**MINISTRY OF HEALTH OF UKRAINE**

**ODESA NATIONAL MEDICAL UNIVERSITY**

Departments of Pediatrics №2

**CONFIRMED by**

Vice-rector for research and educational work

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# METHODOLOGICAL RECOMMENDATIONS ON PRACTICAL CLASSES FOR STUDENTS

International Medical Faculty, course 6

Educational discipline "**PEDIATRICS**"

**Approved**
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**Topic 8 . Practical lesson 21 - 23** . Pallor in children. Pallor in children: a problem-oriented approach in pediatrics. Differential diagnosis of diseases and conditions accompanied by pallor in children. Treatment of diseases and conditions that are accompanied by pallor in children. Emergency care for acute bleeding. Lymphadenopathy in children. Differential diagnosis. Tactics of the doctor. Indications for consultation with a pediatric hematologist. Hepatomegaly in children. Differential diagnosis. Tactics of the doctor. Splenomegaly in children. Differential diagnosis. Tactics of the doctor.

**Purpose:** To identify different clinical variants and complications of the most common diseases accompanied by the symptom of pallor in children. To determine the tactics of patient management in the most common diseases accompanied by the symptom of pallor in children. Demonstrate the ability to keep medical records of children with diseases accompanied by the symptom of pallor in children. To draw up a plan for the examination of a sick child and to interpret the obtained results in diseases accompanied by pallor in children. Carry out differential diagnosis and make a preliminary clinical diagnosis for the most common diseases accompanied by pallor in children. Diagnose and provide emergency care for emergency conditions accompanied by the symptom of pallor in children.

**Basic concepts:**

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| Anemia | pathologic clinical and laboratory symptom complex that characterized by decrease content hemoglobin and / or erythrocytes in a unit volume of blood |
| Deficiency anemia | - this variety anemia, the cause of which is deficiency iron (sideropenic), protein (protein-deficient), vitamins (B12, folate-deficient ), trace elements in the body , associated with insufficient him income or assimilation or elevated output |
| Erythrocyte indices | • average volume erythrocyte (MCV) - MCV - average volume erythrocyte is _ average volume that _ occupies one erythrocyte, determined in femoliters (fl) or cubic micrometers (1 fl = 1 μm3) ( 80-100 fl )<br>• average contents of hemoglobin in the erythrocyte (MCH), determined by the formula: MCH = (Hemoglobin *10)/ amount erythrocytes 1012/l. It is determined most often in picograms (pg). It characterizes number hemoglobin in 1 erythrocyte, corresponds colored indicator. (27-33)<br>• average concentration hemoglobin in volume erythrocytes (MCHC) - Average concentration hemoglobin in the erythrocyte , characterized by " density filling » hemoglobin in the erythrocyte , determined by the formula: MCNS = ( Hemoglobin *100)/Hematocrit (%) 32-36g%<br>This indicator increases with: spherocytosis and ovalocytosis , sometimes with hyperosmolar water- electrolyte disturbances exchange , wrong elevated in hyperlipidemia . It decreases with: iron deficiency anemia , thalassemia , sideroblastic anemia , hypoosmolar water- electrolyte disturbances exchange _<br>• RDW – distribution erythrocytes by volume , this indicator gives quantitative assessment anisocytosis , maybe expressed as: RDW-CV - and shown as a percentage as far as volume erythrocytes deviates from average value , and RDW-SD – shows |

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| | the difference in volume between the smallest and the largest erythrocyte . 11.5-14.5 %<br>Reticulocytes - 0.5-2 %<br>Non-ferrous the indicator is 0.85-1.0 |
| Iron deficiency | iron < 9 μmol /l ( normally 10.6 – 33.5 μmol /l), coefficient saturation transferrin iron < 17% ( normally 20-50%), decrease ferritin serum below 15 μg/l ( normally 32–68 μg/l, increased in inflammatory conditions), zinc protoporphyrin > 80 μg/l ( increases in poisoning lead _ more than 200 μg/l, with leukemia and apastic anemia - more than 100 μg/l, general iron-binding property serum > 63 μmol /l ( normally 45–72 μmol/l), latent iron-binding property serum > 47 μmol /l, hepsidin ( norm - less than 10 ng / mL . Increases during ignition )<br>Soluble transferrin receptors present on the membrane surface of almost all cells , but in the largest amounts on erythroid precursors and therefore can serve indicator activity erythropoiesis Reticulocyte hemoglobin In normal conditions after getting out of the red bone brain reticulocytes need 24 hours to become mature erythrocytes, therefore hemoglobin reticulocytes (Rhethemoglobin) displays recent synthesis of hemoglobin , ie shows the reserves in the body of available fat participates in erythropoiesis of iron in the last 48 hours (In infants and young children <27.5 In adults ≤28.0) |
| Folate deficiency | MCV > 100, CI >1.05, number of reticulocytes low, nucleated erythrocytes with megaloblastic morphology, there may be neutropenia and thrombocytopenia, neutrophils large, some with hypersegmented nuclei (more than 5 segments).<br>level folic acid in the serum blood is <3 (N5-20 ng /ml); level folates erythrocytes - less than 140 (N150-600 ng / ml). Level serum iron and vitamin B12 of blood is usually normal or elevated _ LDH activity (lactate dehydrogenase), an ineffective marker erythropoiesis, noticeable elevated |
| Vitamin B - 12 deficiency | MCV > 100, CI >1.05, number of reticulocytes low , nucleated erythrocytes with megaloblastic morphology , there may be neutropenia and thrombocytopenia , neutrophils large , some with hypersegmented nuclei ( more than 5 segments ). Sirovatkova activity lactate dehydrogenase , an ineffective marker erythropoiesis , noticeable elevated _ |
| Diagnosis | 1. The nature of anemia ( eg iron-deficient - in case if the last was confirmed by definition indicator content serum ferritin _ blood ). If the nature of anemia is not established , it needs description , which 24 provides designation changes morphology erythrocytes and is marked as "unspecified" and needs further referral patient for consultation with a hematologist).<br>2. The cause of anemia ( for example alimony genesis or because of blood loss ). If the cause is specified in the diagnosis blood loss , then information about it is provided source ( if him detection ) or is given the most likely her the source ( under the question mark ) that provides further examination the patient<br>3. Degree severity anemia _<br>4. Availability complications that are a consequence anemia _ |

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| | For example : Iron deficiency anemia because of blood loss from the gastrointestinal tract<br>tract (?), severe , complicated hypoxic myocardial dystrophy . |
| Medical means (the order of placement does not affect the order of assignment): | Drugs divalent iron , oral forms : iron sulfate, iron fumarate , iron oxide saccharate .<br>Drugs trivalent iron , oral forms : complex of iron (III) hydroxide with polymaltose .<br>Drugs iron , parenteral forms : iron dextrin, iron carbomaltose , iron (III) hydroxide-sucrose complex.<br>Drugs iron in combination from folic acid: iron fumarate + folic acid, complex of iron (III) hydroxide with polymaltose + folic acid, iron sulfate + folic acid . Drugs iron in combination with other drugs: iron fumarate + folic acid + cyanocobalamin , iron ammonium citrate + folic acid<br>+ cyanocobalamin , iron fumarate + folic acid + cyanocobalamin + ascorbic acid + zinc sulfate, iron sulfate + ascorbic acid , iron sulfate heptahydrate + ascorbic acid , iron gluconate + manganese gluconate + copper gluconate, iron sulfate heptahydrate + D, L-serine .<br>Products donor's blood that _ they can to be produced in service facilities blood : erythrocytes . |
| Doses | On the first stage in children under 3 years old — 3–5 mg/kg/day of elementary iron ;<br>from 3 to 7 years — 50–70 mg/day of elementary iron; older than 7 years — up to 100 mg/day of elemental iron. At the second stage — therapy saturation ( restocking of iron in the body ) — a dose of elemental iron per day should be 40–50% of primary therapeutic doses , therapy continues for three months _ In case initial heavy degree of ZDA stage therapy saturation iron continues up to 6 months , and in premature babies children - up to 12 months . On the third at the supporting stage therapy — dosing approaches and timing _ appointment drugs iron in each case individual |
| Transfusion of erythrocytes | considered at level hemoglobin <70 g/l, or at higher levels levels (<100 g /L) in patients with severe symptoms, or for those patients who anemia is difficult to tolerate ( patients with lesions cardiovascular and respiratory systems). |
| Rating efficiency treatment patients | On the day of the appointment of the iron preparation patient sent to general analysis blood , which necessary make after the 21st day from the start of treatment with an iron preparation .<br>After the 21st day of treatment is evaluated increase hemoglobin . In the case of an increase in hemoglobin by about 20 g/l (+1 g/l/day) reaction interpreted as positive; in case lack of growth - as negative; intermediate the value is insufficient the answer In case positive reactions continues treatment _ Treatment continues within 3 months (6 months - for a severe degree anemia ) after normalization equal hemoglobin . Every month until normalization equal hemoglobin is evaluated general analysis blood _ After the end of the course of treatment is assigned taking an iron preparation within 3 months for replenishment stocks iron in the body . |
| Hemolytic anemia | **Hemolysis is premature destruction erythrocytes .**<br>Normal duration life erythrocytes - 110-120 days , about 0.85% of erythrocytes are destroyed and removed from circulation , being |

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| | replaced young erythrocytes .. With hemolysis duration life is shortened , the level erythrocytes falls , level erythropoietin grows that _ stimulates making new ones erythrocytes , an indicator this is an increase level reticulocytes .. Indicator – ( index reticulocytes = actual hematocrit/ normal hematocrit for age ) * 1/μ, where μ is the maturation factor . The normal index is 1. With hemolysis , it increases 2-4 times with a severe storm hemolysis and 6-8 times in chronic hemolysis .<br>Also increases Indirect bilirubin blood , LDH level , urobilinogen urine _ The child has younger than 4 years are often found gallstones _ _ bubbles _<br>Hemolytic anemia you can classify as , conditioned pathology membranes , enzymes and hemoglobin and extracellular ones associated with action antibodies , mechanical factors or factors plasma _ Majority cellular defects inherited , the majority extracellular - acquired . |
| Hemolytic anemia, classification | Hemoglobinopathy (α- thalassemia , β- thalassemia , sickle cell disease cells , unstable hemoglobin )<br>Defects membranes erythrocytes Hereditary spherocytosis ( Hereditary elliptocytosis , piporoikilocytosis and related disorders , syndromes hereditary stomatocytosis , xerocytosis , hydrocytosis , Rhesus null syndrome, GLUT1 deficiency , Tangier disease, abetalipoproteinemia , phytosterolemia )<br>Enzymopathies (glucose-6-phosphate dehydrogenase), Emden-Meyerhoff defect (( glycolysis ) ( pyruvate kinase , hexokinase , phosphate isomerase glucose , phosphofructokinase , triosephosphate kinase , phospholfacenate-phosphosphasphenselphasena ), deficiency of 5'-nucleotidase<br>Immune mediated primary , secondary ( autoimmune or inflammatory disorders Evans syndrome , primary immunodeficiency syndrome , Wirsittich syndrome immunodeficiency , Acquired immunodeficiency , HIV infection , ( lymphoproliferative disorders : lymphomas ), Infection , Transplantationb Cold agglutination , fragmentation erythrocytes , others mechanical causes, hypersplenism |
| Clinical and laboratory findings in hemolytic disease anemia | Pallor , and jaundice , with plenomegaly , gallstones _ _ cysts , neonatal jaundice , family history, anemia , splenectomy , cholecystectomy , ↑ number reticulocytes , ↑ RDW ( caused by ↑ number reticulocytes ), abnormal morphology RBC, ↑ Indirect bilirubin ( normal direct bilirubin ), decreased level haptoglobin in serum blood , ↑ level urobilinogen in urine , hemoglobinuria ( absence of RBC in urine ), ↑ LDH level |
| Hereditary spherocytosis (Minkowski's disease Shofar), an autosomal dominant form of inheritance, may be a new mutation. | Related to hereditary an anomaly membrane erythrocytes associated with deficiency _ ankyrin , spectrin and protein 3 in the membrane . Erythrocytes acquire spherical shape, due to this part are destroyed in the spleen , is reduced duration life erythrocytes . Removal spleen significantly improves quality life patients _ The clinical picture varies from asymptomatic form to heavy forms with frequent aplastic crises. inherent **normocytic** (MCV -80-100 fL) anemia , however **diameter erythrocytes reduced** , erythrocytes (up to 20% of erythrocytes ) have **spherical** shape, hyperchromic, |

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| | high reticulocytosis (4-20%, on average 10%), decrease osmotic resistance erythrocytes , and MCH 32-36 g/ dL hyperbilirubinemia, increase spleen , bilirubin gallstones. At loved ones relatives usually meet gallstones _ _ cysts , Gilbert's syndrome (?), hyperbilirubinemia , removal spleen in the anamnesis . Differential the diagnosis is autoimmune hemolytic anemia _ Coombs test - allows exclude autoimmune anemia _ Treatment - for anemia with a level hemoglobin more than 100 g/l and reticulocytosis less than 10% - observation , appointment folic acid in a dose of 10 mg per day. For more difficult forms ( low Indicators physical development , frequent hypo or aplastic crisis , cardiomegaly , - solution question about total or partial splenectomy , appointment penicillin V 125 mg twice a day up to 5 years , constantly .<br>To everyone to children recommended carrying out vaccinations by age , vs meningococcus , annually against flu _ Children are observed by a hematologist.<br>Aplastic and hypoplastic crises usually develop in the background encounters with parvovirus B19, but can be caused by other agents or stress _ Appear quickly increasing anemia , and associated shortness of breath , cardiovascular insufficiency . Leukocytes also they can decrease _ Treatment is symptomatic , aimed at providing functioning body _ |
| Sickle cell anemia, homozygous | HbSS phenotype in severe I will run have place sequestration crisis ( deposit blood in the spleen , liver - increase liver , spleen , blood pressure decrease - BCC decrease , hypovolemic crois ) and vasoocclusive crisis ( pain bones , joints, stomach , chest, presence acute chest ( respiratory ) syndrome (ACS - acute chest syndrome ) - with involvement a whole lot more often left the lungs lungs , respiratory tract deficiencies ). In the first queue corrections needs hypovolemic state - normalization of BCC at the expense of physical solution , in parallel - raising saturation to the level of pO2=95%, 100% oxygen through a mask. Appointment macrolides and cephalosporins . It is possible transfusion erythrocytes , if saturation does not increase . In the future - observation by a hematologist, up to 5 years - penicillin 125 mg twice a day, after 5 years - according to indications . Also full mandatory immunization , + against the flu annually _ After 5 years - solution questions about splenectomy . |
| **Thalassemia** | ***β thalassemia*** is related to completeness absence products ( $\beta^0$) or partial reduced production of ($\beta^+$) β- globin . Common to African Americans , Central Mediterranean , and Central Asian population , Indians . In the blood homozygote β is present only hemoglobin F , or hemoglobin F and E. If carried - differential diagnosis with iron deficiency anemia _ It is characteristically microcytic normochromic anemia with inadequate reticulocytosis ( usually more than 3%, however it does not increase level erythrocytes , therefore inadequate reticulocytosis ), RDW - normal - 11.5-14.5 %. It is believed that trial therapy with iron preparations without effect is beneficial carriers thalassemia , and needs additional examination _ Detection in the future examinations of HvA2 more |

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| | than 3 % and weighted family history is confirmed diagnosis carriers thalassemia . With thalassemia is contraindicated appointment drugs iron for a long time the term can lead to hemosiderosis .<br>With severe form ( homozygotes , severity depends in the type of mutation ) - hemolytic anemia with manifestation for 2-6 months life , with expressed increasing weakness ( naivt lethargy ), poor appetite and progressive cardiac decompensation (when decreasing hemoglobin below 40 g/l). Anemia microcytic with the absence of a peripheral smear of blood normal forms of erythrocytes , with erythrocytes that contain a nucleus, reticulocytosis about 8%, which is not effective , because it does not support normal growth erythrocytes , whiskey level of indirect bilirubin . . Children from an early age age abuse hemotransfusion and chelation therapy , splenectomy after 5 years |
| Anemia associated with shortages enzymes | Deficit pyruvate kinase<br>Glucose-6 phosphate dehydrogenase deficiency is hemolytic anemia that is induced infections and reception some drugs ( trimethoprimsulfamethoxazole , chloramphenicol , nalidixic acid, acetylsalicylic acid, etc., antimalarials drugs , due to hepatitis..), and the so -called favism - use beans causes development hemolytic crisis. |
| Anemia because of violation hematopoiesis | Congenital hypoplastic anemia ( Diamond - Blackfan - erythr usually macrocytic , but neutrophils do not have hypersegmentation , or others signs megaloblastic anemia ( assessed by peripheral blood smear blood ). |
| Congenital dyserythropoietic anemia | It is not homogeneous class inherited violations that _ arise because of deviations late erythropoiesis . These rare state are characterized anemia different degree , ineffective erythropoiesis and secondary hemochromatosis . Dyserythropoiesis is the main cause of anemia , but plays an important role reduced period life circulating erythrocytes . CDA historically classified into 3 main types (I, II and II) based on excellent morphology bone brain and clinical features |
| Acquired pancytopenia | Preparations , chemical substances , toxins , infectious radiation and immune agents _ disorders they can to cause pancytopenia by direct destruction of hematopoietic predecessors , violations microenvironments brain , or mediated immunogenic suppression elements brain _ different medical means and chemical substances , including certain medical means , insecticides , antibiotics , anticonvulsants means , non-steroidal anti-inflammatory facilities and recreational drugs _ One of the most noticeable agents - benzene, chloramphenicol , gold and 3,4-methylenedioxymethamphetamine ( ecstasy ). A number of viruses can directly or indirectly to cause insufficiency bone brain _ Parvovirus B19 is classic associated with isolated aplasia erythrocytes . Lasted pancytopenia maybe arise after infection many hepatitis viruses , herpes viruses , virus Epstein - Barr, cytomegalovirus and HIV. Patients from signs deficiencies bone brain also trace evaluate for inherited forms of cerebral failure , paroxysmal night hemoglobinuria and vascular diseases collagen . |

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| | Pancytopenia without peripheral blasts can be caused substitution cells neuroblastoma |
| Clinic , diagnostics , diff diagnosis | Pancytopenia leads to an increase risk cardiac deficiencies , infections , bleeding and fatigue . Acquired pancytopenia is usually characterized anemia , leukopenia and thrombocytopenia when established elevated values cytokines in serum blood _ Others therapeutic collagen disorders such as cancer vascular disorders , PNH and infections which they can respond to a specific therapy (IV immune globulin for parvovirus ), follow take into account in the differential diagnosis . It is important to be thorough peripheral smear examination blood availability _ morphological signs erythrocytes , leukocytes and platelets . For assessment erythropoietic activity number should be carried out reticulocytes . In children always trace consider possibility congenital pancytopenia , and for assessment anemia Fanconi trace carry out chromosomal analysis . The presence of fetal hemoglobin indicates congenital pancytopenia , but is not diagnostic . For assessment the possibilities of PNG flow cytometric analysis erythrocytes on CD55 and CD59 are the most sensitive test. Examination bone of the brain should include both aspiration and biopsy , and the brain should be carefully to be assessed on the subject of morphological signs , cellular and cytogenetic pathologies |
| Treatment _ | Treatment children from acquired pancytopenia requires a complex supporter treatment along with an attempt treatment main deficiencies brain _ For patients with human leukocyte antigen identical to the donor of a family member , allogeneic transplantation hematopoietic stem cells (HSCT) offers a 90% chance of long-term survival _ A typical scheme of preparation today consists of cyclophosphamide , fludarabine and antithymocyte horse globulin (ATH). The risks associated with such an approach include immediate complication transplantation , graft failure and graft- versus -host disease. late unfavorable consequences associated with transplantation can _ _ comprise secondary cancerous disease , cataract, low growth , hypothyroidism and dysfunction of the gonads |

**Iron deficiency anemia (IDA)** is a pathological condition characterized by a decrease in the hemoglobin content due to iron deficiency in the body in violation of its intake, assimilation or pathological losses.

The causes of IDA in children:
-insufficient initial level of iron in the body (violation of uteroplacental circulation, fetoplacental bleeding, fetal transfusion syndrome in multiple pregnancies, intrauterine melena, prematurity, multiple pregnancies, deep and long-term iron deficiency in the body of a pregnant woman, premature or late intrauterine cord ligation, or abnormalities in the development of the placenta and umbilical cord vessels).
- increased need for iron (premature babies, children with a large body weight at birth, children in the second half of life).
- insufficient iron content in food (early artificial feeding with cow or goat milk, flour, dairy or dairy-vegetarian food, unbalanced diet, in which there is not enough meat products).

- increased iron loss due to bleeding of various etiologies, intestinal absorption disorders (chronic bowel disease, malabsorption syndrome), as well as significant and prolonged hemorrhagic uterine bleeding in girls.
- violation of iron metabolism in the body (pre- and pubertal hormonal imbalance).
- violation of the transport and utilization of iron (hypo- and atransferinemia, enzymopathies, autoimmune processes).
-insufficient resorption of iron in the digestive tract (post-resection and agastral states).

**Stages of IDA development**
- Prelative (depletion of tissue iron stores, blood counts are normal; no clinical manifestations).
- Latent (iron deficiency in tissues and a decrease in its transport fund; blood counts are normal; the clinical picture is due to trophic disorders that develop as a result of a decrease in the activity of iron-containing enzymes and is manifested by sideropenic syndrome - epithelial changes in the skin, nails, hair, mucous membranes, distortion of taste, smell, violation of the processes of intestinal absorption and astheno-vegetative functions, decreased local immunity).
- Iron deficiency anemia (more pronounced depletion of tissue reserves of iron and mechanisms for compensating for its deficiency; deviations from the norm of blood counts depending on the severity of the process; clinical manifestations in the form of sideropenic syndrome and general anemic symptoms caused by anemic hypoxia - tachycardia, muffled heart sounds, systolic noise, shortness of breath during physical exertion, pallor of the skin and mucous membranes, arterial hypotension, an increase in asthenic-neurotic disorders).

Iron deficiency occurs when the need for iron exceeds absorption or in the event of excessive iron loss. This is often due to many reasons. Usually, iron deficiency is observed in children during periods of increased body demand for this trace element, which is associated with accelerated growth rates and an increase in the number of erythroid cells. This is especially true for premature babies; children born with low body weight, children after the second half and second year of life, as well as preschool age and adolescents. Transient iron deficiency can occur with hormonal changes (prepubertal and pubertal hormonal imbalances).

Blood loss is the most common cause of iron deficiency in adults. The loss of each ml of blood (at a hemoglobin level of 150 g / l) results in a loss of about 0.5 mg of iron. Among girls, during the formation of the menstrual cycle in puberty, abundant and prolonged hemorrhagic uterine discharge may occur. Impaired iron absorption can be caused by bowel disease (usually celiac disease), gastric secretion disorders (including due to proton pump inhibitors), and gastrointestinal bypass. Colonization of *Helicobacter pylori* is also often combined with IDA, leading to a deterioration in the absorption of iron from food and an increase in its losses. Anemia in athletes involved in sports requiring endurance is combined with iron deficiency caused by reduced iron intake and impaired absorption as a result of increased hepcidin levels, gastrointestinal bleeding and intense sweating (athlete's anemia). The reasons for insufficient iron intake in children of the first year of life are artificial feeding with unadapted milk formulas, feeding with cow or goat milk, and malnutrition of the mother (wet nurse).

IDA is combined with impaired cognitive development in preschool children, decreased performance, cognitive and behavioral disorders in adults. IDA in pregnant women is combined with an increased risk of having a premature baby or low birth weight, as well as with maternal diseases.

**Risk factors for IDA in children**

| Priority | Secondary |
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| - pregnant women and mothers under 18;<br>- children during the period of intensive growth of growth;<br>- premature infants and children born with a body weight of <2500 g or > 4500 g<br>- children born from multiple pregnancies and with a complicated course of the second half of pregnancy (preeclampsia, placental insufficiency, complications of chronic diseases);<br>- children with insufficient initial level of iron in the body (impaired uteroplacental circulation,<br>fetoplacental bleeding, fetal transfusion syndrome in multiple pregnancies, intrauterine ground, multiple pregnancies, deep and long-term iron deficiency in the body of a pregnant woman, premature or late umbilical cord clipping, intrapartum bleeding due to traumatic obstetric interventions or abnormalities of the placenta and umbilical cord vessels)<br>- conditions in which iron absorption decreases: intestinal pathology;<br>- conditions after surgery on the stomach and intestines, incl. bariatric; peptic ulcer;<br>*H. pylori* infection; Whipple's disease;<br>- diarrhea;<br>- taking medications and medical interventions (long-term use of antacids, H2-receptor blockers, proton pump inhibitors, non-steroidal anti-inflammatory drugs, including acetylsalicylic acid, zinc or magnesium preparations, hemodialysis)<br>- conditions in which blood loss increases: profuse menstrual bleeding, inflammatory processes in the intestines, helminthic invasions, erosive gastritis, paroxysmal nocturnal hemoglobinuria, the use of drugs that increase the risk of gastrointestinal bleeding. | - low socio-economic status;<br>- the postpartum period;<br>- vegetarianism. |

The course of the disease may be asymptomatic. Symptoms usually appear with severe IDA.

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| **Complaints** | • general fatigue<br>• shortness of breath on exertion<br>• dysphagia<br>• general weakness<br>• pallor<br>• asthenization<br>• koilonychia<br>• angular stomatitis<br>• glossitis<br>• membranes of the esophagus and pharynx<br>• tachycardia and heart failure<br>• headache<br>• ringing in the ears<br>• taste disturbance |
| **Anamnesis** | • the presence of melena or unchanged blood in the stool<br>• distorted taste (tendency to consume inedible items)<br>• distorted desire to consume ice |

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| | • the presence of bituria (a condition in which, as a result of eating beets, a pink or red color of urine occurs)<br>• the nature of menstruation (cycle, duration, course) and pregnancy history<br>• gestational age and birth weight<br>• the nature of feeding<br>• preschool children and adolescents - the rate of weight gain and height<br>• drugs that the patient receives (antacids, H2-receptor blockers, proton pump inhibitors, long-term use of non-steroidal anti-inflammatory drugs, long-term use of acetylsalicylic acid, zinc or magnesium preparations)<br>• history of inflammatory bowel disease; celiac disease; previous operations on the stomach and intestines; family history of bleeding disorders and colon cancer; dietary features |
| **Physical examination** | • pallor of the skin and insufficient filling of capillaries<br>• pallor of the conjunctiva<br>• koilonychia<br>• angular stomatitis<br>• atrophic glossitis<br>• tachycardia and other hemodynamic disorders<br>• pay attention to the size of the liver and spleen |
| **Laboratory diagnostics** | • complete blood count: decreased hemoglobin concentration, hypochromia, microcytosis, decreased hematocrit, decreased erythrocyte indices, increased width of red blood cell size distribution.<br>• if changes in the parameters of erythrocytes are detected, their morphology is described on the basis of blood smear microscopy.<br>• blood serum ferritin is the method of choice for confirming IDA (the norm for adults is 15-30 mcg / l for children - 10-12 mcg / l). The diagnosis of IDA cannot be considered confirmed in the absence of data on the level of ferritin.<br>• saturation of transferrin with iron - decrease.<br>• the concentration of erythrocyte protoporphyrin or soluble transferrin receptors - an increase |
| **An example of a diagnosis formulation:** | Iron deficiency anemia due to blood loss from the gastrointestinal tract (?), Severe, complicated by hypoxic myocardial dystrophy |
| **Treatment** | Therapy is carried out, as a rule, with the help of iron preparations for oral administration (preference is given to ferrous iron preparations). Dietary supplements, multivitamin and mineral complexes are not used to treat IDA. Taking oral forms of iron preparations may be accompanied by adverse reactions, mainly from the gastrointestinal tract. To reduce their severity, you should reduce the dose of the drug, for example, take tablets 2-3 times a week. Mild side reactions are also noted in the case of taking drugs at night or with meals.<br>The dose of iron is prescribed in terms of elemental iron:<br>up to 3 years - 3-5 mg / kg / day of elemental iron<br>from 3 to 7 years - 50-70 mg / day of elemental iron<br>over 7 years old - up to 100 mg / day of elemental iron.<br>+<br>- elimination of etiological factors,<br>- rational therapeutic nutrition (for newborns - natural feeding, and in the absence of milk from the mother - adapted milk formulas fortified with iron, timely introduction of complementary foods, meat, offal, buckwheat |

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| | and oatmeal, fruit and vegetable purees, hard cheese, reducing the intake of phytates, phosphates, tannin, calcium, which impair the absorption of iron).<br><br>Patients for whom oral forms of iron preparations are contraindicated, or in the presence of a pronounced adverse reaction to their use, parenteral iron preparations are prescribed.<br>Some patients are shown intravenous administration of iron preparations, while others can independently make a choice in favor of their intramuscular injections.<br>The daily dose of elemental iron for parenteral administration is:<br>for kids<br>1-12 months - up to 25 mg / day<br>1-3 years - 25-40 mg / day<br>over 3 years old - 40-50 mg / day<br>The course dose of elemental iron is calculated by the formula:<br>MT x (78 - 0.35 x Hb), where<br>Body Mass Index, or BMI (kg)<br>Hb - child's hemoglobin (g / l)<br>Heading dose of an iron-containing drug - CDI: IC, where<br>CDI - course dose of iron (mg)<br>IC - iron content (mg) in 1 ml of the preparation<br>Heading number of injections - CDD: DDD, where<br>CDD - course dose of the drug (ml)<br>DDD - daily dose of the drug (ml)<br><br>After the 21st day of treatment with iron preparations, the increase in the hemoglobin level is estimated, which is normally 1 g / l / day of therapy. A positive reaction to drug treatment is observed when the hemoglobin level rises by about 20 g / l from the start of treatment. If the response to treatment is positive, it should be continued; a complete blood count is performed monthly. Therapy continues for 3 months after hemoglobin normalization (6 months in severe anemia). In the event that the response is interpreted as ineffective, it is necessary to persistently look for the reasons for the insufficient response to treatment with oral iron preparations. At the same time, the degree of compliance with the doctor's prescriptions by the patient (compliance) is assessed, a diagnostic search for a hidden source of bleeding is carried out, additional complicating factors are determined, or the option of an incorrect diagnosis is considered. In severe anemia, treatment continues for 6 months. After the end of the course of IDA therapy (3/6 months), in order to replenish the iron depot in the body, iron preparations are prescribed for another 3 months.<br><br>Erythrocyte transfusion remains a method of treating anemia, but it is not a pathogenetically justified tactic of IDA therapy, since it does not replenish depleted iron stores in the body. This method should be prescribed only in cases where it is necessary to provide instant, targeted assistance to patients with high severity anemia that threatens the functioning of target organs (for example, with angina pectoris, heart failure, significant acute bleeding that cannot be stopped). |

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| | Medicines (the order of arrangement does not affect the order of administration):<br><br>• ferrous iron preparations, oral forms: iron sulfate, iron fumarate, iron oxide saccharate;<br>• ferric iron preparations, oral forms: complex of iron (III) hydroxide with polymaltose;<br>• iron preparations, parenteral forms: iron dextrin, iron carbomaltose, iron (III) hydroxide sucrose complex;<br>• iron preparations in combination with folic acid: iron fumarate + folic acid, a complex of iron (III) hydroxide with polymaltose + folic acid, iron sulfate + folic acid<br>• iron preparations in combination with other drugs: iron fumarate + folic acid + cyanocobalamin, iron ammonium quotes + folic acid + cyanocobalamin, iron fumarate + folic acid + cyanocobalamin + ascorbic acid + zinc sulfate, iron sulfate + ascorbic acid, iron sulfate heptahydrate + ascorbic acid, iron gluconate + manganese gluconate + copper gluconate, iron sulfate heptahydrate + D-, L- serine;<br>• donated blood products, which can be produced in blood service establishments: erythrocytes. |
| **Premature babies** | 50-100% of premature babies develop late anemia.<br>For 20-25 days of life with a gestational age of 27-32 weeks, body weight is 800-1600 g, (during a decrease in the concentration of hemoglobin in the blood below 110 g / l, the number of erythrocytes is below 3.1 12 / l, reticulocytes are less than 10%) iron supplements are prescribed (3-5 mg / kg / day)<br>+ Sufficient protein supply (3-3.5 g / kg / day)<br>+ Erythropoietin s / c, 250 units / kg / day three times a week for 2-4 weeks<br>+ Vitamin E (10-20 mg / kg / day)<br>+ Folic acid (1 mg / kg / day).<br><br>Longer use of erythropoietin 5 times a week, with a gradual decrease up to 3 times, is prescribed to children with severe intrauterine or postnatal infection, as well as children with a low reticulocytic response to therapy. |
| **Prophylaxis** | **Antenatal:** for women from the 2nd half of pregnancy, iron supplements or multivitamins fortified with iron.<br>With repeated or multiple pregnancies, iron supplementation is required during the 2nd and 3rd trimester.<br>**Postnatal:** for children at high risk of developing IDA (premature babies, babies born from multiple pregnancies and with a complicated course of the second half of pregnancy, children with food allergies): regular diagnostics of the possible development of IDA and, when it is determined, prophylactic doses of iron preparations are prescribed (0.5-1 mg / kg / day) for 3-6 months |

**The severity of the anemia**

| Age group | Norm | Degree |
|---|---|---|

| | | mild | average | severe |
|---|---|---|---|---|
| Children 6-59 months | ≥ 110 | 100-109 | 70-99 | <70 |
| Children 5-11 years old | ≥ 115 | 110-114 | 80-109 | <80 |
| Children 12-14 years old | ≥ 120 | 110-119 | 80-109 | <80 |
| Non-pregnant women (over 15 years old) | ≥ 120 | 110-119 | 80-109 | <80 |
| Pregnant | ≥ 110 (105) * | 100-109 (105) | 70-99 | <70 |
| Men | ≥ 130 | 110-129 | 80-109 | <80 |

* For the I and III trimester, 110 g / l should be considered the norm, the norm for II trimester is 105 g / l.

The lymphatic system is an important component of the immune system. It includes lymph fLNid, lymph vessels, lymph nodes, spleen, tonsils, adenoids, Peyer 's patches and thymus.
Lymphatic fLNid consists of ultrafiltrate of blood collected in the lymphatic ducts that flow throughout the body. The fLNid flows slowly and is transported from the head and limbs to large vessels, which then flow into the venous system. There are approximately 600 lymph nodes along these channels.
Lymph nodes (LN) consist of follicles and contain a large number of lymphocytes. Lymph is filtered through the sinuses of the lymph nodes, where particles and infectious microorganisms are detected and removed. Due to the infLNence of immune challenges, immunity mediated by antibodies and cells is mediated. As a result of such normal processes, lymph nodes may increase either due to the proliferation of normal cells or due to infiltration by abnormal cells.

**Lymphadenopathy** (LP) is defined as an enlargement of one or more lymph nodes as a result of a normal reactive process or pathological condition.
While (increased) lymph node size is the most common change, an abnormal amount or change in consistency may indicate a pathological condition that requires investigation and possible intervention.

Clinicians are faced with the task of distinguishing "real" enlarged lymph nodes associated with the pathological process, from what is often called "shot down" lymph nodes (" shotty " lymph nodes ).
"Down" lymph nodes are small mobile lymph nodes in the neck that are palpable and are benign changes, usually associated with viral disease.
Removal of lymph nodes to determine the etiology of their increase has been practiced for many years. This procedure is often performed by general pediatric surgeons, as well as surgical specialists such as otolaryngologists.

**The pathophysiology** of LP differs in etiology. In the reactive process, a physiological increase in the number of lymphocytes and macrophages leads to an increase in the size of the node. In addition, with changes associated with pathological processes, the node may increase in size, as bacteria, fungi, viruses or malignant or metastatic cells can fill the node.

**Etiology.**

There are five common etiological categories that lead to enlarged lymph nodes:

1. Immune response to infectious agents (eg bacteria, viruses, fungi). The most common cause of lymphadenopathy is infection. Most of them are secondary to non-specific viral and bacterial infections. The most commonly diagnosed viral infections are caused by cytomegalovirus (CMV) and Ebstein-Barr virus (EBV).
2. Inflammatory cells in infections involving the lymph node.
3. Infiltration by neoplastic cells transferred to the node by lymphatic or circulatory (metastasis).
4. Localized neoplastic proliferation of lymphocytes or macrophages (eg, leukemia, lymphoma). The most important issue in patients with complaints of lymphadenopathy is the detection of the underlying malignancy (if any), with non- Hodgkin 's lymphoma being the most common.
5. Infiltration of macrophages filled with metabolite deposits .

| **Causes of swollen lymph nodes in children** | | |
|---|---|---|
| **The cause of LP** | **Detailing the cause of LP** | **Clinical characteristics** |
| Infections | Bacterial: *Staphylococcus aureus* , β-hemolytic group A streptococcus, causative agents of brucellosis, tularemia, feline scratch disease ( *Bartonella henselae* ) | Recent sore throat or cough, while physical examination includes impetigo, pharyngitis, tonsillitis or acute otitis media. The main affected areas include the submandibular, upper cervical , submandibular , occipital and lower cervical nodal areas.<br>LV, which are acutely infected with bacteria - most often *S. aureus* or group A streptococci are large, warm, tender, have surrounding edema and erythema. The infection may progress to an abscess. Chronically infected nodes, as a rule, have separate borders, adjoin to the underlying tissues and have minimal signs of inflammation.<br>Cat scratch disease is a zoonotic infection that results from animal scratches, most likely cat scratches. Primary inoculation of the skin, eye, or mucosa leaves a small papule that may or may not be apparent on examination. Patients usually have fever, malaise and fatigue. |
| | Viral: SARS, infectious mononucleosis ( Epstein -Barr virus), cytomegalovirus infection | Infections of the upper respiratory tract, small, mild, bilateral, no heat erythema or hot to the touch skin over LV |

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| Mycobacteria | *M. tuberculosis* | Scrofulosis (a known manifestation of extrapulmonary tuberculosis).<br>Enlargement of the cervical nodes, often around the paratracheal or supraclavicular nodes. Nodule enlargement is usually painless; nodes presumably fester and form sinuses. |
| | atypical mycobacteria ( *Mycobacterium scrofulaceum and Mycobacterium avium-intracelLNlar* ) | Relatively sudden onset of adenopathy ; the size gradually increases within 2-3 weeks. Affected nodes usually have erythema and can be painful. Nodes can progress to fLNctuations and eventually merge spontaneously. |
| Protozoan | Toxoplasma, Plasmodium falciparum | Myalgia , fever, fatigue, contact with cats or being in an endemic area |
| Fungal pathogens | pathogens of histoplasmosis, coccidioidomycosis , aspergillosis | Pneumonia, living in endemic areas |
| Autoimmune diseases | JIA, systemic LNpus erythematosus, serum sickness | Systemic LNpus erythematosus (SLE): more involvement of organ systems. |
| Diseases of accumulation | Neiman -Pick disease, Gaucher disease | Hepatosplenomegaly , severe CNS disorders, bone deformities, anemia, thrombocytopenia |
| Drug agents | Anticonvulsants ( phenytoin ), allopurinol , isoniazid | Maculopapular generalized rash, hepatosplenomegaly , anemia, leukopenia, plasmacytosis |
| Vaccination consequences | BCG vaccination, etc. | Instructions for vaccination |
| Malignant tumors | Lymphomas, leukemias, metastases of solid tumors (neuroblastoma, rhabdomyosarcoma , thyroid tumors, nasopharyngeal cancer) | Nodes are usually painless and continue to grow. Lymphadenopathy associated with malignant neoplasms is described as solid or rubbery , discrete, insensitive, and fixed to the skin or underlying structures.<br>There are usually no signs or foci of inflammation. B symptoms, incLNding fever, night sweats, weight loss, and malaise, may be associated. According to recent studies, more than 3 cm of LV are more likely to have a malignant etiology, the increase of which lasts more than 4 weeks, there are lesions of the supraclavicular joint, |

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| | | as well as deviations of laboratory and radiological data. |
| Histiocytosis | Histiocytosis from Langerhans cells , hemophagocytic syndromes, sinus histiocytosis with massive lymphadenopathy ( Rosai - Dorfman disease ) | Langerhans cell histiocytosis (ie histiocytosis X) is a broad-spectrum syndrome in which lymph node enlargement is common. |
| Immunodeficiency states | Chronic granulomatous disease, deficiency of leukocyte adhesion | Recurrent infections, skin abscesses, purulent adenitis |
| Others | Sarcoidosis, Kawasaki disease, Castleman 's disease, Kikuchi disease | In sarcoidosis , supraclavicular nodes and bilateral vorous nodes are involved.<br>In Kawasaki disease, an enlarged node or group of nodes is unilateral, not fLNctuating , and is usually located in the anterior triangle of the neck.<br>Kikuchi lymphadenitis is a benign and rare disease of unknown origin, which includes bilateral enlargement of the cervical lymph nodes that do not respond to antibiotic therapy , systemic symptoms incLNding fever, hepatosplenomegaly , and weight loss. |

Lymphadenopathy is also seen in multisystem inflammatory syndrome in children (MIS-C; also known as pediatric multisystem inflammatory syndrome [PMIS], pediatric inflammatory multisystem syndrome temporarily associated with SARS-CoV-2 [PIMS-TS], pediatric hyperinflammatory syndrome). and Kawasaki-like disease), which probably correlates with COVID-19, although it causes more severe symptoms.

**Examination of children with LP.**

**Collection of anamnesis.**

In most situations, careful medical history, symptom assessment, and physical examination may establish the probable etiology of lymphadenopathy and make any further tests unnecessary.

Assessment of a child with lymphadenopathy may begin with specific aspects of the enlarged lymph node or nodes and then expand to cover various aspects that may have caused it. A special role is played by the assessment of the duration of LP: benign lymphadenopathy disappears within 4-6 weeks, while persistent or progressive lymphadenopathy increases the likelihood of malignancy.

In addition to duration, the clinician should assess the presence of any concomitant symptoms. Other common issues include recent or past illnesses, infections, local injuries or bites. Exposure to medications and especially antibiotics is also important as it can shrink the lymph nodes. If there are no obvious sources of infection, the presence of constitutional symptoms such as fever, weight loss and night sweats are potential signs of malignancy. They are usually called B symptoms. If the patient has recurrent infections, immunodeficiencies such as HIV should be considered.

Information on family and social history is useful for the excLNsion of associated malignancies and will help dispel fears that cancer may develop in the family. A social history can identify potential sources of lymphadenopathy, incLNding recent travel, drinking unauthorized water, contact with animals that may be carriers of unique infections, exposure to tuberculosis (TB), typhoid, and trypanosomiasis. Information about activities such as sexual intercourse is also important. If there is no history, careful examination of symptoms may establish other aspects of causation.

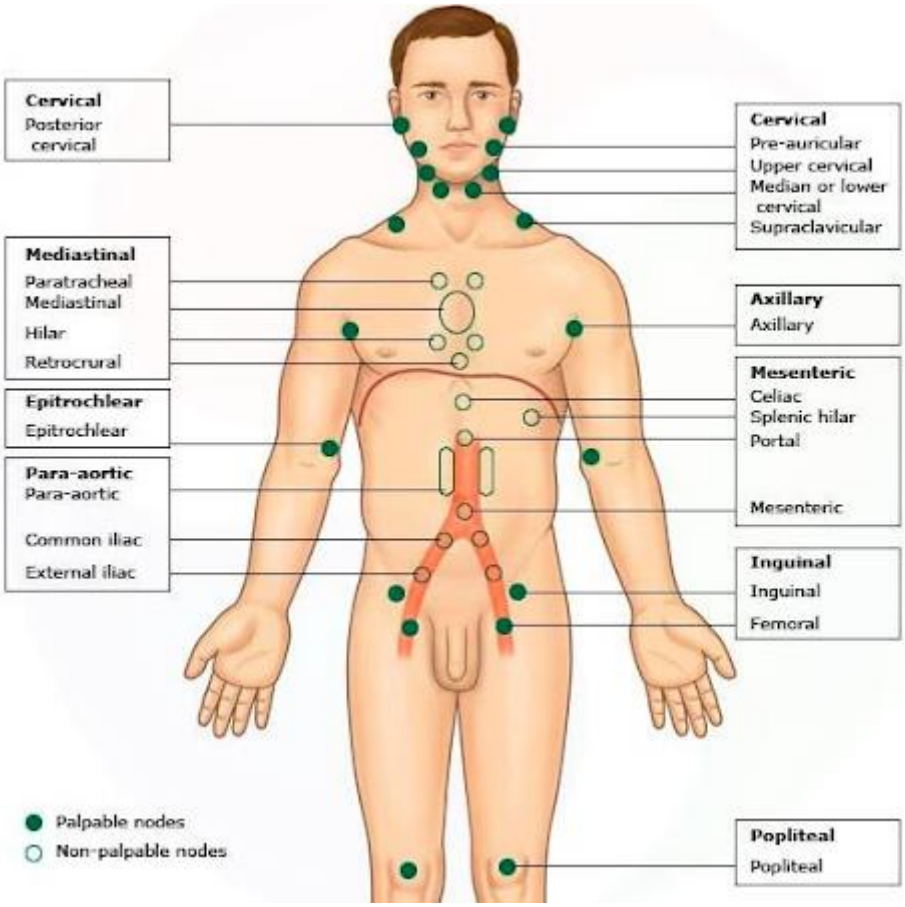
**Physical examination**

**Examination of a child with LP begins as a complete examination and then focuses on the enlarged LN.**

LN can be divided into superficial (peripheral) and deep. Numerous groups of peripheral LN, located in the subcutaneous fat, along the muscles and large vessels, are most available for research in a healthy and sick child.

**Zones (regions, regions) of peripheral LN** :

1. Cervical group LN is formed by submandibular, cervical, supraclavicular, auricular, occipital groups LN;
2. Group of axillary LN;
3. Group of cubital LN;
4. Thoracic group of LN on the edge of the pectoralis major muscle;
5. Group of inguinal LN;
6. Group of popliteal LN.




Palpation of lymph nodes plays a leading role in clinical examination.

**Recommended procedure for examination of LP (top to bottom):**

1. occipital region, then the auricular region and the posterior region;
2. parotid , chin, submandibular, then the cervical region, supraclavicular region;
3. axillary areas, falling down 15-20 cm along the lateral surfaces of the chest;
4. elbow bends;
5. inguinal areas (starting from the pubis, parallel to the inguinal folds in the femoral triangles);
6. popliteal bends.

By the age of 7, five groups of LN are normally palpated : anterior and posterior cervical, submandibular, axillary, and inguinal.
A healthy child older than 7 years may be available for palpation of 3 groups of LN: anterior , posterior and submandibular while maintaining the characteristics of normal LN, and only 1.5-2% remain available for palpation inguinal and axillary LN.

Normal LN soft-elastic consistency, bean -shaped or rounded shape, with a smooth surface, painless, moderately mobile, not fused with the surrounding tissues, the skin over them is not changed. There is no general opinion about their size, but practice shows that much depends on the location of LN - superficial cervical, submandibular, axillary - up to 1.5-2.0 cm, inguinal - up to 3 cm. But there are "dangerous areas" where any palpable LN is suspicious (for example, supraclavicular areas - for malignant lesions, elbow LN - for felinosis , left axillary region in children under 1 year - BCG lymphadenitis).

Unified assessment of the state of LP is performed according to the generally accepted characteristics of LV:

1. Localization of enlarged LN - the name of the group and the nature of the lesion (unilateral or bilateral).
2. Dimensions and shapes of LP - the vaLNe is described only in metric units - centimeters or millimeters (you can not compare LV with fruits, berries, nuts, grains, etc.). LN has two sizes - transverse and longitudinal, hence the shape of LN - rounded, when the longitudinal size is equal to the transverse (one size may be specified - diameter), or oval, when the longitudinal size is greater than transverse (both sizes are specified).
3. Quantity - if in each group no more than 2 LN are palpated, it is accepted to speak about single LN, if 3 LN and more are palpated, then speak about numerous LN.
4. fLNctuations is indicated .
5. Pain - is determined at rest and on palpation.
6. Communication with surrounding tissues - normal LN are not soldered to each other and to surrounding tissues, mobile.
7. The condition of the skin over the LN - possible redness, swelling (symptoms of inflammation), the presence of venous network (indicates additional shunt blood flow, which is characteristic of a specific process), the severity of the capillary network ( accompanying chronic foci of infection), fistula.
8. The presence of compression syndrome - compression of large venous trunks, especially the superior vena cava - the syndrome of the superior vena cava; compression of the trachea and large bronchi - cough and shortness of breath; esophageal compression - dysphagia (violation of the passage of food).

Examination of other lymphoid organs ( lymphopharyngeal ring, liver and spleen, thymus ) is mandatory. The study of these organs gives an idea of the existence of a systemic process.

**Tactics of managing children with LP.**
Considering the increase in LP, it is necessary to answer the question - whether the increase in the lymph node was due to a local problem, or is it a systemic response involving other lymphoid organs?
When detecting a tumor -like formation in typical areas of lymph outflow , it is necessary to answer a few questions:

1. Is it a lymph node at all?
2. If it is LN, what are its characteristics?

3. Is the process local or systemic? Localized lymphadenopathy usually occurs due to abnormalities in the area where the lymph node flows (eg, infection), although it cannot be ruled out as the first sign of premature clinical manifestation in the course of a progressive systemic process. The appearance of generalized lymphadenopathy indicates a systemic disease and orients the clinician more directly to serological and hematological studies.
4. Is the involvement of other lymphoid and parenchymal organs, LNngs, skin.

From regional lymphadenopathy occipital and preauricular localizations are seldom malignant; the former are often associated with infections of the scalp and outer ear, exanthematous diseases and toxoplasmosis, while the latter are associated with infections of the superficial tissue of the orbit, middle ear and parotid glands. Submental lymphadenopathy requires the search for disorders in the front of the mouth and lower lip, submandibular face, nose, maxillary sinus, mucous membranes of the mouth, mouth and submandibular saliva. gland.

Laterocervical lymphadenopathy in the upper neck may be associated with inflammatory or neoplastic diseases of the hypopharynx , larynx, or thyroid, while in the lower neck it may be associated with disorders of the larynx , thyroid, and upper esophagus. Supraclavicular and epitrochlear enlargement should be considered as signs of a potential malignancy.

Enlarged axillary and inguinal lymph nodes, usually of benign etiology. Axillary lymphadenopathy is observed in infections of the upper extremity, chest wall, group tissue and intrathoracic lesions. Inguinal lymphadenopathy is caused by sexually transmitted diseases of the genitals and other infections of the perineum and pelvis.

Enlargement of the popliteal lymph nodes is usually associated with foot and leg infections. Lymphadenopathy of the mediastinum, peritoneum and mesentery are usually not detected during physical examination, but are sometimes suspected on the basis of compression of the surrounding structures.

The minimum examination plan includes a general blood test. Thus, the first step is to assess the clinical signs and results of the general blood test.

**Then you need to decide which step to go next** :

- use tactics of observation ( watch and wait );
- prescribe antibiotic therapy;
- conduct a deeper clinical examination:
- detailed blood test with an assessment of platelet count and urine analysis;
- Ultrasound of lymph nodes of the affected region, lymph nodes of other regions;
- Ultrasound of the abdominal cavity;
- Ultrasound of the thymus ;
- chest radiography in 2 projections (straight and right lateral);
- then according to the indications, depending on the direction of differential diagnosis, computed tomography (CT) or magnetic resonance imaging (MRI), bone marrow puncture, determination of markers of infections, immunological studies, etc.

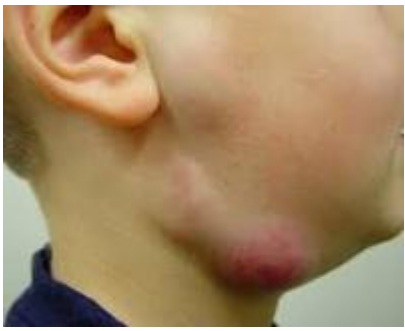
Until an accurate diagnosis is made, patients with swollen lymph node syndrome are not prescribed hormonal drugs, cytostatics , or physiotherapy procedures.

If there is evidence of local inflammatory nature of the increase in LP (acute lymphadenitis), you can start antibacterial therapy.

However, if treatment is ineffective or systemic infection is initially suspected, appropriate studies should be performed (eg, antibodies to pseudotuberculosis, yersiniosis , toxoplasmosis; cytomegalovirus infection, or Epstein - Barr virus ). Adjustment of treatment is carried out at reception of the corresponding results; if necessary, an infectious disease specialist is consulted.

Mantoux test should be included in the examination plan (although interpretation of the results is possible only on the basis of analysis of clinical data, epidemiological history, preliminary results of this test, chest radiography and sometimes histological picture after biopsy). If necessary, a TB doctor is consulted.

If a malignant process is suspected, a bone marrow puncture should be performed; with a negative result of the analysis of the material - an open biopsy of the LP, CT of the affected area, chest and abdomen with intravenous contrast.



Bacterial lymphadenitis is a painful enlargement of the lymph node due to the direct action of an infectious agent on the lymph node tissue (usually reactive), ie the most common cause (eg, periodontitis , submandibular lymphadenitis). Depending on the virulence of the flora and the reactivity of the organism, there are symptoms of varying severity - pain, edema, local and systemic hyperthermia.

Stages of lymphadenitis:

- infiltration stage - at this stage effective antibacterial therapy;
- abscess stage - at this stage surgical treatment is required.

When examining and treating children under 1 year of age with lymphadenitis, it should be remembered that they easily develop adenophlegmon . This is due to the anatomical features of the lymph node - at this age the lymph node capsule is loose, has "pores", so inflammatory exudate easily penetrates into surrounding tissues (subcutaneous fat), which promotes rapid local spread and suppuration.
With a good response to antibacterial therapy, a 7-10-day course is enough, if it is insufficient, it is necessary to change the drug in 3-5 days to an antibiotic of another group. If the lymph node does not shrink after 2 courses of antibacterial therapy, an open biopsy should be considered.

**Laboratory tests**

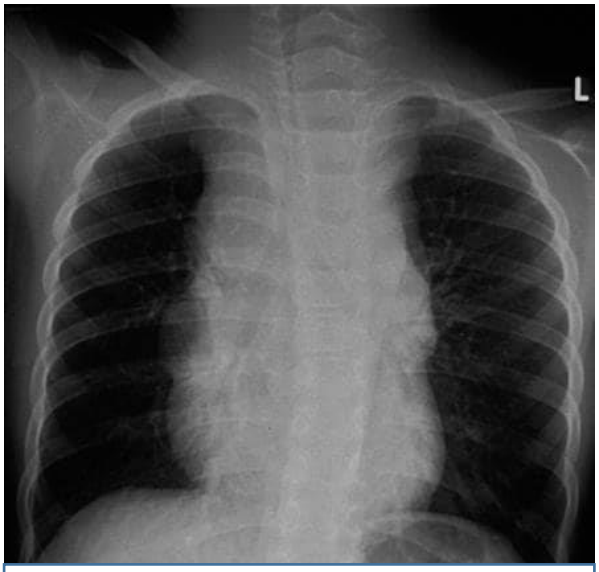
In most patients, only a medical history and physical examination are required to establish a plausible diagnosis. However, if the diagnosis needs to be further clarified, several tests can be performed. Generally, the least invasive test that provides the most information should be performed.
In general, most laboratory indicators of inflammation (eg, erythrocyte sedimentation rate [ESR], C-reactive protein [CRP], glycoproteins , fibrogen levels ) are not conducive to diagnosis, as most results are consistently elevated and do not provide useful guidance on the exact etiology of lymphoma.
A complete blood count (CG) with manual differentiation provides useful information. Leukemia is often accompanied by pancytopenia . An increase in predominantly lymphocytes (> 1 × 109 cells / l) is a practical diagnostic diagnosis of mononucleosis; when the proportion of these cells is less elevated but still predominant, cytomegalovirus (CMV) and toxoplasmosis should be considered. Finding medium and large lymphocytes, which can be classified as transformed or activated, is useful for indicating viral infection.

Serum lactate dehydrogenase (LDH) can be used to determine the rate of cell metabolism in the case of leukemia or lymphoma.

Tuberculin skin test; Epstein-Barr virus [EBV] titers , CMV, feline scratch disease, or toxoplasmosis can be performed to assess specific etiology.

**Imaging studies**

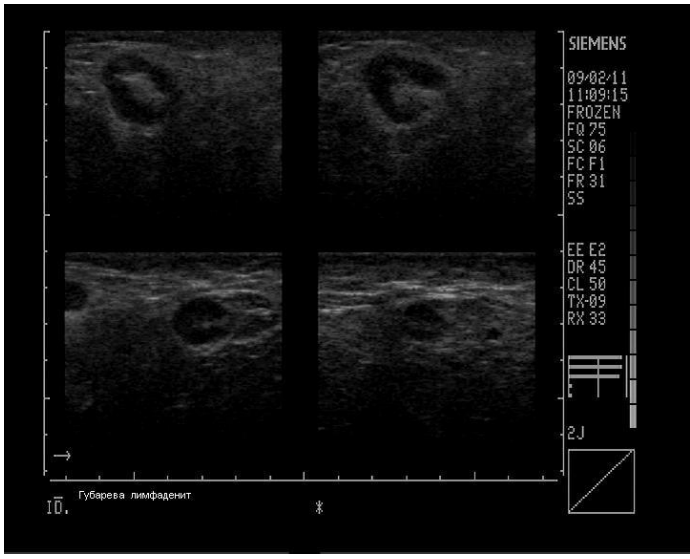
Chest radiography may be useful in identifying potential sources of infection (eg, bacterial pneumonia or tuberculosis) and chest adenopathy in the case of malignancy. Indeed, since numerous reports describe the collapse of the airways under the action of anesthetics in the case of a large formation of the anterior mediastinum, before the introduction of any general anesthesia should consider chest radiography




Narrowed trachea due to the formation of the anterior mediastinum.

**Ultrasound**

Ultrasound (US) can be performed to determine the nature of the site if it is difficult to palpate . In addition, it can be used to distinguish the anomaly from other potential anatomical structures (eg, dermoid cysts, cysts of the thyroid -lingual duct, cysts of the bronchial fissure, inguinal hernias, undescended testicles). Ultrasound can detect connections with adjacent structures and offer information about the contents of an enlarged lymph node or nodes (ie, solid or liquid or gaseous, homogeneous or inhomogeneous).




**Computed tomography**

Computed tomography (CT) is useful for imaging deep lymph nodes, especially in the thoracic and abdominal cavities. This may be the only non-invasive method available to assess these areas for other potential areas of lymphadenopathy and to identify a potential source of malignancy (eg, neuroblastoma, Burkitt 's lymphoma , rhabdomyosarcoma ). In addition, chest CT can add to the information obtained from chest radiography, and can reflect the formation of the anterior mediastinum, as well as the degree of compression of the trachea or bronchial airways (see image below).

**Positron emission tomography on fluorodeoxyglucose**

Positron emission tomography of 18F-fluorodeoxyglucose (18FFDG-PET) has been used in adult patients with lymphoma and later in children to help diagnose and monitor the disease during therapy.

**Fine-needle aspiration biopsy** is widely used in adults and has also been described in children. These benefits include the following:

- It can be performed on an outpatient basis
- It's simple and fast
- Does not require general anesthesia
- Has low levels of complications
- It is cost effective
- Creates minimal scars

The sensitivity and specificity of FNAB in determining the etiology of lymphadenopathy above 90%. Most patients with a benign diagnosis do not undergo a surgical biopsy.

**Excisional biopsy** remains the gold standard of diagnosis. An absolute contraindication for lymph node biopsy is if the etiology is clear and if lymphadenopathy is expected to improve without further treatment. A relative contraindication is recognized if the suspected etiology can be treated as expected (for example, in cases of bacterial infection of the node, when the administration of antibiotics is expected to improve the clinical scenario without the need for biopsy). Another relative contraindication is recognized if the chest radiograph shows the formation of the anterior mediastinum and is considered a high anesthetic risk. In this situation, the risks of anesthesia must be balanced with the need to obtain tissue.

**Drug therapy** is selected based on the most likely etiology if a biopsy has not been performed.

In the case of bacterial infection, the most likely culprits are staphylococci and streptococci; therefore, choose an antibiotic resistant to beta- lactamases . Rifampicin and isoniazid are chosen in patients with tuberculosis .

In cases of non-tuberculous mycobacterial adenitis, most are still in favor of surgical treatment. However, some patients with lymphadenopathy at anatomical sites of concern may benefit from drugs such as clarithromycin, azithromycin, rifampicin, rifabutin, or ethambutol.

Most patients with viral etiology of lymphadenopathy can be treated as expected.

Patients with some more obscure diagnoses, such as Kawasaki disease, systemic lupus erythematosus (SLE), and Langerhans cell histiocytosis , may need immunosuppressants .

**Surgical therapy**

Enlargement of the cervical lymph node to a diameter of 1 cm or more is considered abnormal and requires biopsy if the diagnosis is otherwise uncertain. Lymph node biopsy may include one of two methods. The most commonly used method is surgical biopsy, in which either part of the node or the entire node is excised.

Prior to the procedure, the patient and his family are instructed on the relevant steps and risks and benefits; the form of the consent is received. Before removing the node, the surgeon should discuss the case with a pathologist so that appropriate tests can be performed immediately after receiving the sample. The procedure is performed either in the operating room with general anesthesia, or in a small procedure room with conscious sedation .

Procedural details

An incision is made in the skin above the enlarged node, and the surrounding tissue is carefully cut off from the node. Care must be taken to avoid the surrounding nerves, especially in the areas around the neck. To help remove the knot, the seam on the non-cutting needle can be applied

through the center of the knot to allow it to be pulled out so that it can be pulled into view (see image below). This measure also minimizes the artifact of crushing that can occur as a result of excessive lymph node treatment.
The node must then be sent fresh to a pathologist for treatment (see image below). This will allow you to perform all possible tests; lymph node fixation precludes some important tests (eg, flow cytology, cytogenetics). Usually one large node or a group of smaller nodes is sent to the pathologist for diagnosis.

**Equipment:** a laptop, an Infant auscultation simulator auscultation trainer and smartscope LF01201 W44743 ( Inv . No. 101475072), multi-purpose pediatric care simulator (S157) W45178 ( Inv . No. 101475075), multimedia presentation on the topic of practical training, educational videos

**Plan:**

1. Organizational measures ( greetings , inspection those present , message of the topic, purpose of the lesson , motivation acquirers higher education of studying the topic).
2. Control of the reference level knowledge ( written work, written testing , frontal poll etc. ) is not foreseen.
3. Formation professional ability , skills ( mastery skills , conducting curation , definition schemes treatment , laboratory research _ etc. ):
   a) Oral survey, conversation, collective discussion and discussion on the subject of the lesson;
   b ) List of educational practical tasks, which it is necessary to perform during practical classes: on the basis of the proposed clinical situations, perform the main steps to ensure the effective management of a sick child
   c) Solving a clinical task, for example:

**Task 1.**
A 14-year-old child was admitted with complaints of enlarged cervical lymph nodes.
From the anamnesis , it is known that a month ago, the child noticed an increase in cervical lymph nodes. A diagnosis was made: cervical lymphadenitis. The patient received antibacterial therapy without effect, then received a course of UHF therapy on the area of enlarged lymph nodes, after which their increase was noted, the child's well-being worsened.
He periodically has a high fever up to 38.5°C, accompanied by chills, profuse night sweats bother him, he has a cough, pain behind the sternum, the child has lost weight.
During the examination, the change in the configuration of the neck attracts attention. A conglomerate of lymph nodes is palpated on the left neck, the total size of 5.0x7.0 cm, inside it are palpated individual lymph nodes of 1.0-1.5 cm in size, not fused between themselves and the surrounding tissue, painless on palpation. Other groups of peripheral lymph nodes were not enlarged. In the lungs, the breath is vesicular, there are no wheezing. Tones of the heart are somewhat muffled, rhythmic. The borders of the heart are expanded across. Abdomen is soft, painless. The liver and spleen are not palpable .
Clinical blood analysis: Hb – 132 g/l, erythritol . – 4.5x10 $^{12}$ /l, CP – 0.88, thrombus. - 495.0 x10 $^{9}$ / l, leuk . - 8.4 x10 $^{9}$ / l, p / i - 4%, s / i - 72%, e. - 1%, m. - 3%, l. -20%, ESR - 37 mm/h.
Biopsy of the cervical lymph node: Berezovsky- Sternberg cells were detected .
X-ray of chest organs - expansion of the median shadow due to enlarged intrathoracic lymph nodes.
Ultrasound of abdominal organs: liver, spleen, pancreas of homogeneous structure, enlarged lymph nodes in the abdominal cavity were not detected.
Myelogram – bone marrow cellular, blasts 0.2%, neutrophil sprout 65%, erythroid sprout 21%, lymphocytes 8%, eosinophils 6%, megakaryocytes 1 per 200 myelokaryocytes .

| **Question :** | **Answers :** |
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| 1. Make a preliminary diagnosis .<br>2. Which ones research must be conducted for clarification diagnosis ?<br>3. Whether were errors in tactics patient management , if so,<br>which ones ? | 1. Hodgkin's disease ( lymphogranulomatosis ), stage II B.<br>2. Myelogram<br>3. Yes. The main mistake was the appointment of physiotherapy after ineffective antibacterial therapy until the final diagnosis was determined, which led to the progression of the disease and deterioration of the patient's condition. It was necessary to adhere to the following tactics after the lack of effect from antibacterial therapy:<br>• conduct a deeper clinical examination<br>• extensive blood analysis and urine analysis;<br>• Ultrasound of lymph nodes in the affected region, lymph nodes in other regions;<br>• Ultrasound of the abdominal cavity;<br>• Ultrasound of the thymus ;<br>• X-ray of chest organs in 2 projections (straight and right lateral);<br>further according to indications, depending on the direction of differential diagnosis, computer (CT) or magnetic resonance imaging (MRI), bone marrow puncture, identification of infection markers, immunological studies, etc. |
| **Task 2**<br>Mother of a 2- year-old daughter di actions turned to the family doctor with complaints about the child's fever , sore throat , the girl can drink and swallow . Child is sick 4 days. Court, with words mother , there is no and never was . Consciousness is not impaired . No cough and runny nose. During the examination : the child is restless , the skin is pink , rashes on the skin no , body and limbs hot to the touch . Pharynx Hyperemic , expressed injection vessels _ Tonsils are larger than single tonsils _ _ white plaques . It is noted moderate magnification and anterior cervical tenderness lymph nodes . Above the lungs vesicular breathing , wheezing there is no The breathing rate is 50 in 1 minute . Body temperature is 38.5°C. | |
| **Question :**<br>1. Assess the child's condition according to the IVHDV algorithm<br>2. Does the child have general signs of danger?<br>3. What signs allow you to determine the condition of the child?<br>3 . Prescribe treatment . | **Answers :**<br>1. Acute tonsillopharyngitis , probably bacterial , yellow<br>2. No, there are no general signs of danger.<br>3. Plaque in the throat , soreness anterior cervical lymphatic nodes , temperature bodies child<br>37.5°C or higher , absence of cough, runny nose .<br>4. Treatment:<br>• Smear for diphtheria from the throat and nose<br>• If diphtheria is suspected URGENTLY send to a hospital _ _<br>• And moxicillin orally for 10 days<br>• A daily dose of paracetamol or _ ibuprofen for pain<br>• Sooth your throat with safe tool<br>• Inform mother about states that _ in need immediate reapplication _<br>• Second review in 2 days |
| **Task 3** | |

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| To the reception department hospitals parents with a 3- year-old boy contacted . Complaints about promotions temperature body up to 38.5°C, weakness , absence appetite , insignificant pain in the throat when swallowing . Child sick for 3 days. On examination : a child frail , pale . rash there is no Body temperature up to 38.5°C, cough and runny nose are absent . Slyzova the membrane of the oropharynx is hyperemic from cyanotic shade _ On the tonsils dense filmy grayish plaque , hard is removed Enlarged submandibular lymphatic nodes _ Tachycardia . Others no changes were detected . | |
| **Question :**<br>1. Formulate the previous one diagnosis .<br>2. Make a provisioning algorithm help _ | **Answers :**<br>1. Diphtheria tonsils , medium-severe form<br>2. Take a swab from the throat and nose if diphtheria is suspected ;<br>Administer PDS intramuscularly in a dose of 50,000 IU;<br>Give ceftriaxone at a dose of 100 mg/kg per day intravenously ;<br>Give one dose of Ibuprofen 10 mg/kg;<br>Administer 5% solution intravenously glucose in a dose of 10 ml/kg/day;<br>Administer 0.9% solution intravenously sodium chloride in a dose of 20 ml/kg/day;<br>Urgently direct a child in boxing department infectious hospitals ;<br>Assign rinsing and irrigation of the oropharynx with a disinfectant solution furacilin ;<br>Administer prednisone intravenously at a dose of 2 mg/kg. |

Recommendations (instructions) for the performance of tasks (professional algorithms, orientation maps for the formation of practical skills and abilities, etc.):

a) Questions for self-control:

1. Causes, risk factors of anemia, and the main links of pathogenesis (deficiency, posthemorrhagic , hemolytic, due to a violation of hematopoiesis ).
2. Classify anemia , using erythrocyte indices and indicators reticulocytes
3. Know the main ones biochemical Indicators blood in various types of anemia
4. Analyze typical clinical picture of anemias ( deficiency , posthemorrhagic , hemolytic , due to violation hematopoiesis )
5. Determine features anemia and put previous clinical diagnosis .
6. Make an examination plan and analyze data laboratory and instrumental examinations during a typical course anemia in children .
7. Demonstrate mastering the principles of treatment and prevention anemia in children .
8. Conduct differential diagnosis anemia
9. Know preventive measures regarding development anemia
10. Prognosis of life with anemia in children .

b) Orientation map for independent work with literature

| No | Main tasks | Instructions | Answers |
|---|---|---|---|
| 1 | 2 | 3 | 4 |
| 1. | Familiarize yourself with the literature and the purpose of the lesson | Get acquainted with modern concepts of etiopathogenesis , classification , clinical course and additional methods of diagnosis of diseases | Know the development factors, classification , clinical picture of disease manifestations, hematological , immunological , |

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| | | accompanied by pallor in children | radiological and functional signs of diseases and conditions. |
| 2. | Epidemiology | To know the prevalence of diseases accompanied by pallor among children. | Know: the prevalence of the most frequent diseases and pathological conditions in the children's population . |
| 3. | Etiopathogenesis | To know the causes and mechanism of diseases that are accompanied by pallor in children | Know that these conditions can be caused by the action of medical factors, pathogens of infectious diseases |
| 4. | Clinic | Describe the clinical picture of diseases accompanied by pallor in children | Remember the leading clinical symptoms of ′the main conditions in children with pallor |
| 5. | Diagnostics | Know the schemes of diagnosis and treatment of diseases accompanied by pallor in children | Use schemes for diagnosis and treatment of diseases accompanied by pallor in children |

in) In terms of results work , including preparation : the written work must be written or printed, have a neat appearance, the date, topic, task number, name of the performer must be indicated at the beginning, the text must be legible, structured ;

4. Beating results : announcement of grades based on the results of the lesson. The grade for one practical lesson is the arithmetic average of all components and can only have a whole value (5, 4, 3, 2), which is rounded according to the statistical method.
5. List of recommended literature :

- *main:*


1. Nelson Textbook of Pediatrics, 2-Volume Set, 20th Edition, 2016 by Robert M. Kliegman , Bonita MD Stanton, Joseph St. Geme and Nina F Schor, 5315 p.
2. Foundations pediatrics according to Nelson: in 2 volumes. Volume 1 / Karen J. Marcdante, Robert M. Kligman ; translation of the 8th Eng. publication _ Scientific translation editors V.S. Berezenko , T.V. Rest Kyiv : VSV "Medicine", 2019. T1-378 p., T2- 426 p.
3. Pediatrics : textbook for students. higher teach _ institutions Level IV Accreditation / under the editorship Prof. O.V. Heavy .- Type. 5th correction . and added.- _ Vinnytsia : Nova Kniga, 2018. -1152 p.: ill .
4. Pediatrics : assistant professor . for students higher honey _ teach _ app . IV level accreditation / O. V. Tyazhka, N. G. Horovenko, S. O. Kramarev [and others ]; under the editorship O. V. Tyazhkoi . - 4th ed., ed . and added _ – Vinnytsia : Nova Kniga, 2016. – 1151 p.


- *additional:*


1. Order of the Ministry of Health of Ukraine dated November 2, 2015 No. 709 Unified clinical protocol of primary and secondary medical care "Iron deficiency anemia".
https :// dec . gov . ua / wpcontent / uploads / images / dodatki /2015_709_ ZDA /2015_709_ YKPMD _ ZDA . pdf
2. World Health Organization / Iron deficiency anemia: Assessment, prevention and control. — Geneva: WHO, 2001. — 114 p.
3. Bordiy T. Differential diagnosis of anemia in children (part 2) / T. Bordiy , S. Skotar , V. Popovych, Yu. Ponomarenko // With care for the child . — 2013. — No. 4(40). — P. 11–15.
4. "Diagnosis and management of iron deficiency anemia : a clinical update" (2010),

5. "National Iron Plus Initiative Guidelines for Control of IDA" (2013).

- *electronic information resources* :

1. http://moz.gov.ua – Ministry of Health of Ukraine
2. www.who.int - World Health Organization