infarction.

Zimnitsky test.

Through the test, it is possible to determine the amount of urine released in the dynamics and the relative density of urine during the day. Test conditions:

the patient should not have swellings and boils on the head

Diuretics should not be taken on the day the examination is canceled

Avoid excessive fluid intake

if the above conditions are not met, the amount of artificially secreted urine will increase and the relative density will decrease, as a result the test will be incorrect. For the test, daily urine is collected, urine every 3 hours is collected in a separate container. The name of the patient and the order number of the container are displayed on the surface of the container. The patient urinates at 6 o'clock, but this urine is not collected for testing. If the patient does not urinate for 3 hours, this container is left empty and the next container is collected. The amount of water drunk on this day is also taken into account. In the laboratory, the amount of urine in each container, relative density, diurnal diuresis, daytime diuresis, night diuresis are calculated. If the ability of the kidneys to osmoticly dilute and concentrate urine is maintained throughout the day:

1. The amount of urine in each portion varies from 50 to 250 ml.

2. the difference between the maximum and minimum relative density should be significant, that is, it should not be less than 0.012 0.016 (mn, from 1006 to 1020 or from 1010 to 1026)

3. daytime diuresis should be clearly superior to nocturnal diuresis.

The maximum relative density in young people is not less than 1025, in people over 45 50 years old it is not less than 1020 1022, and the minimum relative density is 1010 1012.

Concentration of urine occurs at night and is accompanied by a decrease in diuresis, an increase in relative density to the maximum limit. This is due to increased secretion of ADG and increased osmotic pressure due to increased reabsorption of water in the distal tubules and collecting tubules and urea reabsorption in the collecting tubules in the cerebral part of the kidneys.

Dilution of urine is due to diurnal diuresis, which is associated with a decrease in the reabsorption of water and urea in the distal part of the ADG and in the

collecting ducts. In Zimnitsky's test, even in night urine, the relative density is less than 1020 (hypostenuria). A decrease in concentration properties is observed in diseases with a decrease in osmotic pressure in the cerebral cortex:

Acute glomerulonephritis

UPA: urine density is normal, hematuria, proteinuria, cylinduria (erythrocytic, hyaline), kidney epithelium is determined.

BQA: increase of a2 and g-globulins (ASL-O, ASG), hypoalbuminemia in nephrotic syndrome.

UTT: enlarged kidneys

The Reberg-Tareev test is a decrease in glomerular filtration

Acute interstitial nephritis

UPA: hypostenuria, proteinuria, microhematuria (10-30/1), moderate leukocyturia, the appearance of eosinophils, cylinduria (hyaline, waxy, granular), oxalaturia, calciuria.

UQA: anemia, leukocytosis with a shift to the left, eosinophilia, increased ECHTN.

BQA: sialic acid, seromucoid, haptoglobin, creatinine, increased urea, decreased potassium, sodium.

The Reberg-Tareev test reduces glomerular filtration and tubular reabsorption

Chronic pyelonephritis

UQA: anemia, leukocytosis with a shift to the left, toxic granularity of neutrophils, increased ECHT.

UPA: cloudy urine, alkaline reaction, decreased density, moderate proteinuria, microhematuria, strong leukocyturia, bacteriuria (100,000 microbial cells in 1 ml)

Nichiporenko test leukocyturia.

Zimnitsky test density reduction

BQA: increased sialic acid, seromucoid, creatinine, urea, emergence of SRO.

SBE

An increase in blood creatinine of 0.08-0.1 mmol/l. A decrease in glomerular filtration rate from 80-120 ml/min, a decrease to 40 ml/min indicates severe SBE, a decrease to 15-5 ml/min indicates the terminal stage. Urinary excretion of creatinine decreases from 1-3.2g/milk, urea below 20-35g/milk.

UPA: albuminuria, cylindruria, microhematuria, isohypostenuria.

UQA: anemia, leukocytosis, thrombocytopenia

ANALYTICAL PART

The new pedagogical technology used in the lesson "Romashka"

Chamomile leaves are drawn on hard paper and cut out. On the back of each sheet, questions on the topic "clinical analysis of urine analysis, laboratory diagnosis of

kidney diseases and general analysis of stool analysis" are written. Each student takes one daisy leaf and answers the written question.
Questions:
What is determined in the macroscopic examination of urine.
What is the Nechiporenko test?
Urine collection procedure for the Zimnitsky test.
Changes in urine in acute glomerulonephritis.
Changes in urine in chronic glomerulonephritis
Changes in urine in acute pyelonephritis.
Changes in urine in chronic pyelonephritis
Urine analysis in OBE.
Urinalysis in SBE
Urine analysis in diabetes.

Situational issues.
Issue #1
Patient V is 20 years old. He came to the hospital. AKB has been rising for 10 years. At the same time, swelling and headaches are observed in the legs, arms, face. He was treated several times in nephrology departments. The last digging started a week ago after a cold. Objective: his condition is serious, lethargic, he answers questions quickly, his mouth smells of urea, his breathing is noisy, 30 breaths per minute. The skin is dry, there are traces of scratched nails. AKB 220/120 - 180/110 mm wire is on top. UPT: protein 1.165 oo/o, macrohematuria, cylinders 1-2. Blood urea - 28 mmol/l, creatinine - 0.7 mol/l.
Questions:
Make a clinical diagnosis.
Stage of the disease.
What inspection methods do you recommend?
Answer:
Chronic renal failure.
Terminal period
Rehberg's test and kidney UTT
Issue #2.
A 20-year-old teenager noted that swelling appeared on his face, arms and legs, general weakness and reduced diuresis after being exposed to cold. He was treated as an inpatient at the place of residence, after 2 months, after no effect, he was transferred to the nephrology department. Lens: skin layers are pale, dry. Swelling and ascites are detected on the face and arms. In open percussion, the percussion sound is below the edge of the shovel, and during auscultation, the breath is

sharply reduced in this area. Heart tones are rhythmic. A/B 90/60 mm wire top. UPA: color meat wash. The clarity is not clear.
Relative density -1020, alkaline reaction, protein -2.3 g/l, glucose abs, ketone bodies abs, bilirubin abs, urobilinoids abs, horse acids abs, epithelium - 14-15/1, leukocyte 5-6/1,
erythrocyte in abundance, cylinder erythrocyte, mucus +, salt -, bacteria -.
Question:
1. What disease is the analysis typical for?
2. Disease-specific changes in the analysis?
3. What additional investigation is needed
Answer:
1. Acute glomerulonephritis
2. The color is meat wash, protein - 2.3 g/l, erythrocyte - in large quantity.
3. Kidney UTT, Reberg's test, Zimnitsky's test, blood urea and creatinine
Issue #3.
A 42-year-old patient. I have been suffering from type I diabetes for 15 years. Xar takes 60 ED insulin daily. From 2 years there is an increase in buen A/B: up to 180/100 mm wire. 6 months ago, swelling appeared on the legs, the swelling began to spread throughout the body, shortness of breath, dry mouth, nausea, diuresis decreased sharply.
In the examination: oxil 3.9 g/l in the general urinalysis. In mine analysis: total oxygen 50 g/l, cholesterol 10 mmol/l, sugar content 16.3 mmol/l, glomerular filtration 35 ml/min. Urea 12 mmol/l, creatinine 0.3 mmol/l.
Question:
1. What disease is the analysis typical for?
2. Disease-specific changes in the analysis?
3. What complication occurred in the patient?
Answer:
1. Type I diabetes
2. Oxygen in UPA is 3.9 g/l, filtration of balls is 35 ml/min. Total oxygen in the blood is 50 g/l, cholesterol is 10 mmol/l, sugar is 16.3 mmol/l, urea is 12 mmol/l, creatinine is 0.3 mmol/l.
3. Diabetic nephropathy SBE.
Issue #4
A 40-year-old patient suffered from chronic bronchiectatic disease and decreased diuresis, swelling in the legs, lower back, and face. Lens: pale skin, "soft" swellings all over the body. A/B 120/70 mm wire top.
In general urinalysis: oxil 6.6 g/l, microhematuria, cylindruria.

General mine analysis: hemoglobin 90 g/l, leukocyte-8x109/l, SOE 45 mm/s, cholesterol 7.5 mmol/l, urea 6.5 mmol/l, creatinine 0.12 mmol/l.

Question:

1. What complication occurred in the patient?
2. Tell the methods of additional examination that confirm the diagnosis.
3. Recommendation.

Answer:

1. Renal amyloidosis SBE
2. Kidney UTT, puncture biopsy
3. Nephrologist consultation

Issue #5

The patient complains of headaches, ringing in the ears and head, body temperature periodically rising to 39o C, back pain. He has been ill for 7 years. Objective: average weight, skin and visible mucous membranes of normal color, clean, pulse 88 per 1 min., rhythmic. AKB 150/100 mm s.u. The borders of the heart are around the norm, the tones are slightly mixed. Other parts are unchanged. General analysis of blood: Er-3,2; Hb -104 g/l, RK 0.85; Lake - 9.3; ECHT-28 mm/h. General analysis of urine: protein-0.33 oo/o, leuk.-20-30/1; erit.- 1-2/1, active leukocytes are detected.

Question:

1. Initial diagnosis.
2. What additional checks are necessary?
3. Comparative diagnosis.

Answer:

1. Chronic pyelonephritis.
2. Kidney UTT, Nicheporenko test.
3. Chronic glomerulonephritis, kidney failure

Issue No. 6

A 26-year-old woman named B. complained of an increase in body temperature up to 37.80C, chills, back pain, and painful urination. She is 32 weeks pregnant. Lens: the skin is leaking, the patient is thin. Swollen under the eyes, AKB 160/100 mm s/u. Auscultation: heart sounds are muffled, there is a systolic murmur in all auditory points, tachycardia is detected. The tongue is dry, drooling. The abdomen is soft, the liver and spleen are not enlarged. Pasternatsky's symptom is sharp "+" in the mind, weak "+" on the left. General analysis of blood: Er-3,2; Hb -100 g/l, RK-0.8; Lake - 12.5; ECHT-26 mm/h. Urine analysis: amount-350 ml, cloudy. Comparative weight 1015; oxyl 0.66oo/o, cand-abs; deposit microscopy leuk.- 6-8/1; Land 5-8/1; epithelium 10-16/1, bacteria +; Urinalysis according to Addis-Kakovsky: Er-3500; Lake-1600.

Question:
1. Initial diagnosis.
2. What additional checks are necessary?
3. Comparative diagnosis.
Answer:
1. Late toxicosis of pregnancy
2. Kidney and fetal UTT, blood urea, creatinine
3. Chronic pyelonephritis, glomerulonephritis.
Issue #7
The color is yellowish, clarity - dull, relative density -1015, reaction - alkaline, protein -1.0 g/l, glucose - abs, ketone bodies - abs, bilirubin - abs, urobilinoids - abs, bile acids - abs, epithelium - 4-5/1, leukocyte -45-56/1, erythrocyte - 2-3/1, cylinder leukocyte, mucus - ++, salt -, bacteria - +++
Question:
1. What kind of disease is the analysis?
2. Disease-specific changes in analysis?
3. differential diagnosis.
Answer:
1. Acute pyelonephritis.
2. turbidity, leukocyturia, leukocyte cylinders, bacteria.
3. acute glomerulonephritis, OBE.
Issue #8.
Red color, clarity - not clear, relative density -1020, reaction - acidic, protein - abs, glucose - abs, ketone bodies - abs, bilirubin - abs, urobilinoids - abs, bile acids - abs, epithelium - 4 -5/1, leukocyte – 5-6/1, erythrocyte 6-9/1, cylinder,
Mucus - +, salt urates +++, bacteria -.
Question:
1. What kind of disease is the analysis?
2. Disease-specific changes in analysis?
3. differential diagnosis.
Answer:
1. kidney stone disease.
2. reddish color, erythrocyturia, salts.
3. renal edema, glomerulonephritis, OBE, SBE.
Issue #9.
Color beer color, clarity - clear, relative density -1020, reaction - neutral, protein abs, glucose - abs, ketone bodies - abs, bilirubin +, urobilinoids +, bile acids +, epithelium - 4-5/1, leukocyte – 5-6/1, erythrocyte 1-2/1, cylinder – , mucus +,
Salt -, bacteria -.

Question:
1. What kind of disease is the analysis?
2. Disease-specific changes in analysis?
3. Diff. diagnosis.
Answer:
1. parenchymatous jaundice.
2. beer color, bilirubinuria, urobilinuria.
3. hemolytic anemia, mechanical jaundice.
Issue #10.
Patient M, 46 years old. Complaints: general weakness, rapid heartbeat, pain in the bones. Anamnesis: sick for a year. He was treated by a therapist for "kidney" disease. AKB is normal. Hemogram: Hb 80 g/l, er.2.2x1012/l, RK 0.8, thrombocyte 150x109/l, leukocyte 3.2x109/l, t/ya 3%, s/ya 54%, eosinophil 2%, basophil 1%, lymphocyte 38%, monocyte 2%, ECHT -76 mm/h. Urinalysis: oxil 3.3%, epit. 1-2/1, leukocyte 3-4/1, melt. 0-1/1, TB. 0-1/1. Biochemical analysis of blood: urea 22 mmol/l, total oxygen 120 g/l.
Question:
1. Your initial diagnosis?
2. Disease-specific changes in analysis?
3. What complications can be observed in the disease?
Answer:
1. myeloma disease
2. a sharp increase in total protein in blood and urine, an increase in ECHT
3. pathological fractures, myeloma nephropathy

TESTS.

What method is used to determine the shape elements in 1 ml of urine
Zimnitsky test
Garrison test
Nicheporenko test
Nikolaev test
Addis is the goal of maintaining the Kakovsky method
Glucose in urine
Shaped elements in 1 ml of urine
Relative gravity of urine
Worms in the garbage
Shaped elements in urine collected in one day
The purpose of Zimnitsky's film

Determination of the relative density of urine collected every 3 hours during the day
Study of the chemical composition of urine
Examination of urine sediment
Determination of the amount of urine in one week
Watch the urine. Planting for infection
Normal urine reaction
Alkaline
Neutral or slightly acidic
Acidic
Weakly alkaline or neutral
Like the correct answer
How can glucose be determined in urine?
Larionov test
Fonio test
Simmons test or reaction with hcl
Gaines test, glucose oxidase test
Nicheporenko method
Normal physical properties of urine
color straw yellow, clear, pH 7.0 8.0
color straw yellow, clear, pH 5.0 7.0
color straw yellow, clear, pH 3.5 4.0
color is orange yellow, dull, pH 3.5 4.0
orange color, clear, pH 3.5 4.0
The reason for the excretion of glucose from the urine
pressure drop
diuretic hormone secretion
pressure increase
poor nutrition
diabetes mellitus
In which pathology does bilirubin appear in the urine?
arthritis
viral hepatitis
cirrhosis
jade
diabetes mellitus
In which pathology, fatty acids in the urine increase
Poisoning
TTA

Pneumonia
Viral hepatitis
Everything is correct
Normal amount of erythrocytes in urine
67
1-2
14
130
29
The amount of epithelium in the urine
67
4-5
14
130
29
How much urea is excreted in the urine per day
1g
3.7 g
4.9g
1.9g
Reagent needed to detect diastase in urine
1% starch solution
Dezrastvor
Furacillin
5% starch solution
A method for the determination of Bence Jones oxide in urine
to boil
Fizrastvor 0.9%
1% starch solution
2% starch solution
What methods can be used to detect bilirubin in urine?
Foujet and Rosen
Quicken Schetta syndrome
Lyell's syndrome
Stevens Jones syndrome
In which pathology, when urine is examined, a positive reaction to ut pigments is observed.
viral hepatitis
syndrome Quicken SHetta

Lyla syndrome
Stevens Johnson syndrome
In which pathology, oligouria is observed
pyelonephritis
acute nephritis
Diabetes without sugar
nephrotic syndrome
In any of the listed diseases, the relative density of urine can be high (1030-1050).
chronic nephritis
pyelonephritis
diabetes
diabetes mellitus
In which disease is ketonuria observed?
chronic nephritis
pyelonephritis
diabetes
diabetes mellitus
normal number of bacteria in urine
50000/ml
150000/ml
200000/ml
300000/ml

CHAPTER. LABORATORY DIAGNOSTICS OF LIVER DISEASES.

1. Objectives of the training:
Normal biochemical blood analysis;
laboratory diagnosis of liver diseases.
liver function test methods.
Clinical analysis of biochemical blood analysis.
3.1. Biochemical blood analysis.
Blood for biochemical analysis is usually taken on an empty stomach, from the medial or lateral subcutaneous vein of the arm.

Proteins

Proteins are nitrogen-containing high-molecular organic compounds that contain more than 20 amino acids. Simple proteins consist only of amino acids, and complex proteins (lipoproteid, glycoproteid, nucleoproteid, chromoproteid) contain non-protein components other than amino acids: lipids, carbohydrates, nucleic bases, chromogen, etc. substances are composed. Proteins play a central role in the metabolism of the human body and perform the following functions:
Structure (forms the basis of the structure of cells, organelles, fibrillar proteins);

transport (lipoprotein, albumin, hemoglobin);
contraction (muscle proteins actin, myosin);
catalytic (enzymes);
regulator (hormones);
protection (immunoglobulins, antibodies, interferon; proteins in blood coagulation and fibrinolysis system);
energetic (18% of the energy consumed is generated from the breakdown of proteins);
The normal intensity of tissue and organ protein biosynthesis depends on several factors:
protein overflow (not less than 100 g/s) rich in amino acids that cannot be adequately replaced by food.
stomach (pepsin, gastrixin), pancreatic (trypsin, chymotrypsin, carboxypeptidase A and B, elastase), and small intestine (enteropeptidase) enzymes ensure complete satiety in the digestive system.
Absorption of protein hydrolysis products in the small intestine depends on the state of the small intestine mucosa, motility, and amino acid transport proteins.
in organs and tissues (primarily in the liver), delivery of energy for protein biosynthesis (ATF, GTF) and control of this process by anabolic hormones (sex hormones, insulin, STG) and vitamins (S, V6).
Violation of any of the above factors can lead to protein biosynthesis disorder and protein deficiency. The amount of protein in the plasma ranges from 65 to 85 g/l. 90% of plasma proteins are albumin, globulin, fibrinogen.
Albumin is a homogeneous fraction of simple proteins, synthesized almost exclusively in the liver. 40% of albumin is located in plasma, 60% is located in cell fluid. The main functions of albumin are maintaining colloid osmotic pressure, participating in the transport of many endogenous and exogenous substances (free fatty acids, bilirubin, steroid hormones, magnesium, calcium ions, antibiotics, cardiac glycosides, barbiturates, aspirin).
Globulin - blood serum has 4 different fractions (a1, b, a2, g), each of which consists of several proteins with different functions. α1 - globulin contains 2 proteins:
α1 - antitrypsin, an inhibitor of a number of proteases (trypsin, chymotrypsin, kallikrein, plasmin);
α1 - glycoprotein, participates in the transport of rogesterone and testosterone and binds a certain part of these hormones.
α2 - globulins consist of the following proteins:
α2 - macuroglobulin is an inhibitor of a number of proteolytic enzymes (trypsin, chymotrypsin, kallikrein, plasmin, thombin), synthesized outside the liver;

haptoglobin binds free hemoglobin A and ensures its transport to the reticuloendothelial system;
ceruloplasmin has an oxidizing property and converts divalent iron into trivalent iron and transports it with transferrin.
Apoproteid A, V, S, lipoprotein follow.
B-globulin fractions also consist of several proteins
transferrin is involved in trivalent iron transport.
Hemopexin is a transporter of free heme and porphyrin, binds heme-containing chromoproteins (hemoglobin, myoglobin, catalase) and delivers them to the cells of the reticuloendothelial system.
lipoproteins
part of immunoglobulins
protein components of the compliment.
g - globulins - these are immunoglobulins that act as antibodies against harmful substances in the form of antigens in the body. There are several classes of immunoglobulins (Ig G, Ig A, Ig M, Ig D, IgE).
Fibrinogen extrinsic factor of blood coagulation system (factor I) forms the basis of a blood clot, forming a three-layered membrane that holds blood cells in it.
Total protein is determined by the biuret method, albumin by the photometric method, protein fractions by the electrophoresis method.
The main reasons for changes in the amount of total protein and albumin in the blood serum.
Nitrogen storage components of blood
Residual nitrogen is a good indicator of metabolism in the body. The norm is 14-28 mmol/l, or 20-40 mg/dl. Residual nitrogen content includes urea (50%), amino acid (25%), creatinine (7.5%), uric acid (4%), ammonia and indican (0.5%).
Urea.
As a result of the process of deamination of aminoxolates, purine and pyrimidine bases, biogenic amines and other nitrogen-preserving substances, ammonia is produced, and urea is neutralized by synthesizing urea. The process takes place in the liver, and 1 molecule of ammonia and 1 molecule of aspartic acid produce 1 molecule of urea, fumarate, ornithine. Urea is excreted through the kidneys. Its amount in blood serum is determined in two ways. 1. color reaction with diacylmonooxime. 2. method with urea. The normal blood serum level is 2.7-8.3 mmol/l, or 20-50 mg/dL. The amount of urea in blood serum depends on the intensity of its synthesis and excretion through the kidneys.
Causes of increased urea:
kidney nitrogen excretory function disorders (acute and chronic kidney failure).

violation of the flow of urine in the urinary tract. Urinary tract compression, hangovers, bladder paresis, urethral stricture, prostate cancer or adenoma (extrarenal oliguria and anuria).

congestive heart failure, vascular insufficiency, transient shock with a decrease in renal glomerular filtration.

dehydration of the body, frequent vomiting, diarrhea, increased diuresis.

increased protein catabolism (starvation, cachexia, poor-quality hangovers, leukemia, light disease, severe burns, bleeding from the gastrointestinal tract).

In acute and chronic kidney failure, the amount of urea increases significantly, and the concentration of other nitrogen storage substances (creatinine, uric acid) also increases, and the functional tests of the kidney are also disturbed. Hyperazotemia outside the kidney occurs only with an increase in the amount of urea and changes in the residual and relative density of urine, and functional tests of the kidneys are not changed.

Reasons for the decrease in the amount of urea: in severe liver diseases (cirrhosis, chronic active hepatitis) due to a violation of urea synthesis in the liver. As a result, the neutralization of ammonia is disturbed, and this is considered one of the causes of hepatic coma. The concentration of urea in the blood can decrease when the protein-containing products are on a diet and the absorption of amino acids through the intestines is disturbed (celiac disease).

Creatinine. End product of creatine metabolism. Arginine, glycine, and methionine are produced from 3 amino acids in the liver and spleen. As a result of its combination with phosphorus, creatine phosphate is formed, and it is considered a necessary source of energy for muscle contraction. As a result of energy expenditure, creatinine is produced from it. Creatinine is not reabsorbed by the renal tubules, it is completely removed from the body by the kidneys with the help of glomerular filtration. This indicator is useful for determining the level of glomerular filtration, for which creatinine clearance in blood and urine is checked. The amount of creatinine in the blood is determined photometrically using picric acid. The concentration of creatinine in blood serum is 1-2mg/dl or 60-125mmol/l.

Increase in serum creatinine. A sign of reduced kidney function, primarily glomerular filtration.

Decrease. It can be observed when the muscle mass decreases.

Uric acid. Nucleotides (RNA, DNA) and nucleoproteins are the final product of the metabolism of purine bases. It is synthesized in the liver and completely excreted by the kidneys. The amount in blood serum is determined by the phosphorotungsten method. The normal concentration in blood serum is 3-4mg/dl or 180-530mmol/l. The change in its amount depends on its formation in the liver and excretion through the kidneys.

Causes of increased uric acid in the blood serum:

gout, the acceleration of uric acid synthesis is associated with an increase in the activity of phosphoribosylpyrophosphate synthetase, a catalyst for the synthesis of purine bases.

some blood diseases (leukemia, polycythemia, V12-deficiency anemia, poor-quality asthma, burns and other transient diseases with a large amount of protein breakdown)

some internal gland diseases (acromegaly, hypoparathyroidism, diabetes).

disorders of excretion of uric acid through the kidneys (kidney failure, lead nephropathy, polycystic kidneys, acidosis, fetal toxicosis).

eating foods rich in purines (meat, liver, kidney).

obesity, hyperlipoproteinemia, atherosclerosis, arterial hypertension.

Decrease:

hepatocerebral degeneration of the liver Wilson Konovalov's disease.

lymphogranulomatosis.

myeloma disease.

Enzymes

Protein is a natural substance that ensures the normal course of all chemical reactions in the body. They are characterized by high specificity for the substrate they act on, which is explained by their protein nature. The active part of the enzyme is the place where the substrate binds and must exactly match the shape of the substrate molecule. Even a small disturbance reduces the activity of the enzyme. Factors that show the activity of enzymes in blood serum:

the rate of enzyme synthesis in cells.

the rate of enzyme exit from the cell, this condition is observed when the cell membrane becomes damaged or is damaged by inflammation, ischemia, dystrophy, necrosis, autoimmune processes.

the rate at which cells remove enzymes from interstitial fluid by inactivating, breaking down, or excreting through urine or urine.

change in the activity of natural inhibitors and activators of enzymes.

Depending on which reaction they catalyze, enzymes are divided into 6 groups, groups and classes. 3 groups of enzymes can be found in blood serum.

cellular enzymes are catalysts of non-specific (pertaining to all tissues) or organospecific (pertaining to one organ or tissue) metabolic reactions in the cell. In the human body, the activity of these enzymes is low, but their pathological synthesis may increase or their activity may increase as a result of cell damage.

enzymes secreted in the body are formed in some organs and tissues (lipase, α-amylase, alkaline phosphatase).

enzymes that perform special physiological functions for plasma and are synthesized in the liver.

Aminotransferases. participates in nitrogen exchange, breaks down unused amino acids in the process of biosynthesis. They are the catalyst of the repletion reaction, that is, the exchange of an amino group between an amino acid and a keto acid. Pyridoxal phosphate, an amino acid coenzyme. All amino acids except lysine and threonine are affected by aminotransferases. The worst of these are aspartate aminotransferase and alanine aminotransferase.

Aspartate aminotransferase. Catalyst of reamination reaction between asparagine and α-ketoglutaric acid. Aspartic acid loses its amino group and becomes shavelux acid. α-Ketoglutaric acid binds amino group and turns into glutamic acid.

Alanine aminotransferase. A catalyst for the reamination reaction between alanine and α-ketoglutaric acid. losing the alanine amino group, it turns into pyruvic (pyruvic) acid. α-Ketoglutaric acid binds amino group and turns into glutamic acid. Glutamine and aspartic acid participate in the biosynthesis of urea and neutralize ammonia formed in the body. Aminotransferases are found in all organs, but are more active in the liver, skeletal muscles, heart, and kidney. The activity of aminotransferases in erythrocytes is 6 times higher than in blood serum. When damage is observed in these organs, the amount of aminotransferases in the blood increases. Reasons:

necrosis or damage of liver cells (acute viral hepatitis, chronic hepatitis, liver cirrhosis, liver tumors, alcohol intoxication, obstructive jaundice, taking hepatotoxic drugs).

acute myocardial infarction, acute myocarditis

skeletal muscle necrosis or trauma.

erythrocyte hemolysis.

In clinical practice, the ratio of AST/ALT activity in blood serum (de Ritis coefficient) is of great importance.

AST activity is higher than ALT in acute myocardial infarction. Ritis coefficient is higher than 1.3.

ALT activity is high in acute viral and chronic hepatitis, especially in the early period. Ritis coefficient is less than 1.0.

γ - glutamyltranspeptidase is a transferase that participates in nitrogen metabolism. A reaction catalyst that transfers a glutamine group to an acceptor peptide or L-amino acid. Enzyme activity does not exceed 66-106 ME in the norm determined in liver, kidney, pancreas. Increased activity is observed in the following pathological conditions:

in obturation of intrahepatic and extrahepatic horse ducts

liver diseases (hepatitis, liver cirrhosis, metastases of liver tumors), especially when accompanied by cholestasis.

pancreatitis and pancreatic tumors

intoxication of ethanol, drugs and sedatives.

Creatine kinase (creatine phosphokinase) catalyzes the reaction of creatine phosphorylation, resulting in the formation of creatine phosphate. Its activity is determined in skeletal muscles, heart, and brain. There are 3 factions.

MM-fraction (muscle)

MV-fraction (heart)

VV-fraction (brain)

Normal blood serum activity is 66.6 mmol (ch. l). Reasons for increased activity:

acute myocardial infarction. Mainly MV-fraction increases.

acute myocarditis, heart trauma and operations. Mainly MV fraction increases.

in some variants of unstable angina pectoris (severe and prolonged angina pectoris attacks, Prinsmetal's angina pectoris). An increase in MV-fraction is observed or is at the upper limit of the norm.

damage to skeletal muscles: polymyositis, dermatomyositis, muscular dystrophy, any trauma and surgery.

Intravenous and intramuscular injections

in rare cases, seizures, physical exertion, pulmonary artery embolism, prolonged hypothermia, congestive heart failure, severe arrhythmias.

The decrease in activity does not matter.

Lactate dehydrogenase is a cell enzyme that participates in the process of glycolysis, and catalyzes the reverse reaction of converting pyruvic acid (pyruvate) to lactic acid (lactate). The final product of pyruvate glycolysis is converted to pyruvate lactate under anaerobic conditions. 5 isomers of LDG can be separated by electrophoresis or photometry. Among them, LDG1 and LDG2 are important.

The LDG1 fraction catalyzes the conversion of lactate to pyruvate more actively. Normally, it works in aerobic conditions and is in the heart muscles.

The LDG5 fraction catalyzes the reaction of lactate formation from pyruvate and is found in the liver and skeletal muscles. It works in anaerobic conditions (strong physical exertion, rapid fatigue), the lactate produced comes to the liver through the blood and is used in glyconeogenesis, it is oxidized to pyruvate in the heart and other organs and participates in the Krebs cycle.

Normal serum LDG activity is 195 ME at 25oC and 320 ME at 30oC. Reasons for increased activity:

heart damage (acute MI, myocarditis), more LDG1, LDG2 increases.

liver damage (viral hepatitis, liver cirrhosis, cancer, obstructive jaundice), more LDG5 increases.

skeletal muscle damage, muscle inflammation and degenerative diseases, more LDG5 increases.

blood diseases with cell breakdown: acute leukemia, hemolytic anemia, V12 deficiency anemia, diseases with platelet breakdown and pathological conditions (massive hematotransfusion, pulmonary artery thrombosis, shock).

more LDG2,3,4 increases.

Glucose-6-phosphate dehydrogenase is one of the main enzymes of the pentose phosphate cycle, a catalyst for the oxidation of glucose-6-phosphate to 6-phosphategluconolactone. Enzyme activity is determined more in erythrocytes. The presence of the pentose phosphate cycle in erythrocytes is primarily a source for NADF.N, and also ensures a normal concentration of glutathione.

In addition, it protects erythrocytes and hemoglobin from disintegration and denaturation as a result of various oxidizing agents. These are: antimalarials, PASK, sulfanilamide, phenacytin, large doses of vitamin C, viral infections, fungi, and legumes. These agents cause glutathione oxidation in erythrocytes. Glucose-6-phosphate dehydrogenase deficiency blocks the breakdown of glucose by pentose phosphate in cells and the release of a large amount of NADF.N and the release of oxidized glutathione into its SH-form. As a result, the concentration of restored glutathione decreases and leads to the accumulation and deformation of denatured hemoglobin in the erythrocyte membrane (Gains bodies), which causes erythrocyte hemolysis.

Alkaline phosphatase (phosphomonoesterase) enzyme hydrolyzes orthophosphoric acid esters in an alkaline environment. The enzyme is present in almost all organs, but its activity is more pronounced in the liver, placenta, intestine, and bone marrow. Reasons for increased enzyme activity:

diseases of the liver: obturative jaundice (more characteristic), cholangitis, cirrhosis, cholestasis of the liver, cancer, metastases in the liver.

bone diseases, diseases with increased activity of osteoblasts, Paget's disease (deforming osteitis), rickets, osteosarcoma, metastases, osteomalacia, myeloma disease, formation of bone mass, hyperparathyroidism.

diseases caused by intestinal damage: ulcerative colitis, ileitis, intestinal bacterial infections.

taking hepatotoxic or cholestasis-enhancing drugs.

pregnancy.

Acid phosphotase - hydrolyzes orthophosphoric acid esters in an acidic medium. Its activity is more pronounced in the prostate gland. Its activity increases in diseases of the prostate gland, and it also increases when the cancer in this area metastasizes to the bones.

α- Amylase - breaks down starch, glycogen and other polysaccharides into maltose and oligosaccharides. 60-70% of the enzyme activity corresponds to solic amylase, 30-40% to pancreatic amylase. Excreted by the kidneys. An increase in activity is observed:

mumps

pancreatitis, pancreatic cancer, diabetic ketoacidosis

kidney failure (due to reduced output through the kidneys)

bladder cancer, ovarian tumors, intestinal obstruction, peritonitis, acute appendicitis, burns, cholecystitis.

Lipase is produced in the pancreas. It breaks down the triacylglycerol formed by the emulsification of fats into mono-diacylglycerol and free fatty acids, which are then absorbed into the blood. Its activity in blood serum does not exceed 0-28 μmol/(min.l). An increase in activity is observed:

acute apncteatitis

gallstone attack, intestinal obstruction, peritonitis, intestinal infarction, intestinal Oki gastric perforation.

Carbohydrates

Provides energy to organs and tissues. A component of the cell envelope. Participates in immunological reactions. Hyaluron and chondroitin sulfate are part of connective tissue. It is divided into 3 main groups:

monosaccharides

Hexoses contain 6 carbon atoms (glucose, galactose, fructose).

Pentoses contain 5 carbon atoms (ribose, deoxyribose).

2. disaccharides

Lactose (glucose + galactose)

Sucrose (glucose + fructose)

Maltose (glucose + glucose)

It contains polysaccharides and monosaccharides (glycogen, starch, cellulose).

400-500 g of carbohydrates enter the body through food per day. Carbohydrates are broken down by amylase in the mouth. In the small intestine, pancreatic amylase breaks down polysaccharides into maltose. Intestinal juice contains a large number of hydrolase enzymes that break down disaccharides into monosaccharides. It is absorbed through the walls of the small intestine and goes to the liver, only monosaccharides are absorbed into the blood, unabsorbed disaccharides are excreted unchanged through the kidneys. Monosaccharides absorbed in the liver and other organs are converted into glucose. Songra participates in enzymatic reactions that generate the necessary energy for protein, fat, and carbohydrate metabolism.

Glucose metabolism begins with its phosphorylation and conversion into glucose-6-phosphate in all organs. This reaction accounts for the consumption of 1 molecule of ATF in the cell cytoplasm. Then it continues in 4 directions:

glycolysis. The end product is pyruvate. As a result of decarboxylation of oxygen under aerobic conditions, acetyl-CoA (the end product of the Krebs cycle in mitochondria) is formed. As a result of the oxidation-phosphorylation reaction, SO2 and water, a large amount of energy are produced. Under anaerobic conditions, lactate dehydrogenase produces lactic acid from pyruvate.

pentose phosphate pathway. Ribose, which is needed for the synthesis of nucleotides and nucleic acids, is produced in the cell cytoplasm under anaerobic conditions.

glycogenesis. the pathway for converting glucose 6 phosphate into glycogen in its reserve form.

The fourth pathway is the reverse pathway of glucose formation from glucose 6-phosphate.

The amount of glucose in the blood is 3.3-5.5 mmol/l. Hyperglycemia is observed 1.5-2 hours after eating. During this period, the liver converts monosaccharides in the blood into glucose, and it into glycogen. Short-term hyperglycemia stimulates the release of insulin from the islets of Langerhans in the pancreas. Insulin performs the following functions to normalize glucose levels:

activates the entry of glucose into the cell.

ensures participation in energy production in the Krebs cycle.

accelerates glycogen synthesis in the liver and muscles.

accelerates the synthesis of fatty acids and amino acids from the intermediate products of glucose breakdown.

inhibits lipolysis.

inhibits glycolysis (formation of glucose from glycogen).

inhibits gluconeogenesis (formation of glucose from amino acids and fats).

In the absence of carbohydrates through food, the amount of glucose is normalized at the expense of glucose released by the liver, the resulting hypoglycemia stimulates the release of glucagon from the pancreatic islets and thereby keeps the glucose level at a normal level.

accelerates the breakdown of glycogen in the liver.

accelerates gluconeogenesis.

inhibits the effect of insulin and activates glycogen synthesis.

inhibits protein synthesis and accelerates proteolysis.

Such compensatory hypoglycemia lasts for a short time (up to 24 hours), if it lasts for a long time, the hypothalamo-pituitary-adrenal gland system is activated, glucocorticoids, adrenaline, somatotropic hormone are produced. They accelerate

the breakdown of glycogen in the liver and stimulate the use of fats as an energy substrate. The amount of glucose in the blood taken after a meal is 3.5-5.7 mmol/l for people under 50 years of age, 4.4-6.2 mmol/l for people over 50 years of age, hyperglycemia when the glucose level exceeds 6.2 mmol/l, 3, A decrease of 3 mmol/l is called hypoglycemia. Causes of hyperglycemia:

diabetes type I or II (lack of insulin production or increased tissue tolerance to this hormone).

pituitary diseases with increased somatotrope and ACTG secretion (pituitary tumors, Itsengo-Cushing's disease, acromegaly).

when diseases of the adrenal glands are accompanied by increased production of catecholamines or glucocorticosteroids (pheochromocytoma).

thyrotoxicosis.

pancreatic diseases (acute and chronic pancreatitis, pancreatic edema).

side effects of some drugs (corticosteroid, thyroxine, ACTG, adrenaline, estrogens, indomethacin, large doses of nicotinic acid, thiazide diuretics, ethocrine acid, furosemide).

physiological hyperglycemia (consumption of products rich in easily digestible carbohydrates, intensive physical exercises, strong emotional strain, stress).

Causes of hypoglycemia:

overdosage of insulin or sugar-lowering drugs in patients with diabetes.

Ulcerogenic adenoma developed from α-cells of pancreatic islets of Langerhans (Zollinger-Ellison syndrome).

arsenic, chloroform poisoning, algae intoxication, in which the process of glycolysis and gluconeogenesis is disturbed due to a decrease in liver function.

diseases of endocrine organs (Addison's disease, hypothyroidism, hypopituitrism).

suspensions of different localization.

carbohydrate absorption disorders (enteritis, pancreatic diarrhea).

alimentary hypoglycemia (prolonged starvation).

Glucose tolerance test. For 3 days, the patient is on a diet with foods containing no more than 150 g of carbohydrates. The examination is performed on an empty stomach. Eating and smoking are prohibited during the examination. 75 g of glucose is dissolved in hot water and given to the patient, then the amount of glucose in the capillary blood is determined after 60, 90, 120 minutes. Normally, the amount of glucose rises to a maximum in 60 minutes and returns to the previous level in 120 minutes. The diagnosis of diabetes is confirmed if it is higher than 7.2-7.8 mmol/l, tested on an empty stomach, and higher than 11 mol/l after a meal.

Reduced glucose tolerance:

decrease in the ability of tissues to dispose of glucose, hidden diabetes, steroid diabetes.
increase in the rate of glucose absorption in the intestines (12 b. intestinal ulcer disease, after gastroectomy, hyperteriosis).
increase in the intensity of glucogenolysis (breakdown of glycogen) and gluconeogenesis. Increased activity of the adrenal gland, hyperthyroidism, pheochromocytoma, during pregnancy.
due to liver damage, a decrease in glycogen synthesis.
Glucose tolerance improvement: reduction of fasting glucose to 3.3 mmol/l.
disorders of absorption and breakdown of monosaccharides in the small intestine (enteritis, Whipple's disease, hypothyroidism, decreased adrenal gland activity).
excessive production of insulin in pancreatic adenoma or cancer of the islets of Langerhans.
As a result of the reaction of glycolyzed hemoglobin with glucose or other monosaccharides, a monosaccharide residue is added to the protein molecule. The amount of glycated hemoglobin depends on the concentration of glucose in the blood. Glycolyzed hemoglobin is checked when choosing a drug in patients with diabetes. Its amount in blood is 4.5-6.1 molar%.

3.2. ANALYTICAL PART

Discussion of the new pedagogical technology used in the lesson
It is conducted to evaluate students' knowledge. The teacher divides the group into two. He marks each group as a team on the board. Teams ask each other questions about hepatitis and liver cirrhosis. The team that answers the question correctly gets 1 point. The team with the most points is considered the winner.
Situational issue.
Issue #1
The patient is 37 years old. The patient received sulfanilamide because of URK (ORZ). 2 hours after taking the first pill, the patient experienced weakness, sweating, nausea, and an increase in body temperature up to 370C. Lens: skin and mucous membranes are yellow, urine is dark in color. Hemogram: N 70 g/l, er.2.1x1012/l, RK 0.9, ret. 40%, leuk. 5.2x109/l, t/ya 4%, s/ya 61%, eoz. 6%, lim. 32%, mon. 7%, ECHT 20 mm/h. Blood biochemical analysis: the indirect fraction of bilirubin increased dramatically. Urine analysis: cloudy, bluish color, relative density 1026, protein 3.3%. Microscopy: solution. 1 new, lake.1. Coombs reaction is positive. Hemosiderin and hemoglobin reactions are positive.
Question: 1. Are there any changes in the hemogram?
2. Your initial diagnosis.
3. Your inspection plan.

Answer: 1. N 70 g/l, er.2.1x1012/l, ret. 40%, ECHT 20 mm/h.
2. Autoimmune hemolytic anemia.
3. analysis of urine, biochemical analysis of urine, general analysis of urine, general analysis of feces
Issue #2
Patient M, 46 years old. Complaints: general weakness, rapid heartbeat, aching bones. Anamnesis: sick for a year. The therapist treated the "kidney" disease accordingly. AKB is normal. Hemogram: N 80 g/l, er.2.2x1012/l, RK 0.8, trom.150x109/l, leuk.3.2x109/l, t/ya 3%, s/ya 54%, eoz.2 %, base.1%, lim.38%, mon.2%, ECHT 76 mm/h. Urinalysis: oxil 3.3%, epit. 1-2/1, lake. 3-4/1, melt. 0-1/1, TB. 0-1/1.
Biochemical analysis of the deposit: urea 22 mmol/l, um.oxyl 90 g/l.
Question: 1. What are the changes in the hemogram
2. Your initial diagnosis.
3. Your inspection plan.
Answer: 1. Anemia in hemogram, trom. 150x109/l, ECHT 76 mm/h, Urinalysis: oxil 3.3%, urea 22 mmol/l in biochemical analysis, um. oxil 90 g/l.
2. Myeloma disease.
3. myelogram, X-ray of flat bones.
Issue #3
A 28-year-old patient fell ill. He did not take alcohol or drugs. The liver is painful on palpation in the rib cage. Bilirubin 184 μmol/l, bound 100 μmol/l, unbound 84 μmol/l, alkaline phosphatase 210 ed., ALT 4.5. Prothrombin time 65%.
Question: 1. Which type of jaundice is the analysis?
2. presumptive diagnosis?
Answer: 1. parenchymatous jaundice.
2. taking into account cytolysis, cholestasis syndromes and cellular failure (hypoprothrombinemia) diagnosis: viral hepatitis, acute course.
Issue #4
A 35-year-old woman complains of skin itching. He has been sick for 3 years. On examination, the liver is hard, 10 cm below the rib cage. Bilirubin 95 μmol/l, bound 80 μmol/l, alkaline phosphatase 400 ed., ALT 0.86 mmol. ch/l.
Question: 1. What kind of disease is the analysis?
2. Disease-specific changes in analysis?
Answer: 1. biliary cirrhosis.
2. chronic course of the disease, cirrhosis of the liver, cholestasis syndrome (itching of the skin, increased bound bilirubin and alkaline phosphatase).
Situational problem 5:

A 45-year-old patient came to the clinic with a complaint of headache, rapid fatigue, weakness, decreased work performance. Objective: the skin is earthy, dry. Sur has been suffering from pyelonephritis for 15 years. UKA: HB 70g/l, erythritol-3.6x1012/l, r.k 0.85, leukocyte 9x109/l, ECHT 16 mm/h In biochemical analysis: urea 20 mmol/l. Urinalysis: oxygen 3.5 g/l, density 1030.
Questions: 1. Approximate diagnosis?
2. What are the changes in urine?
3. Changes in BQA?
Answer: 1. kidney failure
2. oxygen 3.5 g/l, density 1030
3. Mechevina 20 mmol/l
Situational issue 6
Patient R, 31 years old, came complaining of general weakness, headache, nausea, epigastric pain, heaviness, 2 vomiting, fever up to 380C, dark colored urine. He considers himself sick for 6 months. Lens: the skin and mucous membranes are yellow, the liver is enlarged +4+5 cm, the spleen is not palpable.
Question: 1. Presumptive diagnosis?
2. What is your inspection plan?
Answer: 1. Chronic hepatitis.
2. Liver UTT, biochemistry, HBS-Ag, HCV-Ag, PSR diagnostics.
Situational issue 7
Patient M, 36 years old. Complaints: pain under the ribs, sudden yellowness, general weakness, rapid heartbeat, pain in the bones, nausea. Anamnesis: he was treated for a year with the diagnosis of chronic stone cholecystitis. AKB is normal. Hemogram: N 80 g/l, er.2.2x1012/l, RK 0.8, trom.150x109/l, leuk.3.2x109/l, t/ya 3%, s/ya 54%, eoz.2 %, base.1%, lim.38%, mon.2%, ECHT 18 mm/h. Urinalysis: oxil 0.033%, epit. 1-2/1, lake. 3-4/1, melt. 0-1/1, TB. 0-1/1, horse acids +, bilirubin +, urobilin +.
Questions: 1. Your initial diagnosis.
2. Your inspection plan.
Answer: 1. Horse stone disease.
2. biochemistry, liver and gallbladder UTT, surgeon.
Situational problem 8
Patient R., 18 years old, was found to have an enlarged liver during the shooting from a rifle before being called up for military service. The patient's complaint is positive, but hepatitis C was detected in the IFA examination. The patient recalled that at the age of 16 he took drugs in a syringe with 7 other people.
Questions: 1. Presumptive diagnosis
2. What additional inspection methods should be used?

Answer: 1. Chronic hepatitis C.
2. UQA, biochemistry, diagnosis of hepatitis C PSR
Situational problem 9
A 58-year-old patient has been drinking alcohol for many years. In the last 3 years, against the background of general weakness and loss of appetite, there was a complaint of decreased urination and enlarged colon. He felt heavy under the stove. Several times the mine went out of his nose.
Lens: the general condition is severe, ascites in the cornea, venous collaterals are enlarged, vascular stars are visible. Taluk is in the area of the navel. UKT: anemia, leukopenia, thrombocytopenia. In biochemical analysis, % hyperbilirubinemia, AST, ALT increased.
Questions: 1. Presumptive diagnosis
2. What additional inspection methods should be used?
3. Treatment plan.
Answers: 1. Portal liver cirrhosis Ascites. Splenomegaly
2. Um. oxyl and its fraction, thymol test, active phosphatase, Coagulogram, cholesterol, urea. creatinine. UZI is a corn bush. Liver scan. Liver biopsy. EFGDS.
3. Diet: table No. 5. Veroshpiron, furosemide, hepatoprotector, vitamins, lactulose, N-2 histamine receptor blockers, ursosan
Situational problem 10
The patient is 32 years old. Suffered from VG 2 years ago. He was treated in the infectious disease hospital for 63 days. Sung was on a diet for 1 year and received outpatient treatment. Elevations of bilirubin and enzymes have been observed several times.
Lens: the general condition is very severe, the sclera is yellow, there are shrinkages. Body temperature is expected to 38. Liver +5-6 cm. There are nodules, hardened, ogre. Spleen enlarged by 1-2 cm, hard. UKA: HB 70g/l, erythritol-3.6x1012/l, r.k 0.85, leukocyte 3.4x109/l, ECHT 16 mm/h. In biochemical analysis: ALT 2.3 mmol/l, AST 1.2 mmol/l
Questions: 1. What additional examination methods should be used to confirm the diagnosis?
2. Presumptive diagnosis.
3. Your recommendation for treatment.
Answers: 1. Um. oxyl and its fraction, thymol test, active phosphatase, Coagulogram, cholesterol, urea. creatinine. UZI is a corn bush. Liver scan. Liver biopsy. EFGDS.
2. Cirrhosis of the liver, active period after the virus, subcompensated
3. Diet: table No. 5. Veroshpiron, furosemide, hepatoprotector, interferon, vitamin, lactulose, N-2 receptor blocker histamine, ursosan

TESTS.

1. How are protein fractions determined?
Photometric
Immunological
Electrophoresis
Urease method
Reductometric
2. The amount of total protein in the serum
65-85 g/l
35-50 g/l
45-85 g/l
50-65 g/l
80-95 g/l
3. The amount of total albumin in the serum
65-85 g/l
35-50 g/l
45-85 g/l
50-65 g/l
80-95 g/l
4. Hypoproteinemia is
total protein less than 65 g/l
total protein less than 60 g/l
total protein less than 50 g/l
total protein less than 85 g/l
total protein less than 75 g/l
5. When total protein increases
Myeloma disease
Light disease
Thyrotoxicosis
Nephrotic syndrome
Liver cirrhosis
6. When the total protein decreases
Myeloma disease
Waldenström macroglobulinemia
Chronic diseases with immune system activation
Liver cirrhosis
Acute inflammatory diseases

7. Amount of urea in blood serum
2.7-8.3 mmol/l
3.4-9.5 mmol/l
2.5-7.4 mmol/l
1.8-6.6 mmol/l
3.0-8.4 mmol/l
8. The reason for the increase in the amount of urea
OBE
SBE
Increased protein catabolism
Dehydration of the body
Everything is correct
9. Where is bilirubin synthesized?
When hemoglobin is broken down in the spleen
When transferrin is broken down in the spleen
Stercobilin is broken down in the spleen
When transferrin is broken down in the liver
Stercobilin is broken down in the liver
10. Amount of bound bilirubin in blood serum
8.5-20.5 μmol/l
0.9-4.3 μmol/l
6.4-17.1 μmol/l
0.08-4.23 μmol/day
50-300 ml/day
11. Amount of unbound bilirubin in blood serum
8.5-20.5 μmol/l
0.9-4.3 μmol/l
6.4-17.1 μmol/l
0.08-4.23 μmol/day
50-300 ml/day
12. Normal concentration of total bilirubin in the blood
8.5-20.5 μmol/l
0.9-4.3 μmol/l
6.4-17.1 μmol/l
0.08-4.23 μmol/day
300-500 mg/day
13. What amount of bilirubin causes jaundice syndrome
30-34 μmol/l
20-27 μmol/l

17.1-20 μmol/l
20-25 μmol/l
17.1-27 μmol/l
14. Unbound bilirubin increases in which pathology
MI
Hemolytic anemia
Atherosclerosis
Diabetes
Pregnancy
15. Unbound bilirubin increases in which pathology
MI
Atherosclerosis
Sickle cell anemia
Diabetes
Pregnancy
16. Unbound bilirubin increases in which pathology
MI
Atherosclerosis
V12 deficiency anemia
Diabetes
Pregnancy
17. Which pathology increases bound bilirubin
MI
Atherosclerosis
Diabetes
Viral hepatitis
Pregnancy
18. Which pathology increases bound bilirubin
MI
Atherosclerosis
Diabetes
Infectious mononucleosis
Mechanical jaundice
19. Laboratory changes in paraproteinemia
hyperproteinemia
hypernatremia
thrombocytosis
(+) proba Gregersena

20. Changes in biochemical analysis in liver and horse liver suspensions of poor quality
hyperbilirubinemia
hypobilirubinemia
abilirubinemia
everything is correct
everything is wrong

CHAPTER 4. LABORATORY DIAGNOSIS OF HEART AND CONNECTIVE TISSUE DISEASES.

1. Objectives of the training:
rheumoprobe analysis;
laboratory diagnosis of heart diseases.
Lipid metabolism.
Clinical analysis of biochemical blood analysis.
Blood for biochemical analysis is usually taken on an empty stomach, from the medial or lateral subcutaneous vein of the arm.
Alanine aminotransferase. A catalyst for the reamination reaction between alanine and α-ketoglutaric acid. losing the alanine amino group, it turns into pyruvic (pyruvic) acid. α-Ketoglutaric acid binds amino group and turns into glutamic acid. Glutamine and aspartic acid participate in the biosynthesis of urea and neutralize ammonia formed in the body. Aminotransferases are found in all organs, but are more active in the liver, skeletal muscles, heart, and kidney. The activity of aminotransferases in erythrocytes is 6 times higher than in blood serum. When damage is observed in these organs, the amount of aminotransferases in the blood increases. Reasons:
necrosis or damage of liver cells (acute viral hepatitis, chronic hepatitis, liver cirrhosis, liver tumors, alcohol intoxication, obstructive jaundice, taking hepatotoxic drugs).
acute myocardial infarction, acute myocarditis
skeletal muscle necrosis or trauma.
erythrocyte hemolysis.
In clinical practice, the ratio of AST/ALT activity in blood serum (de Ritis coefficient) is of great importance.
AST activity is higher than ALT in acute myocardial infarction. Ritis coefficient is higher than 1.3.
ALT activity is high in acute viral and chronic hepatitis, especially in the early period. Ritis coefficient is less than 1.0.

γ - glutamyltranspeptidase is a transferase that participates in nitrogen metabolism. A reaction catalyst that transfers a glutamine group to an acceptor peptide or L-amino acid. Enzyme activity does not exceed 66-106 ME in the norm determined in liver, kidney, pancreas. Increased activity is observed in the following pathological conditions:

in obturation of intrahepatic and extrahepatic horse ducts

liver diseases (hepatitis, liver cirrhosis, metastases of liver tumors), especially when accompanied by cholestasis.

pancreatitis and pancreatic tumors

intoxication of ethanol, drugs and sedatives.

Creatine kinase (creatine phosphokinase) catalyzes the reaction of creatine phosphorylation, resulting in the formation of creatine phosphate. Its activity is determined in skeletal muscles, heart, and brain. There are 3 factions.

MM-fraction (muscle)

MV-fraction (heart)

VV-fraction (brain)

Normal blood serum activity is 66.6 mmol (ch. l). Reasons for increased activity:

acute myocardial infarction. Mainly MV-fraction increases.

acute myocarditis, heart trauma and operations. Mainly MV fraction increases.

in some variants of unstable angina pectoris (severe and prolonged angina pectoris attacks, Prinsmetal's angina pectoris). An increase in MV-fraction is observed or is at the upper limit of the norm.

damage to skeletal muscles: polymyositis, dermatomyositis, muscular dystrophy, any trauma and surgery.

Intravenous and intramuscular injections

in rare cases, seizures, physical exertion, pulmonary artery embolism, prolonged hypothermia, congestive heart failure, severe arrhythmias.

The decrease in activity does not matter.

Lactate dehydrogenase is a cell enzyme that participates in the process of glycolysis, and catalyzes the reverse reaction of converting pyruvic acid (pyruvate) to lactic acid (lactate). The final product of pyruvate glycolysis is converted to pyruvate lactate under anaerobic conditions. 5 isomers of LDG can be separated by electrophoresis or photometry. Among them, LDG1 and LDG2 are important.

The LDG1 fraction catalyzes the conversion of lactate to pyruvate more actively. Normally, it works in aerobic conditions and is in the heart muscles.

The LDG5 fraction catalyzes the reaction of lactate formation from pyruvate and is found in the liver and skeletal muscles. It works in anaerobic conditions (strong physical exertion, rapid fatigue), the lactate produced comes to the liver through

the blood and is used in glyconeogenesis, it is oxidized to pyruvate in the heart and other organs and participates in the Krebs cycle.
Normal serum LDG activity is 195 ME at 25oC and 320 ME at 30oC. Reasons for increased activity:
heart damage (acute MI, myocarditis), more LDG1, LDG2 increases.
liver damage (viral hepatitis, liver cirrhosis, cancer, obstructive jaundice), more LDG5 increases.
skeletal muscle damage, muscle inflammation and degenerative diseases, more LDG5 increases.
lytic blood diseases with cell breakdown: acute leukemia, hemolytic anemia, V12 deficiency anemia, diseases with platelet breakdown and pathological conditions (massive hematotransfusion, pulmonary artery thrombosis, shock) increase LDG2,3,4 more.
Lipids
40% of energy is generated due to the breakdown of lipids.
Fatty acids: if the secondary bond in the structure is unsaturated, fatty acids are called saturated. Unsaturated fatty acids are not synthesized in the body and enter the body through food with vegetable oils. These contain 2, 3 and 4 secondary bonds in linoleic, linolenic, and arachidonic acids. It limits the deposition of cholesterol on the artery wall.
Glycerin-containing lipids contain three hydroxyl groups, fatty acids. When one hydroxyl group is replaced by a phosphate, a phosphoglyceride is formed.
phospholipids form the cell membrane, activate enzymes and take part in blood clotting
steroids cholesteorin, horse acids, progesterone, corticosterone, cortisol, testosterone, estradiol.
Lipoproteins are the transport form of lipids, the carrier of fats in the blood. They consist of proteins and lipids. There are 4 classes.
chylomicrons (XM)
very low density lipoprotein (VLP)
low density lipoprotein (ZPLP)
high density lipoprotein (HDL)

Lipid metabolism - 95% of fats in food consist of complex esters of glycerol and fatty acids. 12b In the intestine, fats are emulsified under the influence of salts of horse acids. An emulsion consisting of thin dispersed droplets of lipids in water is formed. Hydrolyzed by lipase, monoglycerides and fatty acids are formed. Under the influence of salts of horse acids, micelles are formed, and it is absorbed from

the cells of the wall of the small intestine without any obstacles, and triglycerides are formed from monoglycerides and fatty acids. Then, in the form of chylomicrons, it is absorbed into the lymph and enters the blood along the lymphatic route.

Total cholesterol is present in plasma in the form of complex esters with horse acids and in lipoproteins. Total serum cholesterol is 3.9-6.5 mmol/l (150-250 mg/dl). free cholesterol is 25-35%, ester is 65-75%. Causes of hypercholesterolemia:

atherosclerosis of different localization

hyperlipoproteinemia

liver diseases, especially cholestasis

diabetes

nephrotic syndrome, glomerulonephritis, chronic kidney failure.

hypothyroidism

Causes of hypocholesterolemia:

hyperthyroidism

severe liver damage (cirrhosis, cancer, active hepatitis), liver failure.

malabsorption syndrome

prolonged starvation or poor diet

Cholesterol (α-LP) in ZYULP. In adults, ZYULP maintains 0.9-1.9 mmol/l cholesterol. An increase in the amount of cholesterol in ZYULP is observed in biliary cirrhosis, chronic hepatitis, and some intoxications. When the amount of cholesterol in ZYULP drops below 0.9 mmol/l, it has clinical significance and, first of all, the probability of developing atherosclerosis, LUIK, cerebral ischemic diseases increases. In this case, it is necessary to check the ratio of cholesterol in ZYULP to total cholesterol. If the ratio is less than 15%, the probability of occurrence of the above diseases increases.

Triglycerides triglycerides in blood plasma or serum are checked after 12 hours of fasting. The norm is 0.45-1.81mmol/l for women/ 0.40-1.53mmol/l for men.

Causes of hypertriglyceridemia:

hyperlipoproteinemia in types I, IIB, III, IV, V.

viral hepatitis, alcoholic and biliary liver cirrhosis.

equine airway obstruction.

acute myocardial infarction and chronic acute myocardial infarction.

thrombosis of cerebral vessels.

diabetes, gout, hypothyroidism.

nephrotic syndrome.

Causes of hypotriglyceridemia:

hypolipoproteinemia, including abetalipoproteinemia.

hyperthyroidism, hyperparathyroidism.
malabsorption syndrome.
liver diseases with severe functional impairment.
Phospholipids: normal amount in plasma is 1.52-3.62g/l. Causes of hyperphospholipidemia:
hyperlipoproteinemia type IIa and IIb.
alcoholic and biliary cirrhosis.
cholestasis.
acute myocardial infarction and chronic acute myocardial infarction.
pancreatitis.
nephrotic syndrome.
Causes of hypophospholipidemia:
familial abetalipoproteinemia.
hyperthyroidism.
severe liver diseases.
V12-folate acid deficiency anemia.
Lipoproteins In clinical practice, the increase in the amount of lipoproteins is more important, especially ZJPLP and ZPLP, which have atherogenic properties. There are 5 types of hyperlipoproteinemia (GLP).
An increase in the amount of chylomicrons and triglycerides in the type. The amount of ZJPLP may be normal or relatively increased. Abdominal pain, xanthomatous rashes, lipoid thickening of the eyelid, hepatosplenomegaly are observed in the clinic. This type of GLP is found in: 1) primary hyperchylomicronemia, lack of chylomicron-degrading lipoprotein lipase; 2) in diabetes; 3) in pancreatitis; 4) occurs in diseases caused by excessive production of corticosteroids.
The type contains a lot of ZPLP and total cholesterol. There are 2 different types of GLP.
Type a ZPLP and cholesterol levels are high, while ZJPLP and triglyceride levels are normal.
Type b - in the case of high levels of ZPLP and cholesterol, a relative increase in the amount of ZPLP and triglycerides. This manifestation of clinical GLP is observed in the early development of atherosclerosis of various localizations, YUIK, acute myocardial infarction, stroke. The risk of sudden coronary artery disease is high. Occurrence: 1) primary family hypercholesterolemia; 2) nephrotic syndrome; 3) diseases accompanied by hypercorticism; 4) diabetes; 5) hypothyroidism.
Type contains triglyceride, cholesterol, anomalous ZJPLP and ZPLP. Early onset of clinical atherosclerosis is accompanied by xanthomatosis. The main causes: 1)

primary betalipoproteinemia; 2) diabetes; 3) hypothyroidism; 4) dysgammaglobulinemia.

When the concentration of type ZLPP is normal, the amount of ZJPLP and triglycerides is high. Cholesterol levels are normal or slightly elevated. Atherosclerosis and YUIK are observed in this type of GLP. Occurrence: 1) primary family hypertriglyceridemia; 2) obesity; 3) diabetes; 4) hypothyroidism; 5) nephrotic syndrome and uremia; 6) chronic diseases with hypercorticism; 7) alcoholism; 8) taking estrogens;

Type chylomicron and ZJPLPs, triglyceride, and cholesterol are high. It is accompanied by clinical splenomegaly, abdominal pain attacks, development of pancreatitis, xanthomatosis. Reasons: 1) primary family hyperlipidemia; 2) diabetes; 3) hypothyroidism; 4) nephrotic syndrome and uremia; 5) chronic diseases with hypercorticism; 6) alcoholism; 7) taking estrogens;

4.2. ANALYTICAL PART.

The new pedagogical technology used in the lesson is Lottery"

Questions of lectures and practical exercises on clinical analysis of urinalysis, laboratory diagnosis of kidney diseases, clinical analysis of stool analysis are written on one large sheet of paper. All questions are numbered. These numbers are written on small pieces of paper and placed in the box. A question paper will also be posted. All students are invited to choose a piece of paper, then answer the question written on that number. A student who chooses a question without a number is considered lucky and does not answer the question.

Questions:

type of lipoprotein?

atherogenic factors?

What is an LE cell?

What is included in the rheumoprobe?

What is C-reactive protein?

What are acute phase proteins?

What is the scissor symptom in IM?

where is cholesterol produced?

Situational issue.

Issue #1

A 48-year-old patient, who has been suffering from YUIK for 6 years, has been bothered by pain in the heart area for two days. Despite taking nitroglycerin, the patient's severe pain in his heart did not stop, the lack of air increased and he broke into a cold sweat, the arterial blood pressure decreased to 110/60 mm. in a short

period of time, his heart beat fast and then slowed down. When narcotic drugs were given, the pain did not disappear completely.
Questions: 1. Make a diagnosis.
2. What inspection methods do you recommend?
3. What are UASH tactics?
Answer: 1. YUIK. Acute myocardial infarction
2. ECG, ultrasound, coagulogram, lipid spectrum
3. Hospitalization in cardioreanimatology department
Issue #2.
The patient complains of 44 hours, tight sores in the chest area, cold sweat, general weakness. Anamnesis: YUIK has been diagnosed for 5 years. Since this morning, ogriks in the heart area are bothering me, nitroglycerin has been administered several times, they did not remove the ogrik.
Lens: General condition is very heavy, skin cover is pale. Upka vesicular breathing, heart sounds bug, pulse rhythmic 80 beats 1 min. AKB 180/90 mm.cm.us. Sinus rhythm on EKG, YUUS 80 beats 1 min. III, the S-T segment is above the isoline in AVF junctions.
Questions: 1. Make a diagnosis.
2. What inspection methods do you recommend?
3. What are UASH tactics?
Answer: 1. YUIK. Acute myocardial infarction in the anterior wall of the left ventricle
2. ECG, ultrasound, coagulogram, lipid spectrum
3. Hospitalization in the cardioreanimatology department
Issue #3
After 4 weeks, a patient undergoing treatment for a myocardial infarction had a fever, a chest pain, an increase in breathing, and a dry throat.
On auscultation: moist wheezing in the lungs, breathlessness on both sides in the lower parts, muffled heart sounds, AqB 110/70 mm wire. above The pulse is 100 beats per minute. ExoKS reveals fluid in the pericardium and pleural head.
Questions: 1. Make a diagnosis.
2. What inspection methods do you recommend?
3. What complications of myocardial infarction can be monitored?
Answer: 1. YUIK. Acute myocardial infarction
2. ECG, ultrasound, coagulogram, lipid spectrum
3. Dressler's syndrome
Issue #4
A 55-year-old patient complains of morning numbness lasting until 12 o'clock, pain and swelling in symmetrically located small joints. Hb 100 g/l, L 4, 0 10 9 l,

SOE 55 mm/s ASLO 200, sero mucoid 0.18 ed, S-reactive protein +, rheumo factor was determined.
Questions: 1. Make a diagnosis.
2. The reason for the increase in ECHT
3. normal seromucoid index
Answer: 1. rheumatoid arthritis
2. increase in acute phase proteins
3. 0.13-0.4 units
Issue #5
A 1st-year student noticed that a red rash appeared on her face during autumn agricultural work. Soon the body temperature began to rise to 38°C, swelling and pain in the knee joints, pain in the heart area, tachycardia, swelling of the face appeared in the morning.
Questions: 1. Your diagnosis
2. Test methods that confirm the diagnosis.
3. Comparative diagnosis.
Answers: 1. Systemic scarlet fever, acute course
2. General mine analysis, general urine analysis, acute phase tests, LE-cell
3. Acute rheumatic fever, other connective tissue diseases, glomerulonephritis
Issue #6
A 33-year-old patient named T. complains of pain, swelling in the small joints of the knees and ankles, limited mobility, numbness lasting 2 hours in the morning, nodules measuring 1 cm in the elbows, and general weakness. Lens: Overall condition is fair. Skin is visible, the skin is clean. The tongue is moist, drooling. Vesicular breath in the lungs. Heart tones are increased, the pulse is 75 times per 1 min. AKB s.u.b. 115/75 mm. The corn is soft, without ogre. Liver and spleen are not enlarged. Constipation and urination are normal.
Contractures are detected in wrist, elbow and knee joints. Proliferative changes, deformation and atrophy of interosseous muscles are detected in the proximal interphalangeal joints. His knees are swollen, sore, and his mobility is limited.
Questions: 1. Initial diagnosis.
2. Inspection plan.
3. Comparative diagnosis.
Answer: 1. Rheumatoid arthritis, polyarthritis, slowly developing course, activity-II, BFE-II.
2. General analysis of the deposit, acute phase tests, X-ray of ligaments, analysis of synovial fluid
3. Reactive arthritis, osteoarthritis, gout
Issue #7

A 60-year-old female patient has pain and weakness in the muscles of the legs and feet. complained to arthrology. Kurik's muscles are large in size. Erythematous changes in the face and neck, paraorbital swelling were detected. The patient can not wait for his ashes and legs.

Questions: 1. Initial diagnosis.

2. The test that confirms the diagnosis.

3. Treatment.

Answers: 1. It is necessary to rule out that primary dermatomyositis is of an inflammatory nature

2. Muscle biopsy

3. Prednisolone 60-80 mg per day is not less

Issue #8

A young woman is bothered by pain in the joints, decreased diuresis, body swelling, shortness of breath, dry cough, and a rise in body temperature. UPT: oxygen - 6.6 g/l, solution - 3-5, granular cylinder

Questions: Initial diagnosis.

2. Additional checks.

3. Treatment.

Answers: 1. SCT, subacute course, active period. Pneumonitis, polyarthritis, lymphadenopathy with lupus-nephritis nephrotic syndrome

2. UKT, acute phase test, coagulogram., um oxyl and fraction, coagulogram, DNA antibody, ANF, Kidney UTTsi. x-ray chest, kidney biopsy.

3. Prednisolone, cyclophosphan, heparin, curantyl

Issue #9

A 25-year-old patient had an erythematous rash on his face after getting blisters on the beach. Ash felt the ogre in his joints. After 1 week, heartburn and shortness of breath appeared.

Ob-v: The general condition is serious. Body temperature 38 C, "butterfly"-shaped erythema on the face. The pulse is 100. Cystic murmur in the heart.

OAK: anemia, leukopemia, SOE 40mm/s. Anticochlear AT titer is high.

Questions: 1. Additional inspection methods.

2. Initial diagnosis.

3. What diseases should be distinguished.

Answers: 1. Um oxil and fraction, coagulogram, acute phase test, antinuclear antibody, antinuclear factor, SKV cells in the bone, EKG, X-ray chest, USZ kidney, skin biopsy.

2. SCT, acute course, activity 2 (dermatitis, carditis, polyarthritis)

3. Rheumatism, systemic scleroderma, acute allergic dermatitis

Issue #10

The patient, 62 years old, came to the hospital complaining of throbbing pains in the sternum, shortness of breath, and severe general weakness. He has been suffering from YUIK for many years. Now the pain started 2 hours ago, 4 tabs of nitroglycerin did not work. Lens: oozing skin, cold sweat, cyanotic lips, pulse 92 beats 1 min., AKB 80/50mm.rt.st.
Questions: I. Your approximate diagnosis..
2. Inspection plan.
3. Observed complications.
Answers: 1. YUIK: acute myocardial infarction
2. ECG, MV-fraction CPK, LDG, general mine analysis, biochemical mine analysis: ALT, AST; coagulogram, coagulation time.
3. Cardiogenic shock, thromboembolic complications, arrhythmias, cardiac tamponade, acute heart failure, acute cardiac aneurysm.
TESTS.

1. The cause of increased ECHT in a patient with connective tissue diseases:
decrease in globulins
increased albumin
albumin depletion
decrease in the number of erythrocytes
increased acute phase proteins*
2. Risk of atherogenicity
LPVP is high
Low LPVP *
LPNP is high
Low LPNP
Elevated LPONP*
3. What factors increase ECHT?
increased bilirubin
an increase in the amount of erythrocytes
increased acute phase proteins*
decreased albumin *
decrease in the number of erythrocytes*
4. Factors ensuring high LPVP
in men
in women*
high estrogen levels*
smoking
increased physical activity*

5. An increase in the amount of aminotransferases is observed.
IM*
colitis
skeletal muscle necrosis*
liver damage*
pneumonia
6. An increase in the amount of lactate dehydrogenase is observed
IM*
colitis
skeletal muscle necrosis*
liver damage*
pneumonia
7. The main groups of lipids
monosaccharides
fatty acids*
phospholipids*
steroids*
Disaccharides
8. An increase in the amount of aminotransferases is observed, not relevant
IM
colitis*
skeletal muscle necrosis
liver damage
pneumonia*
9. belongs to acute phase proteins, does not belong
transferrin*
C reactive protein
seromucoid
sialic acid
ferritin*
10. Diagnostic criteria of SKV, not relevant
thrombocytosis*
proteinuria
Finding LE cells
leukopenia
basophilosis*
11. Acute myocardial laboratory diagnosis, not relevant
leukocytosis
an increase in the MV fraction of creatine phosphokinase

SOE decline*
An increase in AsT
leukopenia*
12. Causes of hypercholesterolemia
atherosclerosis*
hyperthyroidism
hypothyroidism*
liver cirrhosis
diabetes*
13. Causes of hypocholesterolemia
atherosclerosis
hyperthyroidism*
hypothyroidism
liver cirrhosis*
prolonged starvation*
14. Increase in triglycerides
nephrotic syndrome*
hyperthyroidism
hypothyroidism*
diabetes*
malabsorption syndrome
15. Reduction of triglycerides
nephrotic syndrome
hyperthyroidism*
hypothyroidism
hypolipoproteinemia*
malabsorption syndrome*
16. It is included in acute phase proteins
transferrin
C reactive protein*
seromucoid*
sialic acid*
ferritin
17. Diagnostic criteria of SKV
thrombocytosis
proteinuria*
Find LE cells*
leukopenia*
basophilosis

18. Acute myocardial laboratory diagnosis
leukocytosis *
increased creatine phosphokinase MV fraction*
SOE decline
AsT increase*
leukopenia
19. The main types of lipids in plasma
cholesterol*
fibrinogen
phospholipid*
triglycerides*
methane
20. Factors ensuring low LPVP
in men*
in women
high estrogen levels
smoking*
obesity*

CHAPTER 5. BLOOD COAGULATION SYSTEM.

1. Objectives of the training:
Teaching coagulogram analysis;
Teaching laboratory diagnostics of hemorrhagic diatheses.
Teaching DVS syndrome laboratory diagnosis.
The liquid state of the blood is a necessary condition for the performance of its tasks, and this condition is provided by the blood coagulation system, the anticoagulation system, and the fibrinolysis system in the body. Plasma, tissue factors and shaped elements of blood take part in the process of blood coagulation.
Cell factors of the blood coagulation system
The participation of platelets in the process of hemostasis is important, these blood platelets affect various aspects of the hemostatic process, their decrease in number or changes in quality can be the main cause of bleeding.
Thrombocytes participate in the following processes:
It ensures the integrity of microvessels, their normal resistance and resilience;
Participates in the formation of the primary platelet plug
brings plasma clotting factors to the site of bleeding
provides blood clot retraction
The clumping together of platelets is called aggregation and is controlled by ADF in the platelet hyalo-mer. Dispersion of aggregates is due to the breakdown of ADF to AMF. In plasma, AMF is degraded to inosine, hypoxanthines, and these

substances, like AMF, resist platelet aggregation. Depletion of fibrinogen leads to severe aggregation disorders. During the first 2 minutes, the aggregation process is recalculated. Then, under the influence of thrombin, thrombocyte mucous metamorphosis begins, degranulation occurs in aggregated platelets, thrombocytic factors are released, and irreversible aggregation occurs.

The sticking of platelets to the surface of the blood, the edges of the wound and the inner side of the vessel is called adhesion property. The process of adhesion leads to the formation of a primary thrombocytic thrombus. The process of adhesion is disturbed in the absence of collagen fibers in the vessel wall or in their pathological forms. Heparin, sour mucopolysaccharides and acetylsalicylic acid reduce platelet adhesion.

Under the influence of thrombin and immune proteins, platelet adhesion agglutination occurs, in which platelets disintegrate and platelet clotting factors are released.

Exogenous and endogenous clotting factors of platelets differ:

Exogenous factors:

Factor 1 adsorbed proaxelin accelerates thrombin generation.

Factor 5 - fibrinogen-like factor causes platelet adhesion to the surface.

7-factor antifibrinolysin inhibits fibrinolysis.

Factor 8 serotonin has a vasoconstricting effect in the initial phase of hemostasis and ensures normal permeability of the vessel wall.

9 - factor - fibrin stabilizer - strengthens fibrin.

Factor 10 activates profibrinolysin

Factor 11 inhibits thromboplastin.

Factor 12 inhibits the effect of heparin on plasma.

Endogenous thrombocytic factors have a fibrinoplasty effect and accelerate the reaction of fibrinogen formation:

2 - factor - forms a net-like fibrous substance.

3 - factor - thromboplastic factor

4 factor anti-heparin factor

Factor 6 thrombosteinin ensures retraction of the blood clot.

Thus, the vascular thrombocyte component of hemostasis is important in hemostasis in the injury of small vessels. in bleeding from large vessels with high blood pressure, blood stops due to the formation of a fibrin blood clot.

retraction of a blood clot is a complex biological process, consisting of thickening of fibrin threads and squeezing out serum. When thrombin is activated, thrombosteinin is released, as a result of which the contractile filaments are drawn from one platelet to another, causing the clot to contract. the necessary energy for

contraction is synthesized in platelet gelomer and provided by ATF, which is present in high concentration there.

Retraction begins 15-30 minutes after the formation of a clot and lasts from 30 minutes to 3 hours. Clot retraction is directly proportional to the number of platelets, and inversely proportional to the concentration of fibrinogen and the number of erythrocytes.

Methods of checking vascular thrombocytic hemostasis.

determination of bleeding time. According to Duke, the bleeding time is determined after the integrity of superficial microvessels is broken. Usually 25 minutes. It is prolonged in thrombocytopenia, Willebrand's disease, thrombocytopathies. In addition, in severe forms of thrombohemorrhagic syndrome, severe heparinemia can be prolonged.

capillary resistance. Rumpel-Leede-Kongalovsky cuff test. The number of petechiae growing on the skin of the wrist is counted when the venous pressure is increased. Normally, the number of petechiae does not exceed 10, and the diameter does not exceed 1 mm. In clearly expressed thrombocytopenia, thrombocytopathy and angiopathy, the number of petechiae is 20 or more, and the diameter is more than 1 mm.

platelet count. Two methods are used in practice.

Direct blood count (using a camera or counter).

counting per 1000 erythrocytes in a blood smear and counting per erythrocytes in 1μl or 1l of blood. Normally 180 320 103μl or 180 320 109 l.

A decrease in the number of platelets is especially evident in the period of thrombocytopenia, thrombocytopenic purpura (less than 50 109/l). Thrombocytopenia is observed in acute leukemia, terminal stages of chronic leukemia, aplastic anemia. In collagenosis and cirrhosis of the liver, a slightly expressed thrombocytopenia is observed (above 100 109/l). Thrombocytosis is characteristic of myeloproliferative diseases (chronic myeloid leukemia and erythremia in the initial and exacerbation period) and malignant asthma. Very high thrombocytosis (1000 103μl and above) can be observed after splenectomy.

platelet function test.

Platelet adhesion (retention) is determined by the number of platelets retained in the column after passing a certain volume of blood through a glass ball column at a standard speed, and the retention index is calculated using a special formula. Normally 20-55%. A decrease to 0% is observed in congenital thrombocytopathies, Willebrand's disease.

Platelet aggregation is tested by mixing platelet plasma with an aggregation stimulator, the presence of platelet aggregates in the test tube is determined (macroscopic quality method), and the number of aggregates is determined using

an aggregometer using a quantitative photometric method. Normally, large platelet aggregates should be formed in 10 60 seconds. Platelet aggregation is not observed in Glansman thrombosthenia.

Stabilizers-free blood is taken into a blood clot retraction tube and poured into a 37oC water bath. Then it is determined whether there is retraction of the blood clot. Normally, the blood clot should begin to retract after 30-60 minutes. It is not observed in severe thrombocytopenia and pathologies.

Humoral factors of blood coagulation and
ways of their activation.

Activation of blood coagulation factors provides secondary hemostasis. As a result of secondary hemostasis, a thrombus develops, which completely stops bleeding and prevents rebleeding. blood clotting factors are designated by Roman numerals according to the international nomenclature, the suffix "a" indicates the active form of the factor. 13 factors involved in blood coagulation include prekallekrin (Fletger factor) and high molecular weight kininogen (Fitzgerald Floje factor). blood clotting factors can be divided into 3 groups:

fibrinogen group: factors I, V, VIII and XIII. Factors of this group have the highest molecular mass, are labile and are quickly consumed in the blood clotting process. Only these factors are considered substrates for the fibrinolytic enzyme plasmin. The amount of these factors increases when oral contraceptives are taken during pregnancy, inflammatory processes.

prothrombin group II. VII. The synthesis of factors IX and X depends on vitamin K. Vitamin K enters the body with nutrients and is synthesized by intestinal flora. Factors of this group are highly stable and increase during pregnancy and under the influence of oral contraceptives, their activity is inhibited under the influence of warfarin and other anticoagulants.

contact group: XI, XII, prekallecrein and high molecular kininogen. These factors are involved in the activation of blood coagulation by the inner layer. Moderately stable and not consumed during blood clotting. Contact group factors are activated by contact with the negatively charged surface of subendothelium or collagen, and also participate in kinin formation, fibrinolysis, and activation of the complement system.

Characteristics of blood clotting factors

Factor I fibrinogen stable globulin, molecular mass 341000 Dalton, is synthesized in the liver. Its amount in plasma is 2 4 g\l, the minimum level necessary for hemostasis is 0.8 g\l. Fibrinogen belongs to the "acute phase" proteins and determines blood viscosity and the intensity of platelet aggregation. An increase in fibrinogen is observed in inflammatory processes, tumors, myocardial infarction and provides information about the high risk of thrombosis.

5.2. Analytical part

New pedagogical technology Boiled potatoes"

The assistant takes a piece of paper in the shape of a crumpled potato and throws it to the student asking a question about the topic. The student must answer the question immediately, otherwise he will be burned.

Questions:

How many plasma factors?

How many platelet factors?

What is platelet aggregation?

What is platelet adhesion?

What is clot retraction?

Bleeding time for Duke?

Blood clotting time according to Lee White?

first plasma factor?

involved in primary hemostasis?

What happens in the first phase of blood clotting?

Situational issue.

Issue #1

The patient is 35 years old. Complaints: frequent nosebleeds, profuse bleeding during menstruation, blood clots in minor traumas. Objective: the general condition is relatively satisfactory, numerous hemorrhages on the skin. The liver protrudes +1 cm below the rib cage, the dome of the spleen is palpable. Hemogram: Hb 90 g/l, er.3.4x1012/l, RK 0.8, platelet 5x109/l, t/ya 3%, s/ya 62%, eosinophil 3%, basophil 0%, lymphocyte 25%, monocyte 5%, ECHT 21 mm/h.

Questions: I. Your initial diagnosis:

II. It is necessary to diagnose the mentioned disease:

III. In this disease, hemogram shows:

Answers: 1. Idiopathic thrombocytopenic purpura

2. myelogram, coagulogram, blood clotting time

3. Thrombocytopenia

Issue #2.

The patient is 17 years old. Complaints: general weakness, dizziness, bleeding from the nose and gums, cough, chest pain, body temperature up to 390C. From the anamnesis: he has been healthy since childhood. The above complaints have been bothering me for 1 week. Lens: skin and mucous membranes are pale. Blisters on the skin at the site of injections. On percussive palpation, palpable sound is associated, on auscultation wet and sonorous wheezing, crepitation. Heart tones are related. Liver and spleen protrude +2 cm below the rib cage. Hemogram:

Hb 76 g/l, er.2.2x1012/l, RK 1.0, reticulocyte 10, platelet 35x109/l, leukocyte 4.7x109/l, blast 69%, t/ya 1%, s/ya 24 %, lymphocyte 5%, monocyte 1%, ECHT 34 mm/h.
Questions: I. Your initial diagnosis:
II. It is necessary to diagnose the mentioned disease:
III. In this disease, hemogram shows:
Answers: 1. acute leukemia
2. general analysis of blood, reticulocytes, thrombocytes
3. appearance of blasts, anemia, thrombocytopenia
Issue #3.
The patient is 35 years old. Complaints: nosebleeds, heavy menstruation, bruises on the skin. Lens: hemorrhages on the skin. Liver +1 cm, spleen not enlarged. Hemogram: Hb 100g/l, er.3.4x1012/l, RK 0.8, thrombocyte 50x109/l, leukocyte 8x109/l, t/ya 6%, s/ya 62%, eosinophil 3%, lymphocyte 25%, monocyte 5%, ECHT 21 mm/h. Myelogram: the separation of platelets from megakaryocytes is disturbed.
Questions: I. Your initial diagnosis:
II. It is necessary to diagnose the mentioned disease:
III. Myelogram shows this disease:
Answers: 1. Idiopathic thrombocytopenic purpura
2. coagulogram, blood clotting time
3. an increase in the number of megakaryocytes
Issue #4.
Patient A. 27 years old. His complaint was that his temperature rose to 38.2oC, swelling and pain in the lower leg joints, and his temperature appeared after a rash on his leg. From Anamizi: The disease started suddenly, two weeks ago he had flu in his leg. Objective: there is a symmetrical rash with small dots on the legs, which does not burn when pressed. Peripheral lymph nodes were not enlarged. Calf ligaments have not changed, there is no pain on palpation, the movement is active. Liver and spleen are not enlarged. In the analysis: Hb 130 g/l, er.4.2x1012/l, RK 1.0, reticulocyte 12, platelet 240x109/l, leukocyte 9.2x109/l, t/ya 3%, s/ya 68%, eosinophil 3 %, lymphocyte 30%, monocyte 5%, ECHT 17 mm/h.
Questions: I. Your initial diagnosis?
II. What happens in the capillaries in this disease?
III. Kidney damage in this disease?
Answers: 1. hemorrhagic vasculature
2. aseptic inflammation of the endothelium
3. Inflammation of ball capillaries
Issue #5.

Patient I, 18 years old. He came to the department complaining of an increase in temperature to 38˚C, rashes, swelling and pain in the calf ligaments. Anamnesis: 2 weeks ago he had acute follicular angina and was treated with various drugs. When looking at the lens: there are many hemorrhagic hemorrhagic rashes on the skin and they are symmetrically located (mainly on the legs). The liver is palpable, the spleen is not enlarged. Jgut symptom is positive. In the analysis: thrombocyte 240x109/l, leukocyte 10x109/l, ECHT - 30 mm/h. Blood clotting time starts at 1 min 23 sec and ends at 3 min.

Questions: I. Your initial diagnosis?

2. What group of diseases does this disease belong to?

3. Can it be the cause of the disease?

Answers: 1. hemorrhagic vasculitis

2. immune complex diseases

3. chronic angina

Issue #6.

Patient N 10 complained of pain in the calf joint and lack of movement in this joint. In the anamnesis: the patient had bleeding from the nose and bleeding from the ligaments. In the family, similar symptoms were also observed in 3 brothers. Lens: joints are enlarged, movement is reduced. Leg muscles are atrophied. Blood was burned on the front surface of the abdomen. The shin ligament is enlarged and hyperemic due to hemorrhage, swollen, and movement is reduced.

Questions: I. Your initial diagnosis?

2. Can there be a complication of the disease?

3. Factors to be determined to diagnose the disease?

Answers: 1. hemophilia

2. bleeding

3. VIII, IX, XI

Issue #7.

Patient S. 15 days ago complained that her menstruation was heavy and irregular and turned to the doctor. Anamesida: mensis 14 esshi. Menstruation is abundant, painless, lasts 8-15 days. Objective: the skin is pale, there is a slight petichal rash. When examined: Hb 80 g/l, er.3.0x1012/l, RK 0.9, reticulocyte 24, platelet 20x109/l, leukocyte 8x109/l, t/a 66%, s/a 2%, eosinophil 1%, monocyte 7%, lymphocyte 24%, ECHT 10 mm/s.

Questions: I. Your initial diagnosis?

2. Is it used to treat the disease?

3. Factors to be determined to diagnose the disease?

Answers: 1. thrombocytopenia

2. Prednisolone

3. thrombocytopoiesis
Issue #8.
Bleeding was observed during the eruption of the first tooth in a 6-month-old child. His brother died after hemorrhaging a tooth. Examination revealed hemophilia A.
Questions: I. What clotting factor is missing?
2. What diseases should be distinguished?
3. What treatment does the patient need?
Answers: 1. factor VSH (antihemophilic)
2. ITP, thrombocytopathy
3. VSHfactor cryoprecipitate
Issue #9.
The patient is 38 years old. For many years, skin rashes on the mucous membrane of the mouth are annoying. "Idiopathic thrombocytopenia" was diagnosed. He has been treated with prednisolone for a long time, and the amount of platelets increases to 80-90x10 9/l. But hemorrhagic syndrome persists.

Qumrijon😉, [21/01/2023 00:31]
Questions: 1. Your next treatment strategy.
2. Show the complication of the disease
3. dif. diagnosis
Answers: 1. cytostatic addition
2. iron deficiency anemia, hemorrhage
3. thrombocytopathy, hemophilia, aplastic anemia, leukemia
Issue #10.
A 30-year-old patient turned to the doctor with a complaint of increasing weakness, shortness of breath during physical exertion. Lately, nosebleeds and prolonged periods have been observed. Bleeding in the form of a petechial spot was detected during the examination. Blood analysis: Hb 40 g/l, er.1.8x1012/l, RK 0.8, reticulocyte 0.5, platelet 61x109/l, leukocyte 2x109/l, t/y 10%, s/y 14%, lymphocyte 66%, monocyte 10%, ECHT 50 mm/h.
Questions: 1. What is your presumed diagnosis?
2. What tests should be done to confirm the diagnosis
3. What changes in analyzes are characteristic of the disease?
Answers: 1. Aplastic anemia
2. myelogram, coagulogram
3. Hb 40 g/l, er.1.8x1012/l, thrombocyte 61x109/l, leukocyte 2x109/l, lymphocyte 66%, ECHT 50 mm/h.
TESTS.

1. Schenlein is affected by Genox disease.
Capillaries*
Veins
Arteries
arterioles*
Large veins
2. How is the kidney damaged in Shenlein Genox disease
Ball capillaries*
Inflammation of the joints
capillary inflammation*
Interstitial inflammation
No damage
3. Pathogenesis of hemophilia is related to what deficiency
Platelet
YIII factor *
Factor IX*
Factor XI*
Fibrinogen
4. Characteristic for the III stage of DVS syndrome
deep hypocoagulation
hypercoagulation*
coagulation factor degradation and consumption*
accumulation of proteolysis products*
failure of the anticoagulation mechanism
5. Why is it not recommended to administer fibrinogen in DVS syndrome stage III
failure of the anticoagulation mechanism*
hypocoagulation*
thrombocytosis
metabolic disorders
accumulation of proteolytic products
6. It develops in the pathogenesis of fibrinogenemia
fibrinogen deficiency*
mixed
thrombosis
G. hypercoagulation*
D. hypocoagulation*
7. Characteristic for Verlgof's disease?
thrombocytopenia*
leukopenia

left shift of leukoformula
thrombocytosis
the presence of blast cells in the peripheral deposit
8. Characteristic of Shenlein-Genox disease?
immunocomplex pathology*
inflammatory damage of vessels
vessel wall structure defect
infection
metabolic disorders
9. An important examination for the diagnosis of Verlgof's disease:
myelogram, hemogram, coagulogram *
proteinogram
x-ray
EKG
urinalysis
10. In the diagnosis of Glansman's disease, it is necessary to determine:
platelet function *
amount of fibrinogen
platelet count
erythrocyte count
protein content
11. Not characteristic of Verlgof's disease?
hyperthrombocytosis*
thrombocytopenia
erythrocytopenia
hemoglobin decrease
megaloblastic deposits
12. Is it possible to get basic diagnostic information in DVS syndrome?
from coagulogram*
from hemogram
from myelogram
from biochemical indicators
from EKG
13. The following factors should be identified in the diagnosis of hemophilia:
VIII, IX, XI*
III, XI
platelet
V-X
II, VII

14. To diagnose fibrinogenemia, it is determined:
plasma fibrinogen*
prothrombin
platelet count
Factor XIII
platelet activity
15. What changes are observed in the blood analysis in hemophilia
anik (characters) sorry load*
thrombocytopenia
leukocytosis
pancytopenia
lymphopenia
16. Randy Osler's disease is based on:
metabolic disorder
to immunocomplex pathology
to viral pathology
subendothelial nodal development*
to the breakdown of the mining coagulation system
17. The pathology of Willebrand's disease is based on:
to platelets
mine vessel walls
to erythrocytes
to factors of 8*
to a factor of 9
18. DVS syndrome is not characteristic for the 3rd stage:
hypocoagulation
decomposition and consumption of mining stopping factors
assembly of proteolysis products
hypercoagulability*
the end of the anticoagulation mechanism
19. The main role in the origin of hemorrhagic diathesis is played by:
platelet status
plasma factors in the mine circulation system
plasma factors in the antikone circulation system
condition of the vein wall
all of the above*
20. What are deposits in hemorrhagic vasculitis related to:
increased vascular wall permeability*
fibrinogen deficiency

platelet deficiency
thromboplastin formation disorder

References

1. Иноятова Ф.Х., Бабаджанова Ш.А., Курбонова Н.Н., Курбонова З.Ч. Гемостаз: основные принципы функционирования, методы оценки, патофизиологические аспекты: методическое пособие. – 2014. - Ташкент, 2014. – 43 с.

2. Курбонова З.Ч., Бабаджанова Ш.А. Цитологик ташхисга кириш: ўқув қўлланма. Тошкент, 2022. 137 б.

3. Курбонова З.Ч., Бабаджанова Ш.А. Цитологик ташхисга кириш. ДГУ №16152. Мурожаат № 20221896. Олинди 2022/05.

4. Курбонова З.Ч., Бабаджанова Ш.А. Диагностика и лечение приобретенной тромбоцитопатии: методические рекомендации. – Ташкент, 2018. – 21 с.

5. Курбонова З.Ч. Рак олди ҳолатлари, яхши ва ёмон сифатли ўсмалар цитологик диагностикаси: ўқув – услубий қўлланма/ - Тошкент: Ҳилол нашр, 2021. - 49 б.

6. Курбонова З.Ч., Бабаджанова Ш.А., Саидов А.Б. Гематологик касалликлар цитологик диагностикаси: ўқув – услубий қўлланма/ - Тошкент: Ҳилол нашр, 2021. - 57 б.

7. Курбонова З.Ч., Бабаджанова Ш.А. Цитологик диагностика асослари: ўқув-услубий қўлланма. – Тошкент: ТТА босмахонаси, 2021. - 134 б.

8. Курбонова З.Ч., Бабаджанова Ш.А. Цитологик ташхисга кириш: электрон ўқув қўлланма. – Тошкент, 2021. - 134 б.

9. Kurbonova Z.Ch., Babadjanova S.A. Sitologik tashxisga kirish: o‘quv qo‘llanma. Toshkent, “Hilol nashr”, 2021. 152 b.

10. Kurbonova Z.Ch., Babadjanova Sh.A. Laboratoriya ishi: o‘quv qo‘llanma. Toshkent, 2022. 140 b.

11. Kurbonova Z.Ch., Babadjanova S.A. Sitologik diagnostika asoslari: o‘quv – uslubiy qo‘llanma. Toshkent. - “TTA nashriyoti”, 2022. -47 b.

12. Kurbonova Z.Ch., Sayfutdinova Z.A. Peshobning klinik laborator tashxisi: o'quv –uslubiy qo'llanma. Toshkent, 2022. 49 b.

13. Kurbonova Z. C., Tairova G. B., Karimova A. A. Testlar to'plami : o'quv-uslubiy qo'llanma. – Toshkent tibbiyot akademiyasi, 2022. 76 b.

14. Kurbonova Z. C., Sayfutdinova Z.A. Testlar to'plami: o'quv-uslubiy qo'llanma. – Toshkent tibbiyot akademiyasi, 2022. 86 b.

15. Nuriddinova N.F., Kurbonova Z.Ch. Najasning klinik laborator tashxisi: o'quv-uslubiy qo'llanma. Toshkent, 2022.

16. Saidov A.B. Kurbonova Z.Ch., Babadjanova Sh.A. Gematologik kasalliklar sitologik diagnostikasi: o‘quv uslubiy qo‘llanma. Toshkent, Toshkent tibbiyot akademiyasi bosmaxonasi, 2021. – 56 b.