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SELECTED ISSUES OF CHILDHOOD PATHOLOGY

with tests and clinical tasks

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CONTENT

Acute stenosing laryngotracheitis in children

Epidemiology. Croup (laryngotracheobronchitis) is a very common cause of obstruction of the upper respiratory tract in children - the annual incidence of children under the age of six is 6%. Croup is most common from late fall to early winter, but can occur year-round. Croup is usually caused by the parainfluenza virus, but other viruses can cause it. Croup is most common in children between the ages of six months and three years, but can also occur in children between the ages of three months and 15 years. Boys get croup more often than girls. Croup is reported to be rare in adults. More than 60% of children diagnosed with croup have mild symptoms, about 4% are hospitalized, and approximately one in 4,500 children are intubated.

Etiology. Croup is caused by viral infections of the respiratory tract, most often by parainfluenza virus type 1, 3. Other possible pathogens are influenza virus type A and B, adenovirus, respiratory syncytial virus (RSV), and metapneumovirus. These infections cause generalized inflammation of the respiratory tract and swelling of the mucous membrane of the upper respiratory tract. The lower pharynx narrows, causing upper airway obstruction and symptoms usually associated with croup.

Pathogenesis. Pathogenesis consists of the joint action of inflammation, edema, spasm, necrosis of the epithelium and its exfoliation, and the appearance of inflammatory exudate in the respiratory tract. Children compensate for the narrowing of the upper respiratory tract by breathing more quickly and deeply. As the narrowing progresses, the child increases the effort to breathe, which becomes unproductive. Airflow through the upper airway becomes turbulent (stridor) and the compliant chest begins to “inhale” during inspiration. This leads to inefficient asynchronous movement of the thoracic and abdominal cavities, the child is

exhausted. In this one moment, hypoxia and hypercapnia increase, and respiratory failure and respiratory arrest develop rapidly.

Clinical manifestations: complaints of hoarseness of voice, dry barking cough, shortness of breath, excitement. According to the nature of changes in the larynx, the following forms of acute laryngotracheitis are distinguished: catarrhal - edematous, edematous - infiltrative, fibrinous - purulent, ulcerative - necrotic, hemorrhagic. The last two forms are rare. Symptoms are usually much worse at night and lessen during the day. In most children with croup, obstructive manifestations disappear within 48 hours, although a small percentage of children have symptoms for five to six days. Respiratory failure usually occurs within a few hours. Signs of respiratory failure and the threat of respiratory arrest include a decrease in respiratory movements, lethargy, paleness of the skin. There are 4 stages of respiratory failure in acute stenosing laryngotracheitis: Stage I - compensation, II - subcompensation, III - decompensation, IV - asphyxiation.

The disease always begins with the first respiratory stage insufficiency, and therefore, at the first signs of respiratory failure, the child should be hospitalized.

I. Difficulty breathing and inspiratory suffocation, cough appears sporadically. The child is excited, capricious. With restless behavior, physical exertion, the supraclavicular and subclavian fossae are drawn in, and inspiratory dyspnea increases. Direct laryngoscopy reveals inflammation of the mucous membrane of the larynx.

II. Subcompensated stenosis: as a rule, it develops on the second day of the disease and is characterized by a rough, barking cough, sharply expressed inspiratory dyspnea, pulling in the auxiliary muscles. Stridorous breathing is observed. At this stage, the child needs to be transferred to a specialized otolaryngology or intensive care unit. The mucous membrane of the vocal cords is moderately hyperemic with the appearance of roller-like infiltrates.

III. Decompensated stenosis: severe general condition of the patient, severe inspiratory dyspnea, sharp depression of the sternum, the child is excited. The skin is pale, covered with cold, sticky sweat. In the lungs, breathing is extremely weakened; pronounced cyanosis of the fingertips, lips. Tachycardia. Acidosis is pronounced in the body.

IV. Asphyxia: extremely serious condition of the patient, the skin is cyanotic. Breathing is shallow, silent, heart sounds are dull, bradycardia, pulse is filiform.

Differential diagnosis: performed with epiglottitis, bacterial tracheitis, acute obstructive bronchitis, bronchiolitis, pneumonia, foreign body of the respiratory tract, angioneurotic edema or anaphylaxis.

Treatment: The basis of treatment of stenotic laryngotracheitis is pathogenetic emergency therapy, which includes the following measures:

1. Give the child a raised position in bed.

2. Inhalations through a nebulizer: with nebules of pulmicort (budesonide) - today this is the main pathogenetic treatment of laryngeal stenosis.

3. Plenty of warm drinks.

4. Prescribe distracting procedures: hot gentle or hand baths; mustard seeds (with their tolerance) on the area of the larynx, trachea, calf muscles (at the pre-hospital stage).

Steam oxygen inhalations in the following sequence:

a) with antispasmodics and vasoconstrictor drugs (0.1% adrenaline solution or 5% ephedrine solution);

b) with alkaline solutions (2% solution of sodium bicarbonate, slightly carbonated mineral water of the "Borjomi" type);

c) with expectorant and anti-inflammatory herbs (mother-and-stepmother, thyme, bogulnik, blush, etc.);

In case of stenosis of the 1st degree, inhalations should be repeated every 1.5-2 hours, in cases of the 2nd - 3rd degree - to be carried out constantly.

5. Enter a 2.4% solution of euphyllin at a dose of 2-4 mg/kg IV slowly or IV drip.

6. Enter a 2.5% solution of pipolfen at a dose of 0.1 ml/year or a 0.5% solution of ceduxen at a dose of 0.3-0.5 mg/kg intravenously or intravenously.

7. At II - IV degree of stenosis, inject a 3% solution of prednisone in a dose of 3-5 - 100 mg/kg intravenously.

8. With II - IV degrees of stenosis, intravenously inject 10% glucose solution, 0.9% sodium chloride solution at the rate of 50-60 ml/kg/day in a ratio of 3:1.

9. If there is no effect, perform a direct laryngoscopy followed by the introduction of hydrocortisone into the parapharyngeal tissue in a dose of 12.5-25 mg.

10. If the symptoms of subcompensated stenosis persist for 18-24 hours or if the stenosis is decompensated, perform prolonged nasotracheal intubation with a thermoplastic tube for 3-4 days.

11. Provide mechanical ventilation in the absence of effect from the measures listed above.

TESTS

1. Which of the listed viruses is not a respiratory virus?

A. Adenovirus

B. Parainfluenza

C. Rhinovirus

D. Respiratory syncytial virus

E. Cytomegalovirus

2. Respiratory syncytial virus (RS) infection is accompanied by pronounced:

A. Respiratory failure

B. Cardiovascular failure

C. Intoxication

D. Encephalopathy

E. Kidney failure

3. The following syndrome is typical for adenovirus infection:

A. Pharyngo-conjunctival fever

B. Kidney failure

C. Cardiovascular failure

D. Encephalopathies

E. Liver failure

4. False croup in a child is formed as a result of:

A. Spasm of blood vessels and swelling of the subligamentous space

B. Spasm of small vessels

C. Swelling of the subligamentous space

D. Plaque in the larynx

E. Obstructed nasal breathing

5. Respiratory syncytial virus affects young children with manifestations:

A. Bronchiolitis

B. Acute obstructive bronchitis

C. Pneumonia

D. Acute simple bronchitis

E. Adenoiditis

6. The main pathogenetic means for the treatment of laryngeal stenosis is the appointment of:

A. Antipyretics (ibuprofen, paracetamol)

B. Antispasmodics (no-shpa, papaverine)

C. Inhaled glucocorticoids (pulmicort)

D. Abundant drinking

E. Antiviral treatment (interferons, oseltamivir)

Answers: 1. – E, 2. – A, 3. – A, 4. – A, 5. – A, 6. – C.

Clinical task 1. Child 10 months old, second day of illness, T - 38.5◦C, "barking" cough, hoarseness of voice, in dynamics on the third day, cough is less sonorous, voice is almost aphonic. Breathing is noisy, difficult inhalation, with a frequency of 65 breaths per minute, retracting the pliable parts of the chest. Heart sounds are muffled, tachycardia - 106 bpm, perioral cyanosis is observed. Other organs

without pathological changes. What is your previous diagnosis? Specify the probable etiological factor.

Answer standard. Acute stenosing subcompensated laryngotachitis. The child has an acute respiratory infection caused by respiratory viruses (parainfluenza).

Clinical task 2. A 3-year-old child is ill for the second day with pronounced intoxication and catarrhal syndromes. On the third day, signs of laryngotracheitis appeared with manifestations of inspiratory suffocation with a frequency of 58 breaths/min., tachycardia of 110 beats/min. Specify the main pathogenetic treatment of stenotic laryngotracheitis, the ways of drug administration.

Answer standard. The main pathogenetic method of treatment of stenosing laryngotracheitis is the appointment of glucocorticoid inhalations through a nebulizer - budesonide (Pulmicorta) at 0.25 - 1.0 mg/day and intravenous glucocorticoids at a dose of 3 - 5 - 10 mg/kg per day.

Bronchitis in children

Etiology. In the etiology of bronchitis in children, viral factors are observed in 90% (influenza, parainfluenza, adeno-viral, rhino-viral, respiratory-syncytial infection, metapneumoviruses, bocaviruses and other respiratory-viral infections), bacterial agents (mycoplasma, chlamydial and other atypical flora). Bronchitis can be caused by physical, chemical and other factors, including allergic ones. The frequency of bronchitis with one or another infection depends on the age of the child. Thus, respiratory syncytial (RS) virus, parainfluenza virus (type 3) cause damage to the lower respiratory tract in children under one year, and in adults - damage to the upper respiratory tract. Influenza A and B viruses affect children of all ages, meeting mainly in the cold period and during epidemic outbreaks. Rhino - viral, enteroviral, adenoviral infections are more often found in the cold period of the year and in the form of sporadic cases. Mycoplasma bronchitis is rarely observed in children under one year of age, in addition, they are characterized by seasonality and the possibility of defeating organized groups.

Among the bacterial factors of secondary bronchitis, Haemophilus bacillus, pneumococcus, staphylococcus should be noted. Bronchitis often accompanies diphtheria, whooping cough, measles.

Classification. Groups of bronchitis – primary and secondary, forms – acute, recurrent, chronic, severety – mild, moderate, severe. Subdivision of bronchitis:

acute bronchitis (common), acute obstructive bronchitis, acute bronchiolitis, recurrent bronchitis, chronic bronchitis, stages of process – acute attack, remission.

Clinical manifestations

Acute simple bronchitis: is a form of bronchial damage in which the signs of airway obstruction are not clinically expressed.

Acute obstructive bronchitis: is a form of bronchial damage in which clinical signs of obstruction due to bronchospasm, swelling of the mucous membrane and/or hypersecretion of mucus are observed.

Acute bronchiolitis: - this is a form of damage to small bronchi as a variant of obstructive bronchitis, mainly in children under 2 years of age with severe respiratory failure.

Recurrent bronchitis:

- this is bronchitis that recurs at least 3 times a year, without clinical signs of obstruction and has a tendency to be prolonged, more than 2 weeks, run.

Chronic bronchitis is a condition accompanied by a productive cough for at least 3 months for two years in the absence of other diseases that can cause these symptoms.

Clinical manifestations

Acute simple bronchitis - clinical manifestations:

1) Cough is dry, becomes wet after a few days, can last 7-10 days;

2) Intoxication is moderate (except for flu, adenovirus infection);

3) Absent palpatory and percussive pathological changes;

4) Auscultatively: hard breathing, rales on both sides are dry, then moist (medium, large-bubble rales), which have a spilled, diffuse character.

Acute obstructive bronchitis - clinical manifestations:

1) A whistling and prolonged exhalation that can be heard at a distance - in English-language literature, wheezing;

2) Participation of auxiliary muscles in the act of breathing; suffocation is moderate, cyanosis is absent;

3) The cough is dry, paroxysmal, long-lasting, becomes wet on the 5-7th day.

4) Percussion: box tone of lung sound;

5) Auscultation: exhalation is prolonged, a large number of dry, and then wet, medium- and large-bubble rales.

Acute bronchiolitis - clinical manifestations:

1) Catarrhal syndrome is expressed moderately;

2) The temperature is normal or subfebrile, sometimes up to 38°C.

3) Marked respiratory insufficiency: expiratory dyspnea, the wings of the nose, auxiliary muscles take part in the act of breathing, there is retraction of the intercostal spaces, cyanosis of the nasolabial triangle is observed.

4) There are signs of reduced bronchial patency: horizontal location of the ribs, low position of the diaphragm, expansion in the anteroposterior dimension of the chest;

5) Box tone with percussion;

6) Auscultatively: diffuse, moist, voiceless, small bubbling rales, there may be dry rales over the entire surface of the lungs;

7) Tachycardia is sharply expressed, tones are weakened.

Recurrent bronchitis:

- this is bronchitis that recurs at least 3 times a year, without clinical signs of obstruction and has a tendency to be prolonged, more than 2 weeks.

The clinical picture of disease relapse consists of symptoms of acute bronchitis. Relapse is often delayed for 3-4 weeks or more. The clinical signs of an acute respiratory disease (rhinitis, hyperemia of the throat, etc.) precede the relapse of the disease and are mostly preceded by many earlier clinical signs of bronchitis. The seasonality of relapses coincides with the seasonality of acute respiratory infections (November, December, January, February).

Recurrence of the disease begins with moderate fever. The temperature is very rarely high. Bronchitis relapses are possible even without a fever. Prolonged low-grade fever is possible. The general condition of the child is not disturbed.

1) The leading symptom is a cough, dry at first, moist for most of the exacerbation period, with mucous or mucous-purulent sputum

2) Percussion sound over the lungs is unchanged or with a slight box tone.

3) The auscultatory picture is also diverse. Often, against the background of hard breathing, coarse dry and medium- and coarse-bubble wet rales are heard on both sides. The auscultatory picture in the lungs is variable.

In the inter-recurrence period, a readiness to cough can be observed: the appearance of a cough during cooling, during physical exertion, during nervous and mental stress, which is a sign of increased bronchial reactivity.

Chronic bronchitis - clinical manifestations:

It consists of the following main symptoms - cough, sputum secretion, feeling of respiratory discomfort, shortness of breath. Symptoms such as weakness, general malaise, paleness of the skin, sweating, increased body temperature, decreased work capacity, and school performance are important for assessing the severity of the course and prognosis of the disease.

The most typical representative of the clinic of chronic bronchitis is cough. It can be unproductive, dry, but more often it is accompanied by the release of a small amount of sputum, which can be watery, mucous, mucous - purulent, purulent, sometimes with blood streaks. Intense coughing can contribute to the fact that sputum flows into less infected areas of the bronchi, supporting the inflammatory process. With viscous sputum sticking to the wall of the bronchi, a long-lasting cough is observed, which is extremely unpleasant for the child and sharply worsens the patient's quality of life.

In the early stages of the disease, a cough bothers the child in the morning, after waking up in the morning, during morning exercises. It occurs sporadically during the day, under the influence of increased breathing and physical exertion. Cough often worsens in the cold and wet season, especially when breathing through the nose is disturbed. Sometimes the cough intensifies when the child is in a horizontal position, in bed. This cough phenomenon is caused by a cutaneous-visceral reflex from a cold bed and is provoked by a change in the flow of sputum from the small bronchi due to their short-term spastic state. Cough in children manifests itself mainly only in the acute phase of the disease.

Shortness of breath appears in a sick child in the later stages of the disease, when chronic bronchitis lasts for years. In the earlier stages of the disease, the phenomenon of respiratory discomfort can be observed - a kind of dissatisfaction with breathing, which can be a variant of bronchospastic syndrome.

Physical data accompanying primary chronic bronchitis in the period of exacerbation do not differ from those in acute.

Treatment

Treatment of acute simple bronchitis:

1. Treatment is possible at home, hospitalization - for suspected complications.
2. The regime against the background of acute manifestations of the disease is semi-bed rest, in the future - at home.
3. Drinking regime with a sufficient volume of liquid, sometimes up to 1.5 - age-old daily needs of the child.
4. The diet is complete, according to the child's age, enriched with vitamins, high-calorie. Table No. 5 is used as a basis in stationary.
5. Etiological therapy is prescribed taking into account the fact that in 90-92% of cases the cause of acute bronchitis is a viral infection, therefore specific antiviral therapy should be used in modern etiotropic treatment.

A. Antiviral drugs are effective in the first 2-3 days of the disease. Among the wide variety of antiviral drugs, neuraminidase inhibitors (oseltamivir, zanamivir), interferon α2b (laferon, laferobion, nasoferon, viferon), interferon inducers (amixin, amizone, cycloferon), other drugs with immunomodulatory and antiviral effects (groprinosin, arbidol) etc.

Example:

• interferon intranasally 5 drops 4-6 times a day or in aerosols using an inhaler;
• ribavirin 10 mg/kg per day in 3-4 doses for 3-5 days;
• intramuscular (in/m) anti-influenza immunoglobulin 0.1-0.2 ml/kg 1-2 times with an interval of 12-24 hours;

• groprinosin at the rate of 50 mg per 1 kg of body weight per day in 3-4 doses, with a severe course, the dose is increased to 100 mg per 1 kg of body weight in 4-6 doses for 5 days, etc.

6. Aerosol inhalations - alkaline, alkaline - saline.

7. Expectorant and mucolytic drugs of synthetic and plant origin (ambroxol, acetylcysteine, bromhexine, carbocysteine, guaifenesin, althea root, plantain drugs, their combinations, etc.). The drugs are used enterally and by inhalation, less often parenterally. Ambroxol syrup 15 mg/5 ml is prescribed for children aged:

• from 12 years old – 10 ml 3 times a day;

• 6-12 years – 15 mg (5 ml) 2-3 times;

• 2-6 years – 7.5 mg (2.5 ml) 3 times;

• 1-2 years – 7.5 mg (2.5 ml) 2 times a day.

8. Vibration massage together with postural drainage is effective for productive cough.

9. Physiotherapy procedures: ultrahigh-frequency therapy, microwave therapy, diadynamic and sinusoidal currents, various options of electrophoresis (with CI, CaCl2, MgSO4).

10. Antitussive drugs are prescribed only for intrusive, unproductive, dry cough - for the purpose of suppressing cough, also glaucin, prenoxdiazine, butamirate, dextromethorphan are used.

Treatment of acute obstructive bronchitis:

1. Hospitalization in moderate and severe cases.

2. The diet is hypoallergenic, complete, according to the age of the child.

3. Emergency aid for a mild form of acute obstructive bronchitis - 1-2 doses of one of the bronchospasmolytic drugs using an aerosol inhaler through a suitable spacer with a face mask (for children under 4 years old) or with a mouthpiece (from 4 years old): β2-agonist (salbutamol, fenoterol), used mainly in older children and when other inhaled bronchodilators are ineffective, or M-cholinergic (atrovent (ipratropium bromide)), used mainly in young children, or combined bronchospasmolytic (β2-agonist in combination with M-cholinergic - berodual

(ipratropium bromide + fenoterol)), used as a universal drug with a high safety profile.

The best option for the use of bronchospasmolytics may be inhalation through a nebulizer using special inhalation solutions in single doses with a face mask (for children under 6 years old) or with a mouthpiece (over 6 years old):
salbutamol in a dose of 0.1-0.15 mg/kg (no more than 5 mg at one time) or berotec (fenoterol), children under 6 years old – 5-10 drops, older than 6 years old – 10-20 drops or Atrovent (ipratropium bromide) , children under 6 years old – 10 drops, older than 6 years old – 20 drops of aboberodual (ipratropium bromide + fenoterol), children under 6 years old – 10 drops, from 6 years old – 20 drops.
If effective, bronchospasmolytic therapy is continued with the starting drug, using it orally or by inhalation every 4-6 hours, or methylxanthines (theophylline) of short (euphylline) or long-acting (teopek, teotard, etc.) are prescribed.

Emergency care for moderately severe acute obstructive bronchitis: inhalation of 1-2 doses of a bronchospasmolytic drug (see above); in the absence of a metered-dose inhaler or nebulizer, a 2% solution of euphilin at a dose of 4-5 mg/kg is slowly injected intravenously (i.v.) or, in some cases, it is used orally in the same dose or in inhalations on a physiological solution of sodium chloride; oxygen therapy through a nasal catheter or mask;

In the case of inefficiency of the applied therapy, it is possible to prescribe systemic glucocorticosteroids parenterally or orally in a dose of 1-2 mg/kg; repeat inhalation of a bronchospasmolytic. Emergency care for severe acute obstructive bronchitis: oxygen therapy through a mask or nasal catheter; inhalation therapy through a nebulizer or spacer with β2-agonists (berotek (fenoterol), salbutamol) every 20 minutes in the first hour and thereafter every 1-4 hours if necessary or long-term nebulization with individual selection of the dose; in the absence of inhalation technique or its insufficient efficiency, inject IV 2% solution of euphilin at a dose of 4-5 mg/kg as a jet and further - IV drip at a dose of 0.6-0.8 mg/kg/h for 6- 8 hours; glucocorticoids IV in a dose of 2 mg/kg with prednisone; infusion

therapy in the volume of 30-50 ml/kg using glucose-salt solutions in the ratio 1:1 during the first 6-8 hours.

4. To improve the drainage function of the bronchial thre, it is possible to use ambroxol in the form of a solution for inhalation through a nebulizer, ambroxol in the form of syrup or bromhexine in age-related doses orally.

5. Vibration massage and postural drainage.

6. Physiotherapy procedures: electrophoresis with euphilin, MgSO4.

7. Antiviral means.

8. Indications for the appointment of antibacterial drugs may be the following signs: prolonged hyperthermia, lack of effect from the therapy, the presence of persistent areas of hypoventilation in the lungs and/or asymmetry of physical data, increasing toxicosis, signs of brain hypoxia, the appearance of purulent sputum, uneven enhancement of the lung pattern on x-rays, in blood tests - leukocytosis, neutrophilia, increased ESR, sensitization by previous frequent acute viral infections or transferred shortly before this episode of the disease.

Treatment of acute bronchiolitis:

1. Hospitalization.

2. The diet is complete, according to the child's age, enriched with vitamins, high-calorie.

3. Inhalation of humidified oxygen.

4. First-line drugs are β2-agonists. The drug of choice should be considered salbutamol (2-4 doses after 20 minutes when using a metered-dose inhaler; 0.15 mg/kg (maximum 5 mg) or 0.03 ml/kg of a 0.5% solution in 3 ml isotonic solution through a nebulizer).

5. Anticholinergic drugs (atrovent, ipratropium bromide) by inhalation of 0.25 mg for young children and 0.5 mg for older children, in 3 ml of isotonic sodium chloride solution every 20 minutes). Use of combined bronchodilators in the form ofe berodual in drops for inhalation with a nebulizer 10-15 drops 2-3 times a day.

6. Glucocorticoids (prednisolone - 1 mg/kg every 4-6 hours, bolus dose of 2 mg/kg) simultaneously with the start of treatment with β2-agonists or IV in case of ineffectiveness of their use within 1 hour (according to the dose corticosteroids are increased until obtaining a clinical effect and restoration of sensitivity to inhalation of β2-agonists).

7. Euphilinization in cases of resistance of bronchospasm to inhalation of β2-agonists, corticosteroids or in case of problems in conducting artificial lung ventilation. The bolus dose (if the child has not received Euphilin before) is 7 mg/kg and is administered intravenously slowly in isotonic sodium chloride solution or 5% glucose solution. The maintenance daily dose of euphylin in mg/kg depends on age and is:

in newborns – 3.84;

in children aged 2-6 months. - 12;

7-11 months - 20.04;

1-9 years – 24;

9-14 years - 19.2.

8. Oral rehydration or, if necessary, infusion therapy for the purpose of correcting the water-electrolyte exchange at the rate of hydration: 12 ml/kg per 1 hour with 5% glucose solution or isotonic sodium chloride solution; physiological needs: 50-80 ml/kg per day of 5% glucose solution with 2 mmol of potassium and 3 mmol of sodium per 100 ml of infusate; ongoing pathological losses: 20-30 ml/kg per day of 5% glucose solution and isotonic sodium chloride solution.

9. Correction of acidosis: if the pH is lower than 7.3 and there is a base deficit (negative VE indicator) greater than 5 mmol/l, sodium bicarbonate is administered, the amount of which (in mmol) is calculated according to the formula:

(VE) × 0.3 × body weight (kg).

10. Desobstruction of the tracheobronchial thre; vibromassage and vibropercussion; bromhexine enterally, lasolvan (ambroxol) in age-related doses.

11. The volume of respiratory support and its invasiveness depend on the patient's condition.

12. Antibacterial drugs for joining bacterial flora: cephalosporins (cefazolin, ceftriaxone), semi-synthetic penicillins (amoxicillin; augmentin, amoxiclav (amoxicillin + clavulanic acid), azithromycin.

13. Antiviral drugs at the beginning of the disease - interferons, inosine).

14. Cardiotonic drugs in the presence of pronounced tachycardia (strophantin, corglycon).

Treatment of recurrent bronchitis. At the inpatient-polyclinic stage during an exacerbation, the following are prescribed:

1. Expectorant and mucolytic drugs of synthetic and plant origin (ambroxol, acetylcysteine, bromhexine, carbocysteine, guaifenesin, althea root, ivy drugs, plantain drugs, their combinations, etc.), ambroxol and others are widely used.

2. Antioxidants (vitamins of group B, C, E, A, unitiol).

3. Immunocorrectors (immunal, respibron, ribomunil, bronchovaxom, bronchomunal).

4. If necessary, bronchodilators (atrovent (ipratropium bromide), salbutamol).

5. Respiratory physiotherapy: inhalations with the addition of drugs, vibration massage and postural drainage, medical bronchoscopy).

6. Detoxification therapy (enterosorbents, vitamins, pectins).

7. Speleotherapy in artificial microclimate chambers with dry chloride-sodium mixtures during disease remission.

8. Application of laser acupuncture on biologically active points related to the respiratory and immune system, as well as a hydrolaser shower.

Treatment of chronic bronchitis

Etiotropic therapy of exacerbation of chronic bronchitis is started taking into account the type of possible causative agent. The indication for the appointment of antibiotics is an active bacterial process, which is indicated by a febrile temperature that lasts for more than 3 days, corresponding changes in the blood test (leukocytosis and shift of the blood formula to the left, accelerated ESR, high level of CRP).

If there is a dry, painful cough in the first days of the disease, antitussive drugs of central action are prescribed. For dry, irritable tracheal cough - antitussive drugs of peripheral action. With obstructive syndrome, the appointment of bronchodilators is shown: short-acting b2-agonists - salbutamol, fenoterol, atrovent etc., anticholinergic drugs and theophylline (methylxanthine) drugs.

Mucolytics (acetylcysteine, bromhexine hydrochloride, ambroxol hydrochloride) are prescribed to thin and release sputum. To improve the microcirculation in the lungs, drugs such as sermion, cinnarizine, complamin, trental, etc. are indicated in order to normalize the energy balance of cells (ATP, riboxin).

To prevent the accelerated development of pneumosclerosis, the use of non-steroids that inhibit the activity of inflammatory mediators and enhance the therapeutic effect of antibiotics is shown. Respiratory gymnastics includes elements of training mechanisms or constituent components of the respiratory act.

The gymnastics complex should include static breathing sound exercises for training prolonged exhalation. In addition to static, the complex also has dynamic breathing exercises, when physical exercises are combined with breathing. Removal of sputum is best achieved in the drainage position with the upper part of the body lowered, this is facilitated by vibromassage (manual or with the help of a vibromassager).

In the case of insufficient effectiveness of the therapy in catarrhal purulent and purulent endobronchitis, bronchoscopic remediation is indicated.

TESTS

1. Bronchitis in children is characterized by:

A. Cough;

B. Normal or subfebrile temperature;

C. Dry and single medium- and large-vesicular rales;

D. Tympanic percussion sound;

E. All of the above

2. Bronchiolitis in children is characterized by:

A. A small number of small vesicular and crepitant rales;

B. Sharp shortness of breath with an expiratory component;

B. Shortening of the percussive sound;

G. Tympanic shade of percussion sound;

D. Bronchial breathing during auscultation

3. Acute obstructive bronchitis in children is characterized by:

A. Moderate shortness of breath with an expiratory component;

B. A large number of small vesicular and crepitant rales;

C. Shortening of the percussive sound;

D. Tympanic shade of percussion sound;

E. Bronchial breathing during auscultation

4. In a 6-month-old girl, on the background of febrile body temperature, sharply expressed expiratory shortness of breath, tachycardia, and cough appeared for the first time. Allergological history is not burdensome. On examination, hyperemia of

the pharynx, box percussion sound, diffuse fine-vesicular and whistling rales. X-ray - signs of emphysema. RS virus was isolated from the nasopharynx. What is the most likely diagnosis?

A. Bronchiolitis.

B. Acute bronchopneumonia.

C. Bronchial asthma.

D. Aspiration of a foreign body.

E. Allergic alveolitis.

5. In patient V., 1.5 years old, shortness of breath with prolonged, whistling exhalation appeared for the first time against the background of SARS. Allergic reactions in the form of dermatitis to a number of food allergens were noted in a 1-year-old child. The condition is of medium severity T-36◦8, BH-42 per minute, pale, cyanosis of the nasolabial triangle is absent, shortness of breath is moderate of expiratory nature. Percussion over the lungs is a lung sound with a box tone. During auscultation - prolonged exhalation, dry whistling wheezing on exhalation. In an. blood leukocytes - 8.6 x109/l, lymph - 50%, e. - 1%, ESR - 12 mm/h.

A. Recurrent bronchitis

B. Bronchial asthma

C. Obstructive bronchitis

D. Pneumonia

E. Chronic bronchitis

Answers: 1. - A, 2. - B, 3. - A, 4. - A, 5. - C

Clinical task 1. A 3-month-old child was hospitalized for shortness of breath, cough, and weakness. She got sick 2 days before. Body temperature rose, shortness of breath and cough appeared. They treated with home methods. Child from IV

term pregnancy, weight at birth - 3600 g. Condition is serious. Pale skin, cyanosis of the nasolabial triangle. Trophics are reduced (body weight 5300 g). ChD 80 in 1 min with the participation of auxiliary muscles. Breathing is superficial. On percussion - a box percussion sound, on auscultation - breathing is hard, exhalation is prolonged, over the entire surface of the lungs there is a large number of fine bubbling wet rales. Limits of relative cardiac dullness: right - parasternal line, upper - II rib, left - 2 cm outward from the left midclavicular line. Heart sounds are muffled, heart rate - 165 in 1 minute. On the X-ray of the lungs, there are no shadows in both lungs, the bronchopulmonary pattern is enhanced.

Task. 1. What disease can be suspected in a child? 2. Leading syndromes that aggravate the course of the disease.

Answer standard. The child will have acute bronchiolitis. Respiratory and cardiovascular insufficiency syndromes.

Clinical task 2. A 1-year-old child is hospitalized for cough, increased body temperature, moderate expiratory shortness of breath. Ill for 2 days. Moderate condition. The skin and visible mucous membranes are pale. When percussing the chest, a box sound is observed; during auscultation, hard breathing, exhalation is somewhat prolonged, isolated dry wheezes. BH - 36 per 1 min, heart rate - 110 per min. Limits of relative heart dullness are normal, heart tones are clear.

According to the radiograph of the chest organs, an increased bronchopulmonary pattern is observed.

Task. 1. Make a diagnosis. 2. On the basis of what is this assumption made?

Answer standard. The child has acute obstructive bronchitis. The child has moderately pronounced shortness of breath of an expiratory nature, a box-like sound on percussion, a small number of dry wheezes.

Clinical task 3. The child is 7 years old. She fell ill 2 weeks before hospitalization in the clinic. A runny nose and cough appeared, then the body temperature rose to 38 °C. The child became frail, his appetite worsened. It is known from the anamnesis that the child is sick 3-4 times a year with the same clinical course of

the disease. On examination, the skin is pale. ChD - 26 in 1 min. Lung sound is heard over the lungs on percussion on both sides, on auscultation - breathing is hard, isolated isolated dry and wet rales of various calibers. Heart tones are clear, rhythmic; Heart rate - 86 in 1 minute. According to the radiograph of the chest organs: there are no foci of shadows, the roots of the lungs are dense.

Task. 1. Diagnose the child. 2. What means should be prescribed to the child in order to prevent recurrence of the disease?

Answer standard. The child has recurrent bronchitis in the acute stage. Speleotherapy in artificial microclimate chambers with dry chloride-sodium mixtures.

Pneumonia

Etiology. In most cases, in children, the infectious process in the lungs is caused by bacterial factors. In recent years, the dominant role of pneumococci in the etiology of ambulatory type pneumonia has been proven again. Haemophilus influenzae is the second most common cause of pneumonia in children. In addition, community-acquired pneumonia can be caused by atypical pathogens - mycoplasma and chlamydia. The causative agents of hospital pneumonia are Staphylococcus aureus, Pseudomonas, Escherichia, Klebsiella, Proteus, as well as pneumococci, which have beta-lactamase activity. In newborns in the first days of life, causative agents are gram-negative bacteria, group B streptococci, anaerobes, bacterioids, listeria, mycoplasma, aspiration, cytomegalovirus, herpes virus. From the 5th day to the 1st month, the etiology of pneumonia is dominated by aureus and other types of staphylococci, coliform bacteria, listeria, chlamydia, cytomegalovirus, herpes, RS virus. In children older than 1 month, pneumococci takes the leading place, and further, in terms of age, it retains the leading etiological role. It should be noted that bacterial-viral, virus-viral associations, as well as simpler ones (pneumocysts and fungi) will be the causative agents of pneumonia in children with immunological deficiency.

Pathogenesis. In children with pneumonia, the main route of infection into the lungs is the bronchogenic route with the spread of the infection along the respiratory tract up to the respiratory departments. The hematogenous route of infection into the lungs occurs with intrauterine pneumonia and septic conditions in the form of metastases. Bacteria enter the lower respiratory tract with mucus, which prevents the bactericidal effect of bronchial secretions on the microbe and, thus, promotes the reproduction of the infection.

The initial inflammatory changes in the lungs begin in the respiratory bronchioles, where there is no ciliated cylindrical epithelium. The infection, spreading beyond the respiratory bronchioles, causes inflammatory changes in lung parenchyma, i.e. pneumonia. In addition, during coughing, there is a possibility of

bronchogenic spread of infection by getting into the large bronchi, and then, spreading to other respiratory bronchioles, causing new foci of inflammation. With a limited prevalence of infection, the inflammatory reaction around the respiratory bronchioles causes focal and focal-draining pneumonia.

If the segmental bronchus is blocked with infected mucus, segmental pneumonia occurs due to the valve mechanism and, as a rule, with atelectasis of the segment. If a large amount of infected fluid is produced, it gets into the lobes of the lungs, causing granular (partial) pneumonia.

In children of the 1st year of life, pneumonia is more often localized in the 2nd segment of the right lung and in 4-6, and 9-10 on both sides. In older children, the 2nd, 6th, and 10th segments on the right and the 6th, 8th, 9th, and 10th segments on the left are more often affected.

In the pathogenesis of pneumonia, oxygen deficiency plays a leading role, which, first of all, is reflected in the activity of the central nervous system in the form of dysfunction of the vegetative department of the nervous system. At the height of the disease, sympathicotonia predominates, which is replaced by a vagotonic reaction. Changes in the cardiovascular system in children with pneumonia are caused by changes in the central nervous system, toxicosis, hypoxia and are accompanied by spasm of the arterioles of the small blood circulation, which leads to pulmonary hypertension and an increased load on the right heart, and thus causes a drop in the contractile ability of the myocardium, which leads to disorders of peripheral hemodynamics, disorders of microcirculation. Patients with severe pneumonia develop energy-dynamic insufficiency of the myocardium (Hegglin's syndrome).

The main phases of the development of pneumonia can be imagined as follows: after the infection penetrates into the tracheobronchial thre, there are inflammatory - edematous changes with the corresponding clinical picture. At the site of penetration of the bacterium into the lung tissue (due to the action of toxins

and enzymes), there is an alteration of the alveolar and interstitial tissue with its swelling, increased permeability and filling of the lumen of the alveoli with inflammatory fluid. Along with this, free radical oxidation processes are activated, which is accompanied by peroxidation of cell membrane lipids and, what is especially important, the surfactant content decreases. Ultimately, the pathological process due to manifestations of hypoxia and hypercapnia leads to the formation of respiratory failure. Hypoxia, which is mostly mixed (respiratory, circulatory, hemic, tissue) in nature, is the basis of respiratory failure.

Respiratory insufficiency is a manifestation of such a condition when the lungs do not maintain the normal gas composition of the blood or the normal gas composition of the blood is achieved due to the increased work of the external breathing apparatus, which leads to a decrease in the functional capabilities of the body. There are three degrees of respiratory failure.

In the first degree, respiratory failure occurs only during physical exertion and is characterized by pulmonary hyperventilation with the absence of hemodynamic and respiratory disorders. The ratio of the pulse to the frequency of breathing is 2.5 - 2: 1. At the second degree, which manifests itself already at rest, there are clinical and laboratory signs of disturbance not only of external breathing, but also of hemodynamics and mechanics of breathing. The ratio of the pulse to the respiratory rate is 2 - 1.5: 1. At the third degree of respiratory insufficiency, decompensation of both external breathing and hemodynamics and mechanics of breathing is clinically and laboratory diagnosed. The respiratory rate is more than 150% of the norm, generalized cyanosis, possible pathological types of breathing.

Classification. In children, it is recommended to use the classification, according to which the origin of pneumonia is established, its clinical and radiological form, localization, severity and course of acute pneumonia are revealed.

The origin of acute pneumonia is determined according to the conditions where the lung tissue was infected. Therefore, out-of-hospital (ambulatory, “home”, “sthret”) pneumonias and nosocomial (hospital, in-hospital) pneumonias, which develop during the first 48 hours of the patient’s stay in the hospital or during the 48 hours after discharge from the hospital, are distinguished by origin. inpatient Among nosocomial pneumonias, early ventilation (first 4 days on artificial ventilation) and late ventilation (more than 4 days on artificial ventilation) pneumonias are distinguished. Features of pneumonia in children are the presence of intrauterine (congenital) pneumonia, which manifests itself in the first 72 hours of a child's life. Newborns also have postnatal (acquired) pneumonia, which can also be out-of-hospital and nosocomial. A separate group consists of pneumonia in children with congenital and acquired immunodeficiency conditions.

Classification of pneumonia in children

Origin	Clinical and radiological form	Localizationtion	Severity	Clinical course
Outpatient (outpatient) Nosocomial (hospital) Ventilation: - early – late With immunodeficiency Intrauterine (congenital) Postnatal (acquired)	Focal bronchopneumonia Segmental pneumonia (mono- and polysegmental) Lobar (croupous) pneumonia Interstitial pneumonia	One-sided Bilateral Diffuse: - lung - lobe - segment	Uncomplicated Complicated: Synpneumonic pleurisy, Metapneumonic pleurisy, Pulmonary destruction, Lung abscess Pneumothorax, Pyopneumothorax, Infectious-toxic shock, disseminated intravascular coagulation	Acute (up to 6 weeks), Prolonged (from 6 weeks to 8 months)

			syndrome, Cardiovascular failure	

Focal pneumonia

The onset of the disease can be gradual, with the slow development of characteristic symptoms at the end of the 1st and the beginning of the 2nd week of the disease. Sometimes the disease develops quickly and already in the first 2-3 days the clinical picture makes it possible to diagnose pneumonia. Intoxication syndrome in such children is characterized by elevated body temperature, headache, decreased appetite, and sleep disturbances.

Shortness of breath can be moderate and has a mixed character. Each integument is pale, not sharply expressed perioral cyanosis, possible participation of auxiliary muscles in the act of breathing in the form of swelling of the wings of the nose and retraction of the supraclavicular fossa and intercostal space.

Physical changes are characterized by shortening of the percussion sound over the lesion and increased bronchophonia. Auscultatively, hard breathing is heard, locally it can be weakened with crepitation and various wet fine and medium-vesicular rales. Characteristic persistence of local symptoms.

Clinical blood analysis is characterized by leukocytosis, a shift of the leukocyte formula to the left, increased ESR, an increase in the level of C-reactive protein and procalcitonin. Radiologically, focal shadows in the lungs are revealed.

Segmental pneumonia

Pneumonias that occupy a segment or several segments are called segmental. There are three variants of the course of segmental pneumonia. In the first variant, with a benign course - intoxication, respiratory failure, sometimes even cough is absent in patients. The diagnosis is usually made after an X-ray examination. Patients with the second variant of the course of segmental pneumonias have a violent onset of the disease with fever, chest pain, pain in the abdominal cavity,

and focal changes in the chest. In the third variant of the disease, the clinical picture is observed as in older children with focal pneumonia with the feature that there are no wheezes. At the end of the 1st and beginning of the 2nd week of the disease, segmental shadows are formed in the chest (according to X-ray data). Atelectasis and pleural lesions are observed in such children in half of the cases, there is a tendency to abscessation, destruction and a protracted course.

Croupous pneumonia

Croupous pneumonia occupies a special place among childhood pneumonias. This condition is caused by the fact that pneumonia develops in a child's organism sensitized by pneumococci, which is prone to hyperergic reactions. This is due to the absence of croup pneumonias in the 1st year of life, their rarity at the age of 1-3 years. The most frequent localization of croup pneumonia in children is the upper or lower lobe of the right lung.

The disease begins acutely, with sharply expressed symptoms of intoxication in the form of a headache, an increase in temperature up to 40˚C. The general condition is sharply disturbed, there are chest pains, a herpetic rash on the lips (as a rule, on the affected side), shortness of breath is of a mixed nature. The patient can secrete "rusty" sputum from the first days. Focal changes are characterized by bronchial breathing, shortening of the percussive tone against the background of crepitating soft rales, pleural friction noise is heard.

Croupous pneumonia is also characterized by extrapulmonary lesions: muffled heart sounds, soft systolic murmur, widening of the boundaries of relative cardiac dullness, mild enlargement and tenderness upon palpation of the liver; changes in the nervous system can be observed in the form of sleep disturbances, changes in tendon and skin reflexes; kidneys ine form of albuminuria, cylindruria, sometimes erythrocyturia.

Interstitial pneumonia

Interstitial pneumonia is observed in one percent of the total number of patients with acute pneumonia. These pneumonias have a somewhat different

pathogenesis of disease development. Thus, they have the following staging of lung lesions: generalized spasm of arterioles, local thrombohemorrhagic syndrome, which contribute to surfactant deficiency, and therefore to the decline of alveoli and the development of microatelectases of the lungs. There are two types of interstitial pneumonias in the clinical picture.

Less symptomatic, subacute type. Such pneumonias are observed in children in the 2nd and 3rd months of life and are characterized by moderate shortness of breath, cough, hypoxemia and diffuse interstitial infiltration. In school-age children, a subfebrile body temperature, rapid fatigue, headache, and moderate cough are detected. Physical changes are very moderate and are manifested by hard breathing with single dry wheezes, moderate shortness of breath during physical exertion, expansion of the root of the lungs. A chest x-ray shows a cellular pattern of lung tissue in the affected area.

Manifest, acute type. This type of interstitial pneumonia occurs in children of early and preschool age against the background of allergic diathesis. The disease begins with the phenomena of neurotoxicosis and respiratory failure, fever and is accompanied by a dry cough. Hard breathing, dry wheezes, sometimes crepitations are heard in the lungs. Percussion reveals tympanitis and narrowing of the borders of relative cardiac dullness. One-sided expansion of the root of the lung can be detected. The course of interstitial pneumonia of the acute type is severe. Mortality at the height of intoxication is due to manifestations of viral encephalitis and virus damage to internal organs. X-ray changes in the pulmonary pattern are observed for 6-8 weeks or more. The consequence of this type of pneumonia can be the formation of pneumosclerosis.

Diagnosis and differential diagnosis of pneumonia

In 1986, WHO recommended that primary health care workers be guided by the following criteria for detecting pneumonia and prescribing antibiotics: cough, intercostal space depression, refusal to drink, the number of breaths in children

older than 1 year is more than 40 per minute, and in schoolchildren - more than 30 per minute.

All children with suspected pneumonia should undergo a chest X-ray. It is advisable to repeat the X-ray after 2-3 weeks, and in the case of a complicated course, according to the indications, in 2 projections. In case of hospital-acquired pneumonia, it is recommended to carry out a bacteriological examination of sputum and blood, both with the help of their culture, and conducting a serological examination to detect bacterial antigens. According to indications, such children are examined for the function of external breathing, an electrocardiographic examination is performed, and stool analysis for dysbacteriosis is performed. In the presence of concomitant diseases - examination of the sick child by relevant specialists.

Differential diagnosis of pneumonia is carried out with stenosing laryngotracheitis, bronchitis, bronchiolitis, foreign body aspiration of bronchi, tuberculosis, and lower lobe pneumonia - with appendicitis, intestinal obstruction, peritonitis, pyelonephritis.

Treatment. Treatment of pneumonia should be complex, taking into account the sanitary and hygienic and medical and protective regime. Treatment patients with pneumonia can be treated both at home and in hospital. The main means of treating pneumonia is the appointment of antibiotics. The choice of the starting drug depends on the sensitivity of the suspected causative agent, the age of the child, as well as the situation preceding the disease. Evaluating the clinical course of the disease with the established diagnosis of pneumonia, antibiotic therapy is started immediately. In a mild case, it is desirable to confirm the diagnosis by X-ray examination, and only after that to prescribe initial etiotropic therapy. It is recommended to use aminopenicillins, new macrolides and cephalosporins for empiric starting therapy of community-acquired pneumonia. For empiric antibacterial therapy, antibiotics should be used that have:

• directed spectrum of antimicrobial action - high activity against the main probable pathogens of pneumonia,

• optimal safety profile (low incidence of side effects),

• the possibility of creating high concentrations in the tissues and biological fluids of the respiratory organs,

• optimal cost/performance ratio,

• ease of use, which contributes to patients' adherence to the treatment regimen (compliance). This is especially important in the outpatient treatment of patients who are socially active, thereby violating the medication regimen. Compliance largely depends on the necessary frequency of taking the medicinal product during the day, as well as on the duration of the course of treatment. In such cases, preference is given to drugs that are prescribed 2-3 times a day or in short courses.

Doses of antibiotics for the treatment of pneumonia in children aged 1 month and older. up to 12 years old

Preparation	Dose inside	The dose is parenteral
1	2	3
Penicillins		
Ampicillin	50 mg/kg/day	50-100 mg/kg/day intravenously, intravenously
Amoxicillin	40-90 mg/kg/day	
Inhibitor-protected penicillins		
Amoxicillin/clavulanate	40 mg/kg/day**	
Ampicillin/sulbactam	-	100-150 mg/kg/day intravenously, intravenously
Cephalosporins		
Cefuroxime sodium	-	50-100 mg/kg/day intravenously, intravenously

Cefuroxime axetil	30-40 mg/kg/day, during meals	
Cefotaxime	-	50-100 mg/kg/day intravenously, intravenously
Ceftriaxone		20-75 mg/kg/day IV, IM
Ceftazidime	-	30-100 mg/kg/day IV, IM
Cefepime***		50-100 mg/kg/day intravenously, intravenously
Carbapenems		
Imipenem		60 mg/kg/day IV
Meropenem****		60 mg/kg/day IV
Monobactams		
Aztreonam		1 20-1 50 mg/kg/day IV
Aminoglycosides		
Gentamicin		5 mg/kg/day intravenously, intravenously
Amikacin		15-20 mg/kg/day intravenously, intravenously
Netilmicin		5 mg/kg/day intravenously, intravenously
Macrolides		
Midekamycin	30-50 mg/kg/day	-
Spiramycin	150,000 Units/kg/day	
Roxithromycin	5-8 mg/kg/day	-
Azithromycin	3-day course: 10 mg/kg/day 5-day course: 10 mg/kg in 1 day, then 5 mg/kg	
Clarithromycin	1 5 mg/kg/day	-
Drugs of other groups		
Lincomycin	30-60 mg/kg per day	10-20 mg/kg per day intravenously, intravenously
Clindamycin	1 0-25 mg/kg per day	20-40 mg/kg per day

		intravenously, intravenously
Vancomycin		40-60 mg/kg per day IV
Chloramphenicol	50-100 mg/kg per day	50-100 mg/kg per day intravenously, intravenously
Co-trimoxazole	8-10 mg/kg per day	8-10 mg/kg per day IV
Metronidazole	20-30 mg/kg per day	20-30 mg/kg per day IV

Notes: * - doses of antibiotics for children older than 12 years correspond to doses for adults; ** - for amoxicillin; *** - allowed in children older than 2 months; **** - allowed in children older than 3 months.

Modern oral antibiotics for children with a dosage regimen of 1-2 times a day

Antibiotic	Dose per reception			Multiplicity of injections per day	Course
	Up to 1 year	1-6 years old	7 years and older		
1	2	3	4	5	6
Macrolides					
Azithromycin					
Suspension 100 mg/5 ml	2.5-5 ml	5 ml	7.5-10 ml	1 time	3 days
Capsules 250 mg		-	1 cap.		
Roxithromycin					
Tablets 50 mg	1/2-1 tab.	1-1.5 tab.	1.5-2 tables.	2 times	7-10 days
Clarithromycin					
Suspension 125 mg/ml	2.5 ml	5 ml	7.5-10 ml	2 times	5-7-10 days
Cephalosporins of the III generation					
Cefodox suspension - 50 mg/5 ml	10 mg/kg	10 mg/kg	10 mg/kg	2 times	5-7-10 days

tablets - 200 mg					
Cefix - capsules of 400 mg No. 5 Syrup-100mg/5ml					
Cephalosporin II generation					
Cefuroxime					
Suspension 125 mg/5 ml	2.5-5 ml	5 ml	5-10 ml	2 times	5-7-10 days
Tablets 125 mg	1/2-1 tab.	1 table	Table 1-2		

Patients with nosocomial pneumonia should start with cephalosporin antibiotics of the 3rd - 4th generation in combination with vancomycin, aminoglycosides, meropenems. The criteria for the effectiveness of the prescribed antibiotics are a drop in temperature below 37.5˚C after 24-48 hours in uncomplicated and after 3-4 days in complicated pneumonia against the background of a decrease in intoxication and shortness of breath. Lack of effect requires a change of antibiotic. The antibiotic should also be changed in the event of serious adverse drug reactions. Duration of the course of antibiotics - about 7 - 14 days. For the prevention of complications of antibiotic therapy, it is mandatory to prescribe vitamins, bifidumbacterin, and lactobacterin. Patients with a compromised immune system are prescribed a 3-day course of antifungal drugs diflucan or fluconazole.

In children suffering from destructive pneumonia caused by staphylococcal infection, intrapleural antistaphylococcal bacteriophage in a dose of no more than 100 ml of bacteriophage is used in addition to antibiotic therapy.

Pathogenetic therapy includes the appointment of anti-inflammatory drugs, ibuprofen, paracetamol, mefenamic acid and other anti-inflammatory drugs.

It is appropriate to use expectorants that are presented on our pharmaceutical market as follows - expectorants: ivy, altei, mukaltin, various chest collections, bronchipret, bronchophyte, licorice root, pertussin. In order to better discharge mucus, mucolytics should be used: Ambroxol, Lazolvan, Acetylcysteine, Carbocysteine, Bromhexine, Bronchosan, Fludytek, Gedelix.

In patients with diagnosed segmental pneumonia, in addition to classic pharmacological therapy, if possible, remedial bronchoscopy should be used, which allows to unblock the valve mechanism of pneumonia in a shorter time, and thus significantly improve the aeration of the corresponding segment and its drainage. Taking into account the important importance of free radical oxidation in the pathogenesis of acute pneumonia, patients are prescribed antioxidants and membrane stabilizers in the form of vitamin E, galascorbin, unitiol, essentiale for 10-14 days. In the event of complications, patients are hospitalized in intensive care units and post-syndromic treatment is prescribed. In severe hypoxia, antihypoxants in the form of oxygen, droperidol, sodium oxybutyrate should be used against the background of metabolic correctors (glucose-insulin-potassium mixture, lipoic acid, riboxin).

A mandatory component is the appointment of physiotherapy in the form of inhaled bronchodilators and mucolytics: acetylcysteine, mucosolvin, chest electrophoresis with lidase, general massage and vibromassage. Patients are prescribed physical therapy. At the same time, the sick child needs to rehabilitate chronic foci of infection and treat underlying diseases.

TESTS

1. Pronounced toxicosis from the beginning of the disease, pallor of the skin with an earthy gray tint, febrile fever, tachycardia on the background of respiratory failure is a characteristic feature:

A. Obstructive bronchitis

B. Intestinal infection

C. Bronchiolitis

D. Staphylococcal pneumonia

E. Laryngotracheitis

2. The reason for the frequent development of pneumonia in young children is all the features of the respiratory system listed below, except:

A. Absence of secretory IgA on mucous membranes

B. Insufficient development of elastic tissue in the lungs and bronchi

C. Intensity and intensity of exchange processes

D. Decreased drainage function of the bronchi

E. A well-developed capillary network

3. Which of the listed symptoms are not crucial for confirming the diagnosis of pneumonia in a one-year-old child?

A. Cough, fever (temperature 38.2 - 39◦C)

B. Respiration rate is 50 per minute with intercostal spaces retracting

C. Hepatolienal syndrome

D. Cyanosis of the nasolabial triangle

E. Microvesicular moist rales and/or crepitus

4. A non-characteristic symptom of bronchopneumonia in children of the 1st year of life is:

A. Wet cough;

B. Shortness of breath of a mixed nature;

C. Fine-vesicular rales and/or crepitus;

D. Weakened breathing, determined locally;

E. Changes in blood pressure.

5. Bronchopneumonia in children is characterized by:

A. The presence of symptoms of intoxication;

B. Signs of respiratory failure;

C. Shortening of the percussive sound;

D. Fine-bubble rales and/or crepitus rales on the background of hard breathing;

E. All of the above.

6. A 5-year-old girl with transient T-system immunodeficiency has been observed for 2 months with a clinical picture of right-sided pneumonia. What course of pneumonia should be considered in this case?

A. Recurrent.

B. Wavy.

C. Sharp.

D. Chronic.

E. Prolonged

7. A 2.5-year-old boy was being treated for destructive pneumonia in the pulmonology department of the hospital. According to the results of additional examinations, the staphylococcal etiology of the disease was established. What is the permissible dose of antistaphylococcal bacteriophage used intrapleurally?

A. 5 ml.

B. 20 ml

C.50 ml.

D. 100 ml.

E. 10-15 ml.

8. A 9-month-old child has a high body temperature, cough, shortness of breath. Gets sick within 5 days after contact with ARVI patients. The child's condition is serious. Body temperature - 38°C, cyanosis of the nasolabial triangle. ChD - 54 per minute, blowing the wings of the nose during breathing. Percussion over the lungs: shortening of the sound on the right below the angle of the scapula, over other areas - a tympanic shade of the sound. During auscultation: fine bubbling wet rales on both sides, more on the right. What is the most likely diagnosis?

A. Pneumonia.

B. Acute bronchiolitis.

C. Acute laryngotracheitis.

D. Acute bronchitis.

E. Bronchial foreign body.

9. A boy, 10 years old, fell ill 2 days ago at home. Pneumonia was diagnosed by a pediatrician at the polyclinic. What is the most likely etiological cause of pneumonia? A. Escherichia coli.

B. Proteus.

C. Pneumococcus.

D. Klebsiela.

E. Staphylococcus.

10. A 10-month-old child was diagnosed with acute bronchopneumonia with destruction of the left lung. What pathogen most likely caused this disease?

A. Proteus.

B. Escherichia coli.

C. Blue fever bacillus.

D. Pneumococcus.

E. Pathogenic Staphylococcus aureus.

11. A child, aged 1 month, was born from the second pregnancy against the background of toxicosis in a woman suffering from chronic adnexitis. Body weight - 2900 g, length - 52 cm. Objectively: the child's condition is serious. The cry is quiet, natural reflexes are suppressed, muscle tone is reduced. Skin with a grayish tint, acrocyanosis. Breathing is shallow, non-rhythmic, 70 per 1 minute. Auscultation: weakened breathing, wet rales of various calibers. HR 150 in 1 min. The liver protrudes 2 cm from under the edge of the rib cage. Meconium was coming out. What is the previous diagnosis?

A. Sepsis.

B. Early postnatal pneumonia.

C. Intrauterine pneumonia.

D. Primary scattered atelectasis.

E. Late postnatal pneumonia.

12. An 8-year-old child was admitted to the hospital in serious condition with complaints of pain in the right side of the chest and hyperthermia for 5 days. At

home, he was treated with low-dose antibacterial drugs. Objectively: on the right below the angle of the scapula - percussive blunting. Auscultatively: breathing is not heard in the area of blunting. X-ray: in the lungs, intense darkening on the right, indistinctness of the sinuses, shift of the mediastinum to the left side. What disease should be suspected in this case?

A. Acute focal pneumonia.

B. Foreign body.

C. Tuberculosis of the lungs.

D. Croupous inflammation of the lungs with pleural damage.

E. Acute exudative pleurisy.

13. A 2-year-old child has a cough, frequent breathing, an increase in body temperature to 38°C for 3 days. In the first year of life, there were manifestations of allergic diathesis. Pale skin, perioral cyanosis. Shortness of breath of the expiratory type, BH - 54 in 1 min, heart rate - 122 in 1 min. On the left, near the corner of the scapula, a shortening of the percussive sound is determined, in other parts - a box sound. Breathing in the area of shortening is weakened, in other departments - hard with prolonged exhalation. Locally, fine bubbling sounds are heard, in other departments - scattered, whistling. What is the most likely diagnosis?

A. Bronchiolitis.

B. Bronchial asthma, period of attack.

C. Obstructive bronchitis.

D. Acute left-sided focal pneumonia with obstructive syndrome.

E. Exudative pleurisy.

14. A 10-year-old boy, after hypothermia, began to complain of a sharp headache, general weakness, and pain in the right iliac region. Body temperature 40°C, vomiting appeared. Objectively: significant shortness of breath of a mixed nature,

pale skin of the face with a blush on the right cheek, lagging of the right half of the chest in the act of breathing, dulling of the percussion sound on the right at the level of the lower lobe of the right lung, in the same area - weakened breathing. The abdomen is soft, painless during palpation. What disease is caused by the deterioration of the patient's condition?

A. Flu. B. Intestinal infection.

B. Acute appendicitis.

C. Acute cholecystitis.

D. Croupous pneumonia.

15. In a child, 2.5 years old, the onset of the disease is acute with an increase in body temperature to 38°C, abdominal pain, one-time vomiting. Objectively: the condition is serious. The skin is pale. ChD - 80 per 1 min. Cyanosis of the nasolabial triangle. The right half of the chest lags behind in the act of breathing. Dullness of the percussion sound on the right below the angle of the scapula, breathing there is significantly weakened, no wheezes are heard. Heart tones are weakened, tachycardia. The abdomen is moderately swollen, painful during palpation in the right hypochondrium. On the X-ray of the chest: on the right, intense homogeneous darkening in the projection of the VII and X segments, the sinuses are free. What is the most likely diagnosis?

A. Acute appendicitis.

B. Right-sided polysegmental pneumonia.

C. Croupous pneumonia.

D. Acute obstructive bronchitis.

E. Right-sided exudative pleurisy.

16. A child, 4 months old, refuses the breast. Against the background of catarrhal phenomena, general weakness, pallor, perioral cyanosis, tension of the wings of the

nose, a deep wet cough, fine-bubble rales over the left lung, hard breathing with prolonged exhalation appeared. BH - 68 in 1 min, heart rate -168 in 1 min. In the blood: er. - 3.6×1012/l, leu. - 22.0×109/l, ESR - 25 mm/h. What research should be done to verify the diagnosis?

A. Bronchoscopy.

B. CT chest.

C. X-ray of chest organs.

D. ECG.

E. Spirographic research.

17. A girl, 10 years old, has been sick for the 6th day. Lethargic, body temperature - 38.3 °C, pale skin, periorbital shadows. Dry cough, shortness of breath, shortness of breath - 32-36 in 1 minute. Over the lungs - a shortening of the percussion sound on the right at the level of the IX-X segments, breathing is hard, in the area of the shortening it is weakened, fine bubbling rales, crepitation. What examination is crucial for establishing a diagnosis?

A. X-ray of the chest.

B. Clinical blood analysis.

C. Bacterial research of sputum.

D. Bronchography.

E. bronchoscopy.

18. A 6-month-old girl, who was born full-term, was admitted to the clinic on the 3rd day of the disease with complaints of frequent dry exhausting cough, shortness of breath, and an increase in body temperature up to 37.6◦C. After clinical and X-ray examination, pneumonia was diagnosed. What pathogen most likely caused the disease in the child?

A. Pneumocyst

B. Pneumococcus

C. Staphylococcus

D. Hemophilic bacillus

E. Klebsiella pneumonia

19. A 5-year-old girl receiving treatment in the pulmonology department for destructive pneumonia, according to the results of a bacteriological examination, the staphylococcal etiology of the disease was established. What antibiotic is the most appropriate to prescribe in this case?

A. Ceftriaxone

B. Penicillin

C. Gentamicin

D. Erythromycin

E. Levomycetin

20. A 14-year-old girl was admitted to the clinic with complaints of general weakness, an increase in body temperature up to 37°C, a non-productive cough, headache, muscle pain. Auscultatively, in the lower parts of the lungs, rales are heard on both sides. Tachycardia. In the analysis of peripheral blood, moderate leukocytosis, elevated ESR. Radiologically, a heterogeneous infiltrative shadow is noted in the lower parts of the lungs. Pneumonia was diagnosed. Several similar cases have been registered in the class where the girl studies. What is the likely causative agent of these pneumonias?

A. Staphylococcus aureus

B. Streptococcus pneumoniae

C. Mycoplasma pneumoniae

D. respiratory viruses

E. pathogenic fungi

21. A 7-year-old boy complains of a headache, feeling hot, feverish temperature, shortness of breath, cough with slight sputum and blood impurities, nosebleeds. Physical shortening of the percussive sound paravertebrally, hard breathing. X-ray signs of emphysema and mesh pattern of lung tissue. The most likely diagnosis

A. Interstitial pneumonia

B. Obstructive bronchitis

C. Miliary tuberculosis

D. Leffler's syndrome

E. Hemorrhagic vasculitis

Answers: 1. - D, 2. – E, 3. - C, 4. - E, 5. -E, 6. – E, 7. - D, 8. - A, 9. - C, 10 - E, 11. - E, 12. - D, 13. - D, 14. - E, 15. - B, 16. - C, 17. - A, 18. - B, 19. - A, 20 - C, 21. – A.

Clinical task 1. A 6-year-old boy complains of an increase in body temperature up to 39◦C, weakness, one-time vomiting, pain in the abdominal cavity, wet cough. During the examination: the condition is severe, pronounced dyspnea of the mixed type, heart rate - 42/min., auxiliary muscles take part in the act of breathing. The lag of the right half of the chest during breathing is noted. Percussionally - a dull sound in the lower right, no breathing can be heard in the same area by auscultation, on the left - breathing is hard. Heart sounds are weak, 116 beats/min. The stomach is soft. Blood saturation - SO2 - 89%.

What is the most likely diagnos?, prescribe treatment.

Answer standard. Out-of-hospital right-sided lower lobar (croupous) pneumonia of a severe course. Dn I-II century, SSN - I century. Amoxacillin 750

thousand × 3 times a day, ibuprofen 200 mg × 2 times a day, ambroxol 0.015 mg × 2 times a day, oxygen support.

Clinical task 2. A 3-year-old girl is being treated on an outpatient basis for an acute respiratory viral infection, she received antiviral drugs and antipyretic therapy. On the 4th day of the disease, the condition worsened: the body temperature rose to 38.9◦C, the dry cough transformed into a wet one, shortness of breath appeared, dulling of lung sounds on the left side of the back surface of the lungs, on auscultation there were small alveolar rales and crepitus against the background of a weakened breath. ChD- 36 per min. In the blood analysis, leukocytosis with a shift of the formula to the left, a high level of CRP and procalcitonin appeared. Blood saturation - SO2 - 94%.

What caused the deterioration of the patient's condition? Adjust the treatment. Answer standard. A bacterial infection with manifestations of left-sided community-acquired bronchopneumonia was added. It is necessary to add antibacterial therapy in the form of amoxacillin 500 thousand × 3 times a day, ambroxol 0.015 mg × 2 times a day.

Clinical task 3. An 8-year-old girl has pneumonia. She started a course of broad-spectrum antibiotic therapy (amoxacillin), but there was no positive development of the disease 48 hours after the start of therapy.

What criteria of positive clinical dynamics of the disease do you know? Prescribe treatment.

Answer standard. Reduction of symptoms of intoxication syndrome (fever), reduction of frequency and intensity of cough. The patient needs to change the starting empiric antibiotic therapy (amoxacillin) to a cephalosporin antibiotic - ceftriaxone.

Bronchial asthma

Bronchial asthma is a chronic allergic inflammatory process characterized by attacks of suffocation or asthmatic status as a result of generalized narrowing of the airways due to spasm of bronchial smooth muscles, hypersecretion, dyscrinia (changes in the properties of mucus) and swelling of the bronchial mucosa caused by immunological and nonspecific, congenital or acquired mechanisms.

Epidemiology. Epidemiology of bronchial asthma. Studies conducted in recent decades in Europe and other continents clearly confirm the steady growth of the incidence of bronchial asthma. In Ukraine, according to official statistics, the prevalence of bronchial asthma has doubled over the past decade. There is such a phenomenon as the rejuvenation of asthma, which is increasingly found in young children. Bronchial asthma is the most common cause of disability in children with non-specific chronic lung diseases.

Etiology. Etiological factors of the occurrence and exacerbation of bronchial asthma are: household allergens (of the genus Dermatophagoides), book dust; allergens of animal origin (hair, down, feathers, dander, excrement of cats, dogs, rabbits); insects (insects, bees, cockroaches); fungal allergens (Alternaria, Cladosporium, Mucor, Candida, Penicillium, Aspergillus); pollen allergens (thres, bushes, cereals, weeds); medicinal products - primarily antibiotics; chemical allergens – xenobiotics (chromium, nickel, manganese, formaldehydes); viruses and vaccines (APD pertussis component); various food allergens.

Pathogenesis. Pathogenetically, three stages of the disease are distinguished - immunological, pathochemical and pathophysiological, which reflect the course of the chronic inflammatory process in the child's body. The basis of the immunological stage is the excessive formation of antibodies in the form of IgE, however, they occur as well as immunocomplex reactions and reactions of the delayed type. As a result of immunological reactions, mast cells are activated, which leads to the release of a large number of inflammatory mediators (histamine,

chemotactic factor of eosinophils, chemotactic factor of neutrophils, leukotrienes, prostaglandins, thromboxanes), which leads to the accumulation of eosinophils, neutrophils, basophils in the focus of inflammation, accompanied by bronchoconstriction, increased permeability of blood vessels, and therefore deepening of inflammatory phenomena in the bronchi.

C**lassification**. As a basis for the classification of bronchial asthma, the consensus documents of recent years recommend the course of a chronic allergic process with a distinction between intermittent and persistent courses of bronchial asthma. The persistent course of the disease is characterized by a mild, moderately severe and severe course of bronchial asthma. The proposed classification was revised and expanded in 2006 by introducing the concept of control over bronchial asthma. We distinguish phenotypically asthma with a predominance of an allergic component, non-allergic asthma and mixed asthma. For doctors, it is recommended to use the classification with the allocation of controlled, partially controlled and uncontrolled bronchial asthma.

On the basis of many years of scientific and practical experience, reproduced in the international agreement documents of the 2006 CINA (Global Initiative for Asthma), specific indicators of asthma control are proposed, namely:

Symptoms are minimal (maximum twice a week), ideally completely absent, and asthma does not wake the child at night. Minimal use (short-acting ß2-agonists (ideally, there is no need to prescribe them).

The PSHV indicator (peak expiratory velocity) is higher than 80% every morning (which means it is normal). There are no exacerbations and emergency calls to the ambulance. There are no restrictions on physical activity and the child leads an age-appropriate lifestyle (does not differ from healthy peers). Based on the above indicators, each practicing physician can clearly identify a group of children who failed to control the disease.

The experience of the world's leading children's allergists proves that there are three variants of uncontrolled asthma in children:

Uncontrolled bronchial asthma against the background of a severe version of the persistent course of the disease (actually in such cases there is a risk of rapid and sometimes fatal development of an asthmatic condition). Uncontrolled bronchial asthma against the background of a mild and moderate persistent course of the disease, when, despite the appointment of seemingly optimal therapy, it is not possible to achieve control over the disease. Uncontrolled bronchial asthma against the background of an intermittent course of the disease with sudden acute attacks that develop without a visible objective reason (a clinical variant of the so-called "fragile" asthma).

Searching for reasons that do not allow to achieve control over the disease is extremely important. In order to solve the issue of controlling the disease, the following questions must be consistently resolved: assess the adequacy of the basic therapy. Analyze: are the medical prescriptions carried out correctly and in full? Repeat differential diagnosis. Look for accompanying pathology (reflux, immunodeficiency, atypical flora, helminthiasis, congenital malformations of the cardiovascular system, etc.). Look for additional, previously not taken into account, provocative factors (allergens, viral infections, environmental factors, smoking, medications, etc.).

Clinical manifestations. Exacerbations of bronchial asthma in the vast majority of children occur against the background of viral infections, cooling, excessive physical exertion, and changes in meteorological conditions. A feature of the attack period in children is the harbinger period, which occurs 2-3 days before the onset of an asthma attack and is characterized by restlessness, excitement, disturbance sleep. Phenomena of disorders of the central nervous system in the form of sensation, tickling in the throat, heaviness and compression in the chest are observed. Against this background, a dry, unproductive cough appears, accompanied by increasing shortness of breath. When examined, such children are

excited, as a rule, the upper shoulder girdle is fixed, the skin is pale, perioral cyanosis is observed. In the lungs, percussion finds a boxy shade, a low position of the borders of the lungs, narrowing of the borders of relative cardiac dullness. Auscultatively, against the background of hard breathing, dry and wet rales of various calibers are heard, which can sometimes be heard at a distance, exhalation in such children is prolonged.

The duration of an attack can vary from 30-40 minutes to several hours or even days (status asthmaticus). The criterion for asthmatic status in children is the presence of a protracted, difficult-to-treat attack of bronchial asthma lasting more than 6 hours. In the course of the asthmatic status, three stages are distinguished: the I stage of relative compensation and is characterized by the formed resistance to sympathomimetics;

II - stage of decompensation, characterized by increasing respiratory insufficiency of the obstructive type, manifested by cyanosis, shortness of breath, absence of cough, and extremely severe general condition of the patient. Such a phenomenon as "silent lung" is observed, that is, there are no rales in the lungs during auscultation. There are significant changes in the psyche in the form of a feeling of fear, depression, prostration.

III - stage of asthmatic status (hypoxemic coma, asphyxiation syndrome) develops as a result of uncompensated respiratory and metabolic acidosis, which is accompanied by prostration, loss of consciousness against the background of generalized cyanosis, shortness of breath, swelling of the neck veins. Wheezing stops over the lungs.

The post-attack period is characterized by a gradual decrease in shortness of breath, the appearance of a productive wet cough, an improvement in bronchial patency, and a decrease in tachycardia.

Diagnosis. The diagnosis of bronchial asthma is made on the basis of the following data:

1. Presence of allergy history in the family and allergy in the child;

2. The connection between an attack of bronchial asthma and infectious and non-infectious factors.

3. The presence of typical attacks of bronchial asthma in the anamnesis.

4. Occurrence of attacks in the evening and at night.

5. Eosinophilia of blood and sputum.

6. High level of general and specific IgE in blood, high level of NO (nitrogen oxide) in exhaled air.

7. Positive skin tests with allergens

8. Reduction of bronchial patency indicators according to spirographic data.

Differential diagnosis. In young children, a differential diagnosis should be made with bronchiolitis, congenital lung defects (aplasia and hypoplasia of the lungs, laryngotracheomalacia), bronchopulmonary dysplasia, with secondary obstructive syndrome with retching, vomiting. In older children, it is necessary to think about a foreign body of the bronchi, true and false croup, epiglottitis, cardiac asthma, the debut of collagen diseases, autonomic dysfunction.

Treatment.The basis of the treatment of bronchial asthma is pathogenetic anti-inflammatory therapy, which is aimed at restoring bronchial patency at all levels of the bronchi, preventing repeated attacks of bronchial asthma and achieving long-term remission, and characterizes control over the disease. Control drugs, as a rule, are taken daily for a long time and are the basic drugs that allow you to maintain control over bronchial asthma. Basic anti-inflammatory therapy primarily includes glucocorticoids, which are now used in the form of pocket inhalers with different active substance content (beclomethasone dipropionate, budesonide, ciclesonide, flunisolide, fluticasone, pulmicort). In addition, modern pocket inhalers combine two active substances, a glucocorticoid and a bronchodilator (formoterol) of prolonged action, seretide or symbicort. Such a

combination is the gold standard of anti-inflammatory therapy and allows you to successfully carry out basic therapy for a long (months) period without complications. Cromons (nedocromil sodium and sodium cromoglicate) and leukotriene modifiers - montelukast and zafirlukast can be used in patients with a mild course of the disease. Antileukotriene drugs can also be used to reduce the level of glucocorticoids among patients with a moderately severe course of the disease. In patients with a severe uncontrolled course of bronchial asthma, therapy can be supplemented with a minimum dose of systemic glucocorticoids. If there is no effect, anti-IgE drugs (omalizumab) are additionally prescribed.

If the doctor has convinced the mother of the need for inhaled corticosteroid treatment, the optimal dosage should be prescribed. Otherwise, it is often necessary to increase the dose of the drug after a short time, which alarms the mother, and the doctor loses the main thing - the patients' trust in the treatment.

However, if the effect cannot be achieved after prescribing medium doses of ICS, then again the doctor is faced with the problem of choosing a therapeutic tactic. There are two possibilities:

• or increase the dose of inhaled corticosteroid,

• or add other drugs (long-acting ß2-agonists, antileukotriene drugs, theophyllines) to inhaled corticosteroid. Practice proves that the best solution in pediatrics is the appointment of medium doses of ICS and a long-acting ß2-agonist (seretide, symbicort).

To improve the effectiveness of treatment, it means establishing cooperation with the patient (improvement of compliance), since in such cases the problem most often lies in patients' non-compliance with medical recommendations.

Do not forget to always ask your patient how he takes his medication, monitors his treatment, etc. A very important aspect of achieving asthma control is the adequate and correct use of means of delivery of inhaled drugs. The success of the treatment largely depends on the adequacy of the selection of the inhalation agent and the correctness of the inhalation. Teaching patients the technique of

conducting inhalations with subsequent control allows to significantly increase the percentage of correct inhalations.

In the event of an exacerbation of the disease, symptomatic inhalation therapy should be started immediately with β2-adrenomimetics of short action (salbutamol, albuterol, fenoterol), for children under 5 years of age, if possible, use them through an inhaler - nebulizer, M - cholinolytics (ipratropium bromide), theophyllines (up to 24 mg/kg/day). Treatment can be carried out with the use of inhalation, oral and injection routes of drug administration. At the same time, drugs for inhalation have the greatest advantages, since they create the highest concentrations of active substances precisely in the respiratory tract with the minimization of systemic complications.

Children with bronchial asthma should have a first-aid kit at home with a kit for emergency care (glucocorticosteroids, adrenomimetics for parenteral administration, a nebulizer with a set of bronchodilator drugs and ICS for inhalation).

Provide daily peak flowmetry and keeping a diary of self-observation. Provide constant control and communication with the attending physician.

Prevention. Preventive measures consist in the identification and reduction of risk factors (hypoallergenic lifestyle), the implementation of highly effective non-medicinal treatment measures (speleotherapy, climatotherapy, treatment with singlet oxygen, nitrous oxide). Highly effective preventive measures are the development of partnership relations between the doctor and the patient.

Tests

1. The leading pathogenetic mechanisms observed in bronchial asthma are:

A. Chronic allergic inflammation of the respiratory tract.

B. Chronic infectious inflammation in the respiratory tract.

C. State of bronchial hyperreactivity

D. Autoimmune inflammation

E. Combination of chronic allergic inflammation and bronchial hyperreactivity.

2. What immunological reactions are most common in bronchial asthma:

A. Reaginov - type I

B. II type - cytolytic

C. III type - immunocomplex

D. IV type - slowed down

E. Pseudoallergic

3. Which allergens occupy a leading place in the etiological structure:

A. Pilkovi

B. Household

C. Hrybkovi

D. Epidermal

E. Viruses

4. The modern classification of bronchial asthma is based on the following approach: A. According to the degree of control of bronchial asthma

B. According to the form of the disease

C. According to the duration of the disease

D. According to the severity of the course of the disease

E. By severity and degree of control

5. The main clinical manifestation of bronchial asthma is:

A. Expiratory shortness of breath

B. Palpitation attack

C. Cough attack

D. High temperature

E. Leukocytosis

6. The severity of the disease depends on:

A. Frequencies of asthmatic attacks

B. Frequency of visits to the doctor

C. The volume of used medicinal products

D. Indicators of spirometry

E. Frequencies of asthmatic attacks and spirometry indicators

7. For bronchial asthma of what degree of severity is it recommended to prescribe leukotriene receptor antagonists as basic therapy?

A. With a mild degree of bronchial asthma

B. In the case of a disease with a moderately severe course

C. With a severe course of bronchial asthma

8. For bronchial asthma of what degree of severity is it recommended to prescribe corticosteroids as basic therapy?

A. With a mild degree of bronchial asthma

B. In the case of a disease with a moderately severe course

C. With a severe course of bronchial asthma

9. From what age can spirometry and peak flowmetry be used as means of monitoring the functional state of the respiratory system?

A. Since one year

B. From the age of three

C. From the age of five

D. From the age of seven

E. From the age of ten.

10. Fast-acting drugs to eliminate an asthma attack include:

A. Salbutamol

B. No-shpa

C. Pulmicort

D. Suprastin

E. Flixotide

11. A feature of the course of an attack of bronchial asthma in young children is;

A. Minimal severity of bronchospasm

B. Predominance of the vasosecretory component

C. A large number of moist rales in the lungs

D. Pronounced expiratory shortness of breath

E. All of the above

12. Respiratory failure during an attack of bronchial asthma due to:

A. Bronchospasm

B. Hypersecretion

C. Edema of the bronchial mucosa

D. All of the above

13. An 11-year-old boy is discharged home from an allergy hospital with a diagnosis of: Bronchial asthma, severe course with frequent attacks, paroxysmal period. DN 1- 2. Which of the listed drugs would you prescribe for long-term basic therapy of bronchial asthma

A. Budesonide

B. Salbutamol

C. Tyled

D. Montelukast

E. Eufilin

14. A 4-year-old boy was admitted to the hospital with complaints of shortness of breath and attacks of dry cough, which have been bothering him 1-2 times a month for the past 6 months. Attacks occur more often at night, mainly against the

background of ARVI. When examining the lungs, there is a percussive box sound, auscultation - many dry whistling rales on both sides. Your diagnosis?

A. Bronchial asthma

B. Acute pneumonia

C. Acute obstructive bronchitis

D. Kashlyuk

E. Exogenous allergic alveolitis

15. During the examination of a 7-year-old child, who periodically has pronounced attacks of expiratory shortness of breath, sharply positive scarification tests with house dust and many pollen allergens were found. In the general blood test - eosinophilia 10%. Your previous diagnosis?

A. Bronchial asthma.

B. Pollinosis.

C. Tuberculosis of respiratory organs.

D. Recurrent bronchitis.

E. Acute obstructive bronchitis.

16. A child with obstructive syndrome was admitted to the clinic. What clinical signs can serve as criteria for establishing a diagnosis of bronchial asthma?

A Positive allergy history (intrauterine sensitization, presence of exudative diathesis, absence of catarrhal phenomena of the upper respiratory tract, food, drug allergy, etc.).

B. Hereditary predisposition to allergic diseases.

C. Eosinophilia in the general blood test, increased level of IgE, increase in T-helpers, decrease in T-suppressors.

D. Positive scarification samples with non-infectious allergens.

E. All of the above.

17. A 10-year-old child was admitted to a hospital for treatment with a diagnosis: bronchial asthma, uncontrolled, severe course. The onset period. Asthmatic status. Determine the maximum therapeutic daily dose of eufilin.

A. 20-24 mg/kg

B. 10-15 mg/kg

C. 15-20 mg/kg

D. 25-30 mg/kg

E. 30-34 mg/kg

Answers: 1. - E, 2. - A, 3. - B, 4. - E, 5. - A, 6. - E, 7. - B, 8. - B, 9. - C, 10. - A, 11. - E, 12. - D, 13. - A, 14. - A, 15. - A, 16. - E, 17. – A.

Clinical task 1. A 5-year-old child is at a doctor's appointment, who has had short-term attacks of nausea during the last year, which recurred 3-4 times a month and disappeared in a few hours. An extreme attack of dysentery was observed in a child after a night's sleep on the eve of a visit to the doctor. It is known that the child was on early artificial feeding, there were periodic manifestations of atopic dermatitis, an allergic reaction to penicillin. During examination - dry cough, moderate expiratory shortness of breath, blood pressure - 34 in 1 minute. During auscultation: breathing is hard, exhalation is prolonged, dry whistling rales on both sides. Heart sounds are moderately muffled, rhythmic, pulse - 110 per 1 minute. No deviations were found in other systems. The child does not fall behind in physical development. What is the most likely diagnosis? Assign basic control therapy.

Answer standard. Bronchial asthma mild persistent course, post-attack period. Budesonide inhalation - 250 mg per day for 3 months.

Clinical task 2. A 10-year-old child has a history of regular weekly attacks of nausea, which have been observed for several years. The child was on basic control therapy with budesonide 250 mg/day, but the parents did not control the regularity of taking the control medication. On examination by the doctor, the child is frail, cough is dry, moderate expiratory shortness of breath, blood pressure - 32 in 1 minute. During auscultation: breathing is hard, exhalation is prolonged, dry whistling rales on both sides. Heart sounds are moderately muffled, rhythmic, pulse - 116 per 1 min., blood saturation sO2 - 94%. No deviations were found in other systems. What is the most likely diagnosis? Assign emergency therapy.

Answer standard. Bronchial asthma persistent moderate course, uncontrolled, attack period. Salbutamol inhalation was prescribed up to 4 times a day and budesonide was resumed.

Pollinosis in children

Pollinosis is a group of allergic diseases caused by plant pollen and characterized by acute inflammatory changes in the mucous membranes and skin.

Epidemiology. In recent years, diseases that arise as a result of sensitization of the human body to plant pollen - pollinosis - have spread significantly throughout the world. According to the WHO, pollinosis affects about 20% of the population of different countries, and the specific weight of this pathology in the structure of allergic diseases is 29%. In Ukraine, the incidence of hay fever has not been sufficiently studied, but there is evidence that adults are more often affected. In children older than 5 years, pollinosis is diagnosed in 5 - 9% of cases, more often in boys, and after 15 years - in girls.

Etiology. The reason for the development of pollinosis is extremely fine pollen (0.02-0.04 mm) of wind-pollinated plants (grasses, weeds, thres), which has a pronounced allergenic activity. The concentration of pollen in the air depends on a number of factors, in particular, it is higher in the morning and evening hours, as well as in the presence of dry and windy weather.

In Ukraine, there are three periods of plant flowering (and, therefore, three “waves” of pollinosis):

- spring (April, May)

- flowering thres; - summer (June, July)- maximum flowering of cereals;

- summer-autumn (July-September) - flowering of weeds, corn, sunflower. In a certain part of patients, a combination of different flowering periods of plants is observed.

The main causes of pollinosis during flowering periods are:

Spring pollinosis: thre pollen (birch, oak, alder, walnut, poplar, hazel, chestnut, maple, birch, ash, etc.). It is important to consider cross-allergic reactions occurring in patients allergic to thre pollen (for example, birch):

- food allergy to pears, apples, apricots, plums, peaches, kiwi, cherries, cherries, nuts, parsley, celery and carrots;

- food allergy to birch sap;

- medicinal allergy to preparations containing birch buds, alder cones, oak bark, etc.

Summer pollinosis: pollen from meadows, cultivated plants and grasses (timothy, fescue, vetiver, ryegrass, sedge, sunflower, corn, rye, etc.). Cross-allergic reactions are possible in the form of food allergy to beverages obtained from the fermentation of cereals: kvass, beer, food allergy to legumes, soy, sorrel, medicinal allergy to drugs containing cereals or their processing products.

Autumn pollinosis: weed pollen (ragweed, wormwood, wheatgrass, quinoa, etc.). Cross-allergic reactions in the form of food allergy to sunflower seeds and products containing them (sunflower oil, halva, mayonnaise), food allergy to pumpkin (cucumbers, zucchini, watermelon, cantaloupe), nightshade (eggplant, tomatoes), quinoa (spinach, beetroot), cruciferous vegetables (mustard, cabbage, radish). Food allergy to drinks containing wormwood extract (absinthe), medicinal allergy to preparations and collections of herbs, which include chamomile, wormwood, mother-and-stepmother, nettle, calendula, can also be observed. In general, the period of exacerbation of pollinosis lasts from 1-2 to 5-6 months.

Pathogenesis. The basis of the immunopathogenesis of pollinosis is mostly the IgE-dependent reagin mechanism of the development of allergic reactions. Allergen/pollen granules settle mainly on the mucous membrane of the nose, pharynx, conjunctiva, contacting the receptor apparatus of immunocompetent cells after 30 seconds. Hidden sensitization by pollen allergens is possible, which can last for several years. Pollen and other allergens, penetrating through the epithelium, come into contact with mast cells, on the surface of which there are antibodies of the IgE class, formed during the sensitization stage. After the interaction of pollen and other allergens with IgE, mast cells degranulate with the

release into the intercellular space of a significant number of different biologically active allergy substances that stimulate the function of eosinophilic granulocytes, monocytes, lymphocytes and other immunocompetent cells. This leads to expansion of capillaries, increase in their permeability, edema of the mucous membrane, hypersecretion and decrease in the protective function of the epithelium. During the exacerbation of pollinosis, the level of general and specific IgE increases in the blood of patients, the high concentration of which can also be determined during the period of remission of the disease. Patients with pollinosis develop an imbalance in the cytokine profile and T-cell subpopulations. Considering the fact that plant pollen can adsorb chemical microelements and microfungi, the development of not only allergic, but also non-allergic reactions, which are formed without the participation of IgE, is possible. Thus, at the basis of the pathogenesis of pollinosis, the first type of allergic reactions, due to the hyperproduction of IgE, prevails, the fourth type is less common. Pollinosis is more often detected in persons with a burdened heredity and a tendency to atopy with hyperproduction of IgE, which is registered in 40% of the population. The formation and development of atopy depends on a set of genes, of which there are already more than 20.

Cinical manifestation. The main clinical manifestations of pollinosis are quite diverse, depending on the degree of severity and localization of the pathological process:

- diseases of the upper and lower respiratory tract (allergic rhinitis, rhinosinusitis, bronchial asthma);
- allergic eye damage (allergic conjunctivitis).
- allergic skin diseases (urticaria, contact dermatitis);

Most often (in 50-90% of cases) rhinoconjunctival syndrome is noted, which has an acute onset and is characterized by symptoms of rhinitis with rhinorrhea, watery, and after the addition of a secondary infection - yellow discharge from the nose, paroxysms of sneezing, impaired nasal breathing, itching of the nose and palate , dry cough. Pollen rhinorrhea is replaced by a period of complete nasal

congestion, difficulty in nasal breathing, development of rhinosinusitis (sinusitis, frontitis, etc.). Patients may experience increased breathing, they wrinkle their nose, rub its tip (a symptom of an allergic salute). Increased sensitivity of the mucous membrane of the nose to cooling, dust, and sharp smells is observed.

The International Association for Allergic Rhinitis (ARIA), together with the WHO, proposed a classification of allergic rhinitis depending on the nature of the course (intermittent or persistent) and the severity of its symptoms (mild, moderate, severe).

Allergic rhinitis (AR) is one of the five most common chronic diseases and affects about 10–30% of adults and up to 42% of children. The average age of onset of the disease is 10 years, and the largest number of cases are registered at the age of 13-19 years. Every third patient with AR is younger than 17 years old. Undoubtedly, this pathology has socio-economic consequences: AR is the cause of absences at school and at work, reduced work capacity and success, difficulties in communication and deterioration of quality life. Unfortunately, in most cases this pathology is not fully diagnosed - the ratio of detected and undetected cases of AR in Ukraine is 1:40-60. In addition, it is necessary to remember the well-known concept of the European Academy of Allergology and Clinical Immunology (EAASI) — ARIA (Allergy, Rhinitis and its Impact on Asthma) regarding the relationship between allergic rhinitis and bronchial asthma as " of single respiratory tracts: single respiratory system, single disease." 80% of children under 10 years of age with allergic rhinitis also have bronchial asthma.

Rhinoscopic criteria (according to direct rhinoscopy):

- swelling of the mucous membrane of the nasal septum;
- swelling of the lower and middle nasal concha;
- the mucous membrane of the nose is pale gray with a blue tint and a shiny surface with a marble and "spotted" pattern (Wojacek's symptom);
- the color of the mucous membrane of the nose is pale pink or bluish;
- swollen adenoid vegetations (according to reverse rhinoscopy);
- lack of vasoconstrictive effect from local application of adrenaline;

- eosinophilic-monocytic infiltration of the mucous membrane during biopsy.

X-ray criteria:

- thickening of the mucous membrane of the maxillary sinuses;
- possible parenchymal sinusitis.

Cytological criteria (according to the data of the examination of secretions from the nose): eosinophilia, possible basophilia.

Pollen conjunctivitis is characterized by itching of the eyes, eyelids, their hyperemia, photophobia and lacrimation. Clinical manifestations of the disease increase when the patient is in the air, in a field where there is a high concentration of causative allergens. Conjunctivitis can be complicated by corneal erosions and keratitis. Fever is possible only in the case of sinusitis or otitis media. 10-30% of patients with a severe course of pollinosis may develop pollen bronchial asthma.

Rare clinical manifestations:

- allergic pharyngitis, otitis (especially in children);
- dermatological syndrome: urticaria, angioedema on open parts of the body (in 4–8% of patients);
- gastrointestinal syndrome: nausea, vomiting, abdominal pain, diarrhea;
- brain phenomena according to the type of Ménière's syndrome, epileptic attacks;
- visceral lesions: lungs (eosinophilic infiltrate), liver, heart. In general, the period of exacerbation of pollinosis lasts from 1-2 to 5-6 months.

Diagnosis of pollinosis:

1. Allergological anamnesis collection.
2. Clinical symptoms (for hay fever, rhinoconjunctival syndrome, seasonality of exacerbations, increased symptoms in dry weather and weakening in wet weather are more characteristic).
3. Provocative and skin-allergy tests using a set of allergens during the remission period to identify individual causative allergens (the main diagnostic criterion).
4. Determination of the total level of IgE (marker of atopy).
5. Determination of the level of specific IgE: if it is impossible to perform provocation and skin tests in vivo; in children under 3 years of age; in the period of

exacerbation of the disease; in the presence of skin lesions (similar in importance to skin tests).

6. Cytological examination of a smear-print from the nasal mucosa (characteristic eosinophilia) and bacteriological examination of washings from the mucous membrane.

7. General blood test (possible eosinophilia).

8. X-ray examination of the nose and paranasal sinuses (for differential diagnosis with infectious sinusitis).

Additional instrumental studies:

– rhinomanometry;

- smell research using the "strip method".

Treatment.Modern treatment of pollinosis in children according to the international program involves:

- elimination therapy (stopping contact with the causative allergen); adherence to a hypoallergenic, elimination diet, implementation of elimination measures (elimination of contact with house dust, epidermal allergens, drug and food allergens);

- specific immunotherapy (SIT);

- pharmacotherapy;

- education of patients and their parents.

SIT (specific hyposensitization, specific allergy vaccination) should be carried out by an allergist in the pre-season period. This is the only method of anti-allergic protection that affects all chains of the pathogenesis of allergic diseases. The method is based on the introduction into the patient's body of an allergen in gradually increasing doses, to which he has an increased sensitivity, with the aim of synthesizing "blocking" protective antibodies of the IgG class. This contributes to a significant reduction in the severity or complete disappearance of the symptoms of the disease. In the absence of contraindications, SIT should be performed by an allergist in the allergology department (office). Treatment is carried out using an accelerated course of SIT for 10–14 days or using a long-term

classical method. They use modern forms of SIT with the allergen in the form of a dragee. It is desirable that the total course of SIT, including maintenance therapy, lasts 3-5 years. The use of SIT requires great mutual understanding between the doctor and the patient or the parents of a sick child, which is a prerequisite for effective treatment of the disease. According to the literature, the effectiveness of SIT is 80-90%.

The main principle of drug therapy for pollinosis is a step-by-step approach to prescribing drugs depending on the severity of the disease.

A stepwise approach to the treatment of seasonal pollinosis:

1. Sodium cromoglycate (intranasal, intraconjunctival)
2. Non-sedating H1-histamine blockers (oral)
3. Inhaled glucocorticosteroids (GCs) of local action.

Preparations of cromoglycic acid: Lomuzol, Cromohexal, Cromoglin, etc. 1 - 2 doses 2 - 4 times a day in long courses up to 3 - 6 months (basic therapy). Antihistamine systemic drugs of the second (loratadine, cetirizine) and third generation (desloratadine, fexofenadine, Nixar) are taken once a day for 10–28 days or longer, and with a preventive purpose - a long course (basic therapy).

Among antihistamines, preference should be given to drugs of the second and especially the third generation, which have a number of advantages:

- quick onset of action;
- sufficient duration of the effect (up to 24 hours);
- high specificity and affinity to H1 receptors;
- inability to block receptors of other types (especially cholinergic);
- do not penetrate through the blood-brain barrier; - well absorbed in the digestive tract, absence of absorption connection with food intake;
- do not provoke the phenomenon of tachyphylaxis;
- a small frequency of peripheral side effects (dry mouth, stomach pain).

Vasoconstrictor drugs of local action (xylometazoline, oxymetazoline, phenylephrine) are used carefully in a short course of up to 6-7 days, taking into account their side effects. It is more appropriate to prescribe drugs for irrigation

therapy such as Salin, Marimer, Aqua Maris, Quicks (symptomatic therapy) in the form of isotonic or hypertonic solutions of sodium chloride.

Glucocorticoids in the form of inhalations are used in case of ineffectiveness of the above drugs - preference is given to the most modern ones - Flix, Nasonex.

TESTS

1. What allergens most often cause intermittent (seasonal) allergic rhinitis?

A. Fungal allergens

B. Epidermal allergens

C. Bacterial allergens

D. Plant - pollen allergens

2. What allergens are more likely to cause persistent (year-round) allergic rhinitis in children?

A. Allergens of house dust, house dust mites

B. Fungal allergens

C. Food allergens

D. All of the above are listed

3. Typical symptoms of allergic rhinitis are::

A. Pale bluish swelling of the tissues of the nasal mucosa, serous-watery secretions from the nasal passages.

B. Red swelling of the tissues of the nasal mucosa, purulent discharge from the nasal passages

C. Absence of swelling of the nasal mucous membranes, purulent secretions from the nasal passages

Г. Red swelling of the tissues of the mucous membrane of the nose, serous-watery secretions from the nasal passages

4. What data or examination results are crucial for suspecting the presence of allergic bronchitis in children?

A. Detection of the obstructive type of breathing according to the spirometry

B. Positive data of an allergic history

C. Detection of humoral immunodeficiency by a decrease in the level IgG, IgM

D. Detection of cellular immunodeficiency at a reduced level CD3+, CD4+, CD8+-lymphocytes

5. What etiological factor causes pollinosis in children?

A. Household allergens

B. Fungal allergens

C. Epidermal allergens

D. Plant - pollen allergens

6. What is the most common disease manifested by hay fever (pollinosis) in children?

A. Allergic laryngitis

B. Allergic bronchitis

C. Allergic rhinitis

D. Allergic rhinoconjunctivitis

7. In what weather are particularly pronounced clinical signs of polynosis?

A. In windless weather during a walk in the yard

B. In dry, sunny and windy weather during a walk in the yard

C. In cold, winter weather while walking in the yard

D. In rainy and windy weather during a walk in the yard

8. Name the duration of clinical manifestations of pollinosis:

A. 1 - 2 months

B. 3 - 4 months

C. 4 - 5 months

D. 5 - 6 months

9. The most likely etiological allergic factor of autumn hay fever is usually:

A. Ragweed.

B. Spores of Penicillium, Mucor, Cladosporium, Candida fungi

C. House dust mites, barn mites

D. Cat and dog fur, horse scales and hair.

10. What immunological indicator confirms the diagnosis of allergic rhinitis ?

A. Increased level of total IgE

B. Increased level of precipitating antibodies

C. Increased level of specific IgG, IgM

D. Increased level of secretory IgA

Answers: 1. - D, 2. - D, 3. - A, 4. - B, 5. - D, 6.- D, 7.- B, 8. - A, 9. - A, 10. - B.

Clinical task 1. A 10-year-old child came to the polyclinic for a control examination and recommendations. He has been suffering from hay fever for 3 years, exacerbations of the disease occur mostly in the spring and summer period. According to the results of allergy tests: increased sensitivity to poplar fluff, field grasses. What is the most likely recommendation of the doctor to change the natural course of the disease?

Answer standard. To change the natural course of the disease, it is recommended to carry out specific hyposensitization outside the period of exacerbation of the disease.

Clinical task 2. The child is 7 years old, the parents observe abundant discharge from the nose, sneezing for the second year in a row in the summer. The disease begins with the words of the parents after going on vacation, to nature. On examination, nasal congestion, watery transparent secretions, periodic sneezing, itching in the nose and throat are noted. Breathing rate - 26 per 1 minute. During auscultation: breathing is vesicular, exhalation is not prolonged, there are no wheezes in the lungs. Heart sounds are moderately muffled, rhythmic, pulse - 110 per 1 minute. No deviations were found in other systems. Physical development according to age.

What is the most likely diagnosis? Prescribe treatment taking into account the possibilities of drug therapy.

Answer standard. Pollinosis. Intermittent allergic rhinitis. Treatment. Desloratadine 5 mg - 1 time a day lasted for the flowering period. Irrigation nasal therapy (Aqua - Maris, No - Salt, Psyk, Kwiks) 3 - 4 times a day.

Congenital heart defects

Epidemiology. Congenital heart defects are detected in 5-8 children per 1000 live births.

Etiology. It is extremely difficult to establish the specific reasons for the formation of the CHD in a child. The formation of CHD is associated with diseases of the chromosomal apparatus, adverse effects of environmental factors, intrauterine infections, adverse effects during pregnancy (smoking, alcohol, drugs, medications, maternal diseases). It is known that syndromic causes of CHD are found in 6-40% of children, and the monogenic nature of CHD is found in 8%.

Pathogenesis. The above-mentioned factors in the period of intrauterine development lead to the formation of pathological structures of the heart, causing the formation of various defects: lack of closure of fetal communications, hypertrophy or aplasia of the ventricles, defects of the valve apparatus, unnatural narrowing of blood vessels. In most cases, until the moment of birth, hemodynamics does not suffer significantly, and its decompensation appears at any time after birth.

Classification based on Marder – 2005.

I. According to the state of pulmonary blood flow.

1. Congenital heart defects with enrichment of the small circle of blood circulation:

Atrial septal defect;

Ventricular septal defect;

Patent ductus arteriosus;

2. Congenital heart defects with impoverishment of the small circle of blood circulation:

Tetrad of Fallot;

Isolated pulmonary artery stenosis;

Transposition of the great vessels;

3. Defects with normal pulmonary blood flow:

Aortic stenosis;

Aortic coarctation

II. According to the degree of hemodynamic disturbance.

Without violation;

With a moderate violation of hemodynamics

With a significant violation of hemodynamics

III. According to the clinical course.

Phase of primary adaptation;

Phase of relative adaptation;

Phase of terminal adaptation.

Congenital heart defects with enrichment of the small circle of blood circulation.

Ventricular septal defect (VSD) - congenital septal defect resulting from underdevelopment of the interventricular septum at its different levels, resulting in the formation of a connection between the left and right ventricles.

Epidemiology. VSD occurs in 1.5 - 3.5 cases per 1000 full-term newborns and in 4.5-7 cases in premature newborns. In children with congenital heart defects, the frequency of VSD is 15 - 20%. Perimembranous (in the membranous part of the septum) defects make up approximately 80% of all VSD, muscle defects make up 5 - 20% of isolated VSD.

Etiology. VSD has a multifactorial origin. Genetic risk factors: high frequency of cardiac anomalies in the genealogical history of parents. Maternal risk factors: diabetes, phenylketonuria, maternal alcoholism.

The mechanism of VSD formation is not studied enough. It is believed that the perimembranous defect occurs due to the disturbed fusion of the heart departments as a result of a transient violation of blood circulation that develops in the septum; muscle defects are the result of cell death in the septum.

Hemodynamics depends on the size of VSD, the number, location, duration of the disease, the degree of pulmonary hypertension, the degree of compensatory hypertrophy of the myocardium of the right and left ventricles of the heart, the ratio of vascular resistances of the large and small circles of blood circulation. After birth, with small defects (0.2-1.0 cm) and physiologically high resistance of the vessels of the small blood circulation, the discharge of blood from the left to the right is small and is carried out only in systole, the pulmonary blood flow exceeds the systemic blood flow only by 1.2-1.5 times. Diastolic overload of the left ventricle causes its hypertrophy. With medium and large defects (2-3 cm), the amount of blood loss depends on the difference in resistance in the large and small circles of blood circulation. Hypertrophy of the left ventricle usually develops. An increase in pressure in the left atrium and pulmonary veins due to the action of neurohumoral mechanisms (Kitaev's reflex) leads to spasm of the pulmonary vessels, which protects the lungs from "flooding" them with blood. An increase in pulmonary vascular resistance causes systolic overload of the right ventricle. The right ventricle and later the right atrium hypertrophy.

Anamnesis, clinic. In the case of small defects of VSD, the child's physical development does not disturb. A systolic murmur is heard in intercurrent diseases or accidentally in the III-IV intercostal space draining from the sternum, radiating to the right and left of the sternum and in the back. With medium and large defects, children have physical developmental delay, hypotrophy occurs, most patients have a history of prolonged and recurrent pneumonia, bronchitis. From the first

weeks of life, signs of insufficient blood circulation are noted: difficulty in sucking milk, shortness of breath, pallor, sweating (due to the release of retained fluid through the skin), perioral cyanosis. Children with a large VSD (due to hypervolemia of a small circulatory circle) have constant shortness of breath, which worsens when feeding, crying, and changing body position. During the physical examination, a heart "hump" is visually determined due to an increase in the right ventricle (Davis' chest). The apical impulse is diffused, intensified, a pathological heart impulse is determined, in 2/3 of patients - systolic tremor in the 3-4th intercostal space to the left of the sternum, which indicates the discharge of blood into the right ventricle. The limits of relative heart dullness are extended to both sides, especially to the left. A coarse, intense systolic murmur is heard, maximally in the 3-4th intercostal space to the left of the sternum and at the xiphoid process. Noise radiation to the left and right from the sternum and back is characteristic. Spontaneous closure of the defect is often observed in children under one year and much less often after two years. Separate types of ventricular septal defect are called Tolochynov-Roger disease and Eisenmenger symptom complex.

Tolochynov-Roger's disease is a small defect in the muscular part of the interventricular septum, often passes without hemodynamic disturbances. The development of such children is the same as that of healthy children. Only the presence of auscultatory data - a coarse systolic murmur with an epicenter in the fourth - fifth intercostal space to the left of the sternum allows the diagnosis of CHD to be established.

Eisenmenger's symptom complex is a high VSD defect with rapid progressive development of pulmonary hypertension with rapid sclerosis. The child first develops crimson, then blue or purple cyanosis of the lips, nail phalanges with the development of "drumsticks". The systolic murmur gradually disappears, but the accent of the II tone on the pulmonary artery increases. Mixed shortness of

breath increases, the child's physical activity decreases, nosebleeds and heart pain appear.

The diagnosis of VSD is based on the detection of organic murmur at most in the 3-4th intercostal space to the left of the sternum and in the xiphoid process, signs of circulatory insufficiency, cardiomegaly, and the presence of recurrent bronchopulmonary diseases.

Examination. ECG. Deviation of the electric axis to the right, signs of combined hypertrophy of the ventricles are determined. Radiologically, hypervolemia in the small circle of blood circulation is detected, an increase in the size of the heart at the expense of both ventricles and atria is noted, there is an exploding arc of the pulmonary artery along the left contour of the heart.

Doppler echocardiography. Two-dimensional (2D) doppler echocardiography with color mapping allows to determine the size and localization of VSD. With the help of dopplerography, hemodynamic indicators are additionally obtained: pressure in the right ventricle, pressure in the pulmonary artery, interventricular gradient.

Differential diagnosis of VSD must be done with defects that occur with enrichment of the small circle of blood circulation.

Treatment. Circulatory insufficiency management is carried out according to general principles. Indications for surgical correction of defects are heart failure.

Atrial septal defect (ASD) - congenital abnormal connection between the two atria.

Epidemiology. The frequency of ASD is 0.1 - 0.53/1000 newborns. Female patients predominate (2:1). Among all congenital heart defects, ASD is found in 10 - 12%. Depending on the nature and degree of underdevelopment of the primary and secondary atrial septa and endocardial ridges, primary and secondary defects and the complete absence of an atrial septum are distinguished (single common atrium, three-chambered heart).

Etiology. Primary ASD occurs due to the underdevelopment of the primary interatrial septum and preservation of the primary connection between the atria. This is, as a rule, a large defect (1/3-1/2 part of the septum), which is localized in the lower part of the septum. ASD is most often combined with Down syndrome (trisomy 21). Secondary ASD occurs as a result of underdevelopment of the secondary atrial septum and in most cases is located in the center of the atrial septum. This type of defect is often combined with valvular stenosis of the pulmonary artery. Isolated secondary ASD is often inherited in an autosomal dominant pattern. Combinations of primary and secondary ASDs are possible. In some cases, a single atrium is formed.

Hemodynamics. The basis of the hemodynamic disturbance is the discharge of blood due to a left-right defect, due to greater pressure in the left atrium than in the right, this causes volume overload of the right ventricle in diastole and hypervolemia of the small circulatory circle. In young children, the direction of the shunt can easily change due to a transient increase in pressure in the right atrium (with diseases of the respiratory system, screaming, sucking) with the occurrence of transient cyanosis. Long-term volumetric overload of the right chambers of the heart leads to their dilation and the gradual development of moderate hypertrophy of the right ventricle.

With ASD, pulmonary hypertension develops quite rarely, since there is no direct influence of the pumping effect of the left ventricle.

Anamnesis, clinic. In children with a small primary defect of the atrial septum, the defect is asymptomatic and is detected only by the presence of murmur. The children's physical development is normal, they play sports, and tolerate stress satisfactorily. In the second decade of life, shortness of breath after physical exertion, increased fatigue, dizziness, fainting may appear. Pulmonary hypertension and heart failure develop before the age of 20. In infants, a small systolic ejection murmur is heard to the left of the sternum. At an older age, the splitting of the II tone on the pulmonary artery and a small systolic murmur in the

second-third intercostal space to the left of the sternum are heard. The murmur is associated with functional stenosis of the pulmonary artery, arising from increased blood flow with an unchanged fibrous ring of the pulmonary artery. With a large or medium-sized primary defect, the manifestation of the defect is possible both in the newborn period (dyspnea, tachycardia) and at the age of 1-2 years. Paleness, hypotrophy, and a moderate delay in physical development are noted. Characteristic tendency to frequent acute respiratory infections, bronchitis and pneumonia. Examination reveals cardiomegaly with expansion of the borders of relative cardiac dullness to the right and up, less often to the left. Possible deformation of the chest in the form of a central cardiac hump due to dilatation and hypertrophy of the right ventricle. Auscultally, the I tone is amplified, the II tone is amplified and split above the pulmonary artery, a systolic murmur of medium intensity and duration is heard in the second-third intercostal space to the left of the sternum. In older children, a short diastolic murmur of relative stenosis of the tricuspid valve (Coombs murmur) may additionally be heard due to increased blood flow through the tricuspid valve.

Children with secondary ASD do not have clinical symptoms. Teenagers have a reduced tolerance to physical exertion. Primary ASD is not afraid of spontaneous closure. Spontaneous closure of a small (often up to 3 mm) secondary defect of the interatrial opening occurs up to one year.

The diagnosis of ASD is established in the presence of cardiomegaly, the presence of a systolic murmur of medium intensity and duration in the second - third intercostal space to the left of the sternum. Frequent bronchopulmonary and cold diseases in the anamnesis.

Laboratory studies. ECG. The electrical axis of the heart is deviated to the right, signs of right ventricular hypertrophy, right atrial hypertrophy; 2/3 of patients have incomplete blockade of the right leg of the bundle of His. Radiologically, the strengthening of the lung pattern is revealed. With large defects

in the direct projection, the heart is enlarged due to the right ventricle, the waist of the heart is flattened due to the bulging of the pulmonary artery.

Doppler echocardiography. Two-dimensional (2D) doppler echocardiography detects an interruption of the echo signal in the area of the atrial septum, the pressure gradient between the atria; color Doppler mapping determines the direction of the shunt.

Differential diagnosis should be carried out with isolated stenosis of the pulmonary artery, triad of Fallot, VSD, abnormal drainage of the pulmonary veins, abnormality of the development of Ebstein's tricuspid valve.

The open oval window (OOW) is an interatrial communication, through it during fetal development blood from the inferior vena cava is directed directly into the left atrium. At birth, the pressure in the left atrium is higher than in the right, the valve of the oval window is pressed against the oval window and its physiological closure occurs. By the end of the first month of life, OOW with underdevelopment of the valve of the foramen ovale or its defect persists in 7 - 3%, up to a year in 2%, OOW is found in 15% of adult patients.

A very common question is whether there are differences between an open oval window OOW and ASD? Currently, pediatric cardiologists designate small atrial septal defects (up to 5 - 6 mm) localized in the fossa ovalis as OOW, and defects larger than 6 mm or in other localizations as ASD.

Treatment. Indications for surgical correction of defects: heart failure, retardation in physical development, lung diseases. A hemodynamic indication for surgery is a ratio of pulmonary to systemic blood flow of more than 2:1. With the effectiveness of conservative drug therapy, the operation can be postponed until 5-6 years of age. Secondary defects are closed by suturing; primary ASDs are closed with a patch of autopericardium or synthetic tissue using thoracotomy and artificial blood circulation. Endovascular repair of the defect with the help of occluders is possible

only with secondary ASD up to 25-40 mm in size, around which there is a border of a septum up to 10 mm wide.

Complications of ASD appear at the sclerotic stage of pulmonary hypertension in the form of Eisenmenger syndrome (pulmonary hypertension, with dilatation of the trunk of the pulmonary artery and a change in the shunt from right to left, with the appearance of permanent cyanosis). This condition is characterized by a combination of an enlarged trunk of the pulmonary artery and the formation of a relative functional stenosis of the pulmonary valve against the background of an increased stroke volume of the right ventricle. In the later stages of ASD with Eisenmenger syndrome, dystrophy and sclerosis of the right ventricular myocardium occurs, which leads to the development of first right ventricular and then total heart failure. Complications after surgery: acute heart failure, heart rhythm disturbances, infectious endocarditis, residual shunt. Possible complications with catheter occlusion of the defect: perforation of the vessel wall, vessel occlusion, incomplete closure of the defect, infectious endocarditis.

Patent ductus arteriosus (PDA) - the presence of an abnormal vascular duct in which blood from the aorta enters the pulmonary artery.

Epidemiology. The frequency of PDA is 0.14 - 0.3/1000 live births. Among all congenital heart defects, PDA occurs in 6-7% of cases. PDA mostly affects girls. In 5 - 10%, PDA is combined with other CHD. In some cases, it is considered as a compensating defect (with tetrad of Fallot, pulmonary artery stenosis, coarctation of the aorta, aortic atresia), in other cases as a defect that increases hemodynamic disturbances: with postductal coarctation of the aorta, VSD, ASD.

Etiology. Normally, the PDA provides fetal blood circulation, usually the duct departs from the arch of the aorta, distal to the exit of the left subclavian artery and flows into the pulmonary artery in the area of its bifurcation or near the exit of the left pulmonary artery. The duct closes in the first 2 weeks of life. If this

does not happen, they talk about a heart defect. In premature babies, the formation of PDA is associated with the immaturity of ductal tissue and an increased concentration of prostaglandins. In full-term children, this is likely due to chronic hypoxia, intrauterine growth retardation.

Hemodynamics. After the first inhalation and the opening of the pulmonary vessels, the pressure in the pulmonary artery quickly decreases with a simultaneous increase in the pressure in the large blood circulation. The right-left blood flow is sharply reduced. As total pulmonary resistance falls, bidirectional and then left-right blood flow through the PDA occurs. In the first months of life, the left-right reset is carried out only in systole, later, when the diastolic pressure in the aorta exceeds the pressure in the pulmonary artery, the left-right shunt acquires a permanent systolic-diastolic character, which leads to pulmonary hypervolemia to one degree or another. With a long and narrow duct, blood loss is small, hypervolemia of the small circulatory circle is moderate. A moderate dilatation of the left ventricle develops, pulmonary hypertension has been absent for a long time. With a short and wide duct, blood loss is significant, which leads to pronounced hypervolemia of the small blood circulation. "Ballast" volume of blood, circulating in the small circle of blood circulation, returning to the left parts of the heart, causes volume overload and dilatation of the left atrium, left ventricle and expansion of the ascending aorta. The effective stroke volume, which enters the periphery of a large circle of blood circulation, is reduced by the amount of discharge into the pulmonary artery. The blood flow in the aorta and the large circle of blood circulation has a "pulsating" character, reminiscent of blood flow in aortic valve insufficiency. In carotid arteries, blood flow is normal in systole, but in diastole it significantly decreases, "diastolic theft" of cerebral blood flow occurs.

Anamnesis, clinic. With narrow and long PDA, patients develop normally until 5-6 years of age, when complaints and clinical manifestation of the defect first appear. Their only manifestation is a continuous systolodiastolic heart murmur, with a maximum on the pulmonary artery. In young children, the noise

can be only moderate systolic. With a short and wide duct, clinical symptoms appear in the first months of life. There is a constant pallor of the skin, as a result of the syndrome of "robbing" of a large circle of blood circulation. Transient cyanosis appears when straining, coughing, screaming, or sucking. Children retard in weight and physical development, they often develop recurrent bronchitis and pneumonia. After 3-5 years, fainting, pain in the heart, palpitations, shortness of breath after physical exertion are observed. The area of the heart can be deformed in the form of a left-sided cardiac hump, the apical impulse is increased, diffuse. In some children, systolic or systolodiastolic tremors are palpable, the limits of relative cardiac dullness are extended to the left and up. During auscultation, the first tone is loud, the second tone can be blocked in intensity by a characteristic continuous intense systolo-diastolic "machine" noise. The maximum noise is heard at the base of the heart, is conducted along the left edge of the sternum, radiates to the back between the upper corner of the shoulder blade and the spine. With a long-term existence of a defect with a large reset, a diastole murmur may occur at the apex of the heart due to the existence of mitral stenosis. The peripheral pulse is high, jumping. Systolic blood pressure is normal or elevated due to a large ejection into the aorta.

Laboratory studies. ECG. Deviation of the electrical axis to the left, signs of overload of the left ventricle, enlargement of the left ventricle, disturbance of exchange processes in the myocardium. X-ray: there is an increase in the pulmonary pattern corresponding to the amount of arteriovenous discharge, expansion or bulging of the trunk of the pulmonary artery. The waist of the heart is smoothed, the left heart sections are enlarged.

Doppler echocardiography. Two-dimensional (2D) doppler echocardiography. When scanning from a high parasternal or suprasternal access, the duct and the direction of the shunt are visualized. The size of the shunt is judged by indirect signs.

Differential diagnosis is carried out with VSD, incomplete atrioventricular communication, in older children - with combined aortic malformation.

Treatment. Specific conservative therapy is possible only in premature babies, by intravenous administration of three doses in 48 hours of prostaglandin synthesis inhibitor (indomethacin at a dose of 0.2, 0.1, 0.1 mg/kg). If there is no effect after 24 hours, an additional three-time administration of the drug at a dose of 0.1 mg/kg with an interval of 24 hours is possible. The effectiveness of treatment is 70 - 80%. The indication for surgical treatment is the presence of PDA. In small children, vessels are clipped. Vascular ligation is performed in older children. Optimal terms of operation - from 6 - 12 months to 3 - 5 years. Postoperative mortality is less than 1%. The removal of the defect is also carried out by the endovascular method - with the help of special spirals (with a duct diameter of up to 3 mm), for larger openings (up to 6 mm) several spirals or a special occluder are used.

Complications of PDA. Long-term overloading of the small circle of blood circulation leads to the formation of pulmonary hypertension, which contributes to the formation of hypertrophy of the right ventricle and right atrium, the change of the shunt to right-left, arterial hypoxemia, chronic right ventricular or total heart failure. With small ducts, the development of bacterial endocarditis is possible, such a complication as a PDA aneurysm with its rupture, thrombosis or infection rarely develops. Postoperative complications: hemorrhages, vascular damage, injury to the recurrent laryngeal and phrenic nerves, infectious endocarditis, congestive heart failure.

Prognosis. After surgical correction of the defect, in the absence of complications, the development of such children is normal. In premature babies with large PDA.

Congenital heart defects with impoverishment of the small circle of blood circulation

Impairment of the small circle of blood circulation is the result of an obstruction in the path of blood outflow from the right ventricle. Clinical - the hemodynamic picture is formed not so much by the character as by the degree of narrowing of the duct.

Fallot's disease (triad, tetrad, pentad). This defect also refers to blue defects of the heart. The most common variant is the tetrode of Fallot (stenosis of the pulmonary artery, high VSD, transposition of the aorta to the right, hypertrophy of the right ventricle).

Hemodynamic disturbances are caused by a decrease in blood flow to the small circle of blood circulation due to stenosis of the pulmonary artery, and venous blood flows into the large circle due to the VSD and the right-shifted aorta. A combination of such defects with ASD is also observed. This variant of the defect was called the pentad of Fallot. If there is no VSD when narrowing the outflow tract from the right ventricle, but there is an open oval window or ASD, the defect is called the triad of Fallot.

Clinic. Cyanosis is the main feature of tetrad of Fallot. The degree of cyanosis and the time of its appearance depend on the severity of pulmonary artery stenosis. In children of the first days of life, only severe forms of the defect are diagnosed based on the sign of cyanosis - the "extreme" form of the tetrad of Fallot. It is mainly characterized by the gradual development of cyanosis (up to 3 months - 1 year), which has different shades (from light blue to blue-raspberry or cast-iron blue): first there is cyanosis of the lips, then mucous membranes, fingertips, facial skin, limbs and trunk. Cyanosis increases as the child's activity increases. "Drumsticks" and "watch glasses" develop early. A constant symptom is shortness of breath of the type of dyspnoea (deep arrhythmic breathing without a pronounced increase in respiratory rate), which decreases at rest and increases sharply with the slightest physical exertion. A delay in physical development gradually develops. Rough systolic murmur along the left edge of the sternum is heard practically from birth. A formidable clinical symptom in tetrad of Fallot,

which determines the severity of the patients' condition, is suffocation - cyanotic attacks. They appear, as a rule, at the age of 6 to 24 months against the background of absolute or relative anemia. The pathogenesis of the attack is associated with a sharp spasm of the infundibular part of the right ventricle, as a result of which all venous blood enters the aorta, causing a sharp hypoxia of the central nervous system. Blood oxygen saturation during an attack drops to 35%. At the same time, the intensity of the noise decreases sharply until it completely disappears. The child becomes restless, the facial expression is frightened, the pupils are dilated, shortness of breath and cyanosis increase, the extremities are cold, then loss of consciousness, convulsions, the development of a hypoxic coma and death are possible. Attacks vary in severity and duration (from 10-15 seconds to 2-3 minutes). In the post-attack period, patients remain lethargic and adynamic for a long time. Sometimes the development of hemiparesis and severe forms of impaired cerebral circulation is noted. By 4-6 years, the frequency and intensity of attacks significantly decrease or disappear. This is connected with the development of collaterals, through which there is a more or less adequate blood supply to the lungs.

Depending on the characteristics of the clinic, three phases of the course of the disease are distinguished: I phase - relative well-being (from 0 to 6 months), when the patient's condition is relatively satisfactory, there is no lag in physical development; II phase - shortness of breath and cyanotic attacks (6 - 24 months), which is characterized by a large number of brain complications and deaths; III phase - transitional, when the clinical picture of the defect begins to take on adult features.

Laboratory data. X-ray changes - the vascular pattern is depleted, the heart is small, often in the form of a boot with a pronounced waist. The ECG reveals a shift of the electrical axis to the right, hypertrophy of the right atrium and right ventricle.

Treatment. The basis of treatment of "cyanotic" defects is oxygen therapy, correction of metabolic acidosis and medication effect on the spasm of the initial sections of the pulmonary artery. Treatment of a hypoxemic attack - without fainting - give the child a knee-elbow position, oxygen, prescribe sedative therapy with morphine, promedol, in the absence of an effect, intravenous obzidan slowly. If there is a seizure, sodium oxybutyrate is prescribed, which is also an antihypoxant. Cardiac glycosides and diuretics are contraindicated in children with such attacks. Operative treatment is carried out in two stages: in the first palliative stage, the amount of blood entering the small circle of blood circulation is increased, radical surgery is carried out after 2 - 6 months.

The prognosis without surgical intervention is unfavorable, after surgery it is positive.

Congenital heart defects with normal pulmonary blood flow

Coarctation of the aorta. Coarctation of the aorta accounts for 7.5% of all birth defects in newborns and early childhood. The defect occurs 2-2.5 times more often in men than in women. 26% of children have other severe extracardiac developmental anomalies, 14% of which cannot be treated.

Clinically, the disease is manifested by an increase in blood pressure in the arteries of the upper half of the body and a decrease in it in the arteries of the lower extremities. With a rather pronounced narrowing, there is pulsation in the head, headache, less often nausea, vomiting, visual disturbances. As a result of the lack of blood supply to the lower half of the body, there is numbness of the lower limbs, heaviness, weakness when walking, and a decrease in blood pressure when measured on the legs. In this regard, in the case of hypertension of unclear etiology, it is necessary to measure blood pressure in the arms and legs. Usually, at the same time sharply expressed signs of hypertrophy and expansion of the left ventricle, a relatively small systolic noise in the second-fourth intercostal space near the edge of the sternum and behind between the shoulder blades are determined. The first sign in children may be the absence (weakness) of pulsation

on the femoral artery (in the inguinal fossa). Coarctation of the aorta can also be indicated by the presence of collaterals in the form of enlarged and visible pulsating intercostal arteries. There may also be complications in the form of hemorrhages in the brain due to arterial hypertension.

Modern treatment of the disease - surgical, conservative therapy only eases the course of the disease, but does not stop its development and does not eliminate the causes of the disease.

TESTS

1. What defects are related to defects of the heart with an enriched small circle of blood circulation

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Isolated pulmonary artery stenosis

D. Atrial septal defect

E. Transposition of main vessels

2.What defects refer to defects of the heart with a normal blood circulation

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Isolated pulmonary artery stenosis

D. Interventricular septal defect

E. Transposition of main vessels

3. What defect is based on the impoverishment of a small circle of blood circulation

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Atrial septal defect

D. Interventricular septal defect

E. Transposition of main vessels

4. Tolochynov-Roger disease is observed with the following defect

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Isolated pulmonary artery stenosis

D. Interventricular septal defect

E. Transposition of main vessels

5. Eisenmenger's symptom complex is observed in the following heart disease

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Stenosis of the mouth of the aorta

D. Atrial septal defect

E. Transposition of main vessels

6. Name the most common complications of tetrad of Fallot

A. Endocarditis

B. Glomerulonephritis

C. Rheumatism

D. Pyelonephritis

E. Nosebleeds

7. Which defect is characterized by high blood pressure in the upper extremities

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Isolated pulmonary artery stenosis

D. Atrial septal defect

E. Transposition of main vessels

8. Which defect is characterized by low blood pressure in the upper extremities

A. Coarctation of the aorta

B. Tetrad of Fallot

C. Stenosis of the mouth of the aorta

D. Atrial septal defect

E. Transposition of main vessels

9. They are not used for the treatment of cyanotic - shortness of breath attack with tetrad of Fallot

A. Cardiac glycosides

B. Obzidan

C. Morphine

D. Seduksen

E. Oxygen

10. What position should the child be in for the treatment of a cyanotic - shortness of breath attack with tetrad of Fallot

A. Standing

B. Sitting

C. Lying on his back

D. Lying on his stomach

E. Knee-elbow position

Answers 1. - D , 2. - A, 3. - B, 4. - D , 5. - D, 6. - A, 7. - A, 8. - C, 9. - A, 10. - E

Clinical task 1. Child S., 3 years old, entered the clinic to find out the reasons for the child's frequent illnesses. When the child was admitted to the hospital, there were complaints of reduced appetite, fatigue, pronounced pallor. Medical history. The main reason for going to the doctor was episodes of repeated, poorly amenable bronchitis. The last episode of coughing was a week ago. At the same time, the child's mother drew attention to the appearance of cyanotic coloration of the skin of the face, mainly in the nasolabial triangle, which noticeably worsens during coughing attacks accompanied by shortness of breath. The child from the second pregnancy (the first ended in a medical abortion, complicated by endometritis), which occurred with preeclampsia in the first half. During pregnancy, the mother smoked, periodically drank alcoholic beverages, and in the eighth month of pregnancy she suffered an acute respiratory infection. Childbirth is urgent with the use of medical stimulation. Independent breathing and a weak cry appeared in the child only after suctioning mucus from the upper respiratory tract, Apgar score 6-7 points. Body weight at birth was 3050 g, body length - 52 cm. Due to asphyxia, she was put to the breast after 8 hours. The newborn period proceeded satisfactorily,

from the first weeks of life, the mother noted that the child tired quickly during feeding and did not gain weight well. He received breastfeeding in the form of irregular 8-10 feedings per day for up to 2.5 months. Next, the child was quickly transferred to artificial feeding, from 5 months - semolina porridge. In the first year of life, a child's static skills were formed with a delay. Indicators of neuropsychological development generally met the standards. At the age of 6 months, he first suffered an acute respiratory infection in the form of bronchitis. In the future, episodes of bronchitis occurred at least once every 3-4 months, and were more severe. In this regard, from the age of two, he was observed in the group often and for a long time sick children. The epidemiological history is calm. The material and living conditions of the family are satisfactory. Objectively, upon receipt, the condition was considered satisfactory. Behavior is adequate, consciousness is clear. The type of constitution is asthenic. Height - 94 cm, weight - 13.2 kg. Psycho - the emotional tone is unstable, the mood is moderately depressed. When examined, the skin is clean, pale, with normal moisture and elasticity. Attention is drawn to periorbital shadows and a slight cyanotic shade of the nasolabial triangle. Subcutaneous tissue is weakly developed, evenly distributed, there is no swelling and pastiness. Breathing is free, regular, with a frequency of 30 per minute. During percussion, a clear lung sound is determined, breathing is heard in the lungs with a slight hard tone, it is performed over all parts of the chest. The pulse rate at rest is 124 per minute, during physical exertion it increases to 140 per minute. With forced breathing, respiratory arrhythmia is clearly manifested in the form of increased heart rate during inhalation. The pulse is the same on both hands, its filling and tension are satisfactory. There is no visible pulsation over the projection of peripheral vessels. The chest above the heart region is not deformed, upon palpation after physical exertion, a weak sensation of local pulsation appears in the second intercostal space to the left of the sternum, the apical impulse is determined in the fourth - fifth intercostal space, high, of moderate strength, with an area of up to 6 cm, its outer edge is shifted 2 cm to the left of the midclavicular line. Percussion of the border of the heart: the

upper - along the second rib, the left - coincides with the outer border of the apical impulse, the right is determined almost 1 cm outward from the right parasternal line. During auscultation, both heart tones are determined: the I tone is well heard at the top of the heart, prevails over the P tone, while at the base of the heart, in the second intercostal space, the II tone is louder. It is split and is better heard on the left at the edge of the sternum, on the projection of the pulmonary artery valve. In the II-III intercostal space on the left side of the sternum, a moderate-intensity, but long-lasting systolic murmur of a blowing nature is heard, which is weakly defined over other points of auscultation of the heart, without going beyond its limits. Blood pressure on both arms is 95/55 mm Hg. Art. No pathological changes were detected in the digestive organs. The kidneys are not palpable, there is no dysuria, there is a narrowing of the foreskin, the tingling symptom is negative. There are no signs of fluid in the abdominal cavity. There are no symptoms of damage to the nervous and endocrine systems. During the examination, clearly expressed stigmas of dysembryogenesis attract attention: a low forehead, an increase in the distance between the eye slits, "gothic palate". Biochemical analysis of blood: total protein 72 g/l, albumins - 60%, α-1globulins - 4%, α-2-globulins - 7%, β-globulins - 12%, χ-globulins - 17%. ECG: sinus rhythm at 108 per minute, upright electrical position. The axis of the heart is not deviated, the voltage of the teeth is equal to 26 mm, in the second lead, the width of the high and pointed P wave and the P - Q interval are the same and equal to 0.11 sec; there are signs of increased electrical excitability of the right parts of the heart. Echocardiographic examination: a slight increase in the thickness of the wall of the right atrium and a slight increase in the cavity of the right ventricle. The anatomical structure of the valves is completely preserved, while the high-amplitude systolic-diastolic flow in the middle third of the atrial septum is clearly visible. X-ray examination of the organs of the chest: a picture of the lung fields with pronounced uniform enhancement of the vascular pattern of both lungs. Examination of the patient by an ENT doctor and an ophthalmologist did not reveal any pathology.

TASK:

1. Formulate a clinical diagnosis.

2. At what age is the most optimal time to perform surgery?

Answer standard. The child has a congenital heart defect - atrial septal defect, compensation phase, insufficiency of blood circulation Ist. Operative intervention is carried out up to 5 - 6 years.

Clinical task 2. Patient A., 2 years old, was admitted to the hospital as planned. It is known from the anamnesis that the child had diffuse cyanosis of the skin and visible mucous membranes since birth. From 3 months to now, she was in a child's home. Upon admission, the skin and visible mucous membranes are moderately cyanotic, acrocyanosis, fingers in the form of "drumsticks", nails - "hour glasses", deformation of the chest. Limits of relative heart dullness: right - 1 cm to the right of the right parasternal line, left - along the left axillary line, upper - II rib. Auscultation: tones are rhythmic, heart rate - 160 bpm, in the III intercostal space on the left edge of the sternum, a systolic murmur of medium intensity is heard, the accent of the second tone is in the II intercostal space on the left. BH - 40 in 1 minute, breathing is deep, loud. The liver protrudes 3.0 cm from under the costal margin. General blood analysis: hemoglobin - 148 g / l, erythrocytes - 4.92x1012 / l, leukocytes - 7.5x109 / l, p / i - 4%, z - 21%, e - 1%, l - 70%, m - 4%, ESR - 3 mm / h. General analysis of urine: color - light yellow, relative density - 1014, protein - absent, glucose absent, flat epithelium - a little, leukocytes - 0-1 in the field of view, erythrocytes absent. Biochemical analysis of blood: total protein 69 g / l, urea - 5.1 mmol / l, cholesterol - 3.3 mmol / l, potassium - 4.8 mmol / l, sodium - 143 mmol / l, phosphorus - 1.5 mmol / l, ALT - 23 units / l (normal - up to 40), AST - 19 units / l (normal - up to 40), seromucoid - 0.180 (normal - up to 0.200).

TASK:

1. Formulate a preliminary diagnosis

2. List additional examination methods to confirm the diagnosis.

Answer standard. Congenital heart disease - Fallot's disease, compensation phase, chronic insufficiency of blood circulation Ist. To confirm the diagnosis, it is necessary to carry out an x-ray of the chest organs, an ultrasound examination of the cardiovascular system.

Acute rheumatic fever

Rheumatism or acute rheumatic fever - is a general disease of the body with systemic inflammation of the connective tissue and involvement of the cardiovascular system and other organs in the process.

Relevance of rheumatism. For the first time, the concept of "rheumatism" was introduced into medical practice in 1635 by Ballonius. This disease was described in detail in 1835 independently by doctors Sokolsky and Buyot. More than 70% of cases of primary disease occur at the age of 8-15 years.

Etiology. Etiology of rheumatism - beta-hemolytic streptococcus group A (scarlet fever, pharyngitis, sore throat, infectious skin lesion-impetigo).

Streptococcal etiology of rheumatism: clinical symptoms of rheumatism appear 2-3 weeks after a pharyngeal infection caused by beta-hemolytic streptococcus group A. Streptococci are found in 65% of patients with rheumatism in the oral cavity, and in the blood in the active phase of the blood of patients with rheumatism antibodies to streptococcus in very high titers. A sharp decrease in the incidence and recurrence of rheumatism after bicillin prophylaxis.

Pathogenesis. The main links of pathogenesis include: I - allergic reaction of the immediate type accompanies the formation of anti-streptococcal antibodies in response to a streptococcal infection, followed by the formation of circulating immune complexes, which are fixed at the level of blood vessels and cause damage to the connective tissue of the heart, kidneys, joints, and skin. II - immunocomplex and autocomplex reactions take place under the influence of M-protein (its affinity with heart tissues causes damage to the pericardium and dysfunction of the pericardium) III - allergic reaction of the delayed type of the disease to the formation of a clan of sensitized lymphocytes, there is fixation of antibodies to the endomyocardium and heart inflammation is damaged.

Classification. Classification of rheumatism: phase: active - activity 1, 2, 3 degrees - inactive (rheumatic myocardiosclerosis, heart disease). Clinical and anatomical characteristics of heart lesions: primary rheumatic carditis, reversible rheumatic carditis (without heart disease, with valvular disease), acute rheumatic fever without cardiac changes.

Clinical and anatomical characteristics of lesions of other organs and systems: polyarthritis - serositis (pleurisy, peritonitis, abdominal syndrome). Chorea Encephalitis. Meningoencephalitis. Cerebral vasculitis. Neuropsychiatric disorders. Vasculitis Nephritis. Hepatitis. Pneumonia. Skin lesions. Iritis and Iridocyclitis. Thyroiditis.

Character of the course of acute rheumatic fever: Acute. Subacute Protracted. Continuously recurring. Latent.

Functional characteristics of blood circulation: H_0-no circulatory insufficiency, no objective and subjective disorders of blood circulation H_1 - shortness of breath and tachycardia during physical exertion, at rest there are no H_{IIA}-congestion in the lungs, moderate liver enlargement, swelling of the feet until the end of the day H_{IIB}- significant enlargement of the liver, edema, ascites, but they are reversible and amenable to treatment H_{III} - acute disorder of hemodynamics, irreversible and not amenable to treatment.

The modified Jones criteria are categorized into Major and Minor criteria. These criteria are based on how specific the manifestation is to the diagnosis of acute rheumatic fever. In other words, a Major criterion is much more specific to acute rheumatic fever than the Minor criteria. Therefore, if a child that has two Major criteria, they can fulfill Jones criteria for the diagnosis, as long as they have some evidence of streptococcal disease. On the other hand, if there is evidence of only one Major criterion, they need two minor criteria to fulfill the diagnosis, along with evidence of streptococcal infection. Since the minor criteria are less specific for the diagnosis of acute rheumatic fever, you cannot make the diagnosis with

just minor criteria. The symptoms may be dampened by giving aspirin or other non-steroidal antiinflammatory medications too early, thus not allowing the manifestations to fully develop. Modified Jones Criteria (two majors or one major + two minors required). Major criteria: carditis, migrating polyarthritis, chorea, erythema marginatum, subcutaneous nodules. Minor criteria: fever, arthralgia, elevated acute phase reactant (CRP or ESR), prolonged PR interval (i.e., first degree AV block). Leukocytosis used to be a minor criterion, plus all must have evidence of streptococcal infection (positive ASO titer, Streptozyme, positive streptococcal throat culture).

Clinical manifestation . In children of preschool and junior school age, 2-3 weeks after angina, the temperature suddenly rises to febrile figures, symmetrical migrating pains appear in large joints (most often knee joints), signs of carditis (pericardial pain, shortness of breath, palpitations, etc.). In special cases, a monosyndromic course with predominance of signs of arthritis or carditis or chorea is observed. A gradual onset is typical for teenagers: after the clinical manifestations of angina subside, subfebrile temperature, arthralgias of large joints or only moderate signs of carditis appear. A repeated attack (relapse) of is provoked by beta-hemolytic streptococcus infection and is manifested mainly by the development of carditis.

The main clinical signs: endocarditis - unpleasant sensations in the area of the heart, palpitations, dizziness, pallor of the skin, pulsation of the neck vessels, tachycardia, weakened heart sounds, systolic murmur at the apex, myocarditis - pain in the area of the heart, shortness of breath, palpitations, pallor of the skin, mucous membranes, cyanosis of the lips, nasolabial triangle, tachycardia turns into bradycardia, heart borders are shifted to the left, heart sounds are weakened, especially the first, systolic murmur at the apex, - pericarditis - sharp pain in the heart region, shortness of breath, dry cough, cyanosis of the skin, forced child's pose , shifted borders of the heart, tones are weakened, - pancarditis - only in severe cases.

Features of rheumatic polyarthritis: Multiple lesions of large joints, less often - small ones. Symmetrical joint damage. Migratory, damage to the joints (inflammatory reactions appear and disappear very quickly). Absence of deformation or any functional changes in the affected joints. Rapid disappearance of manifestations against the background of the use of anti-inflammatory therapy.

Typical symptoms of chorea: Changes in the child's mental state (emotional instability, inattention, fatigue, passivity, deteriorating performance at school). Movement disorders (hyperkinesis). Dysarthria (slurred speech). Movement coordination disorders (handwriting disorders, inability to hold objects, sorting the table while eating, instability in Romberg's pose, negative finger-nose and knee-heel tests), muscle hypotonia . Other signs of rheumatism: ring-shaped erythema - pale pink rashes in the form of a thin ring-shaped rim with clear outer and less clear inner edges. In the center, the skin is not changed. Appears on the trunk, limbs, less often - on the legs, neck, face. Or it is not accompanied by any sensations, usually disappears without a trace. Rheumatic nodules: Painless nodules from 2 mm to 1 cm, rounded, dense, located in fascia, tendons, in the subcutaneous tissue. Localization - the extensor surface of the elbow, knee, metacarpal-phalangeal joints, the area of the ankles, spinous vertebrae. Within 1-2 months, they disappear without residual phenomena.

Minor criteria of rheumatism: clinical: fever, arthralgia, rheumatism in history. Laboratory: reactants of the acute phase (increased ESR, CRP, leukocytosis), prolongation of the P-R interval.

Diagnosis. Laboratory studies: inflammatory activity of the blood: increased ESR and positive CRP. Bacteriological examination: detection of BGSA in a throat swab. Serological studies: the titers of antistreptolysin-O, antistreptohyaluronidase, and antideoxyribonuclease are elevated or increasing in dynamics. Instrumental studies: ECG: Prolongation of the P-Q interval; Echocardiography: signs of mitral and/or aortic regurgitation; MRI of the head: picture of vasculitis of cerebral vessels.

Features of rheumatism in children: a more difficult course of the process due to a pronounced exudative component of inflammation. Cardiac forms of rheumatism are most common. More frequent relapses of the disease. Rheumatic rash and rheumatic nodules are observed much more often. The presence of chorea, which adults do not have. More often, polyserositis is noted. Constant activity is preserved in the interrelapse period. The so-called "dry decompensation" with an enlarged liver without swelling of the legs, which occurs in adults, is characteristic. Children develop rheumatic pneumonia more often than adults. An acquired heart defect is formed more often.

Peculiarities of the regimen for acute rheumatic fever bed rest for 2-3 weeks or more, depending on the severity of carditis and the features of the course. Semi-bed rest - after eliminating the signs of carditis.

Features of the diet for acute rheumatic fever: rational, complete, vitaminized, easily digestible, high-calorie, limiting or reducing the amount of salt, fluid intake, additional appointment of foods rich in potassium (baked potatoes, raisins, dried apricots, prunes).

Etiotropic treatment of rheumatism: penicillin 100,000 units per 1 kg of body weight in 4 doses within 2 weeks. Bicillin-5 in a dose of 750,000 units for up to 30 kg of weight and 1,500,000 units for people weighing more than 30 kg. Cephalosporins or macrolides for penicillin intolerance. Pathogenetic treatment: acetylsalicylic acid, indomethacin, methindol, voltaren, orthofen, brufen, ibuprofen in age-related doses. Steroidal anti-inflammatory drugs: prednisone in a dose of 0.75 - 1-2 mg per kg of body weight for 2-3 weeks, dexamethasone. The dose is gradually reduced over several weeks. To normalize vascular permeability: ascorutin, ascorbic acid. Antihistamines: suprastin, diazolin, desloratidine, etc. To normalize metabolic processes in the heart muscle: panangin, asparkam, riboxin, cardiac glycosides (digoxin, etc.), B vitamins.

Prevention of rheumatism: primary - a complex of state, public and individual measures aimed at preventing the primary incidence of rheumatism. Secondary - a system of preventive measures aimed at preventing relapses and progression of the disease in people who have suffered rheumatism.

Primary prevention: effective treatment of acute streptococcal infections caused by group A streptococcus, prevention of their spread and reduction of contacts. Increasing the body's natural resistance and adaptation capabilities to adverse influences.

Secondary prevention: bicillin prophylaxis - regular intramuscular administration of Bicillin-5 of prolonged action: 750,000 units once every 3 weeks for children with a body weight of less than 30 kg and 1,500,000 units once every 4 weeks with a body weight of more than 30 kg for at least 5 years.

TESTS

1. The main importance in the etiology of rheumatism is:

A. Staphylococcus aureus

B. Beta - hemolytic streptococcus of group A

C. Adenovirus

D. Influenza virus

E. Coronavirus

2. Which does not belong to the big criteria of rheumatic fever

A. chorea

B. cardite

C. polyarthritis

D. arthralgia

E. annular rash

3. Which does not belong to the small criteria of rheumatic fever

A. abdominal pain

B. fever

C. rheumatic nodules

D. arthralgia

E. lengthening of the P-R interval

4. Myocarditis in rheumatic fever is characterized by the following laboratory parameters

A. leukopenia

B. slowed ESR

C. anemia, thrombocytopenia

D. leukocytosis

E. reticulocytosis

5. Rheumatic polyarthritis in children is characterized by damage to the joints of the following departments

A. of the lumbar spine

B. small joints of the limbs

C. large joints of the limbs

D. costosternal joints

E. of the cervical spine

6. Damage to the nervous system during rheumatism in children is manifested by the development of one of the conditions listed below

A. paresthesias

B. spasmophilia

C. eclampsia

D. chorea

E. seizures

7. Skin manifestations of rheumatic fever are characterized by:

A. annular erythema

B. acrocyanosis

C. jaundice

D. papular rash

E. vesicular rash

8. Rheumatic chorea in children manifests itself

A. fever

B. laryngospasm

C. convulsions

D. hypertonus

E. hyperkinesis

9. Laboratory indicators are uncharacteristic for rheumatic fever

A. leukocytosis

B. high sialic samples

C. positive CRP

D. accelerated ESR

E. leukopenia

10. Initial antibacterial therapy of acute rheumatic fever begins with:

A. penicillin

B. azithromycin

C. ceftriaxone

D. gentamicin

E. vancomycin

Answers. 1. - B, 2. - D, 3. - C, 4. - D, 5. - C, 6. - D, 7. - A, 8. - E, 9. - E, 10. - A

Clinical task 1. A 10-year-old boy suffered primary rheumatic carditis without a heart defect. At the age of 12, after a sore throat, the condition worsened: heart pain, shortness of breath, pain in the knee and elbow joints, a rash in the form of delicate pink rings appeared on the legs and chest. The child is in a forced sitting position, pale, lips are cyanotic. The apical impulse is weakened, the boundaries of the heart are widened, especially on the left side to the front axillary line. Tones are deaf, rhythmic, bradycardia. On the left edge of the sternum, a noise resembling a "creaking of snow" is heard, which intensifies when pressed with a stethoscope. The pulse is weak. Liver +3 cm from the edge of the costal arch, soft. On the R-gram, the heart is triangular in shape, with a double contour visible on the left edge.

Your diagnosis?

Features of anti-inflammatory therapy of this case.

Answer standard: Diagnosis: Acute rheumatic fever, active phase, III degree activity, pancarditis, polyarthritis, ring-shaped erythema, CH II A.

A feature of anti-inflammatory therapy in this case is the need to prescribe steroid hormones together with non-steroidal anti-inflammatory drugs (prednisolone 0.75-1.0 mg/kg of body weight in the first 2-3 days evenly throughout the day, in the following days - in accordance with the daily rhythm of the activity of the adrenal glands.

Diffuse connective tissue diseases

Systemic connective tissue diseases, or diffuse connective tissue diseases, are a group of diseases characterized by a systemic type of inflammation of various organs and systems, which is combined with the development of autoimmune and immune complex processes, as well as excessive fibrosis.

The group of systemic diseases of the connective tissue includes the following diseases:

- systemic lupus erythematosus;

- systemic scleroderma;

- diffuse fasciitis;

- idiopathic dermatomyositis (polymyositis);

- Sjögren's disease (syndrome);

- mixed connective tissue disease (Sharpe's syndrome);

- polymyalgia rheumatica;

- recurrent polychondritis;

- recurrent panniculitis (Weber-Christian disease).

In addition, this group currently includes Behçet's disease, primary antiphospholipid syndrome, and systemic vasculitis.

Systemic diseases of connective tissue are united by the main substrate - connective tissue - and a similar pathogenesis. Connective tissue is a very active physiological system that determines the internal environment of the body,

originating from the mesoderm. Connective tissue consists of cellular elements and intercellular matrix. Among the cells of the connective tissue, there are actually connective tissue - fibroblasts - and their specialized varieties, such as chondroblasts, osteoblasts, synoviocytes; macrophages, lymphocytes. The intercellular matrix, which significantly exceeds the cellular mass in quantity, includes collagen, reticular, elastic fibers and the main substance consisting of proteoglycans. Therefore, the term "collagenosis" is outdated, the more correct name of the group is "systemic connective tissue diseases". To date, it has been proven that with systemic diseases of the connective tissue, deep disturbances of immune homeostasis occur, which are expressed in the development of autoimmune processes, i.e. reactions of the immune system, accompanied by the appearance of antibodies or sensitized lymphocytes directed against the antigens of one's own body (autoantigens). The basis of the autoimmune process is an immunoregulatory imbalance, which is expressed in suppression of the suppressor and increase in the "helper" activity of T-lymphocytes with subsequent activation of B-lymphocytes and hyperproduction of autoantibodies of the most different specificity. At the same time, the pathogenetic activity of autoantibodies is realized through complement-dependent cytolysis, where fixed immune complexes circulate, interact with cell receptors and ultimately lead to the development of systemic inflammation. Thus, the commonality of the pathogenesis of systemic diseases of the connective tissue is a violation of immune homeostasis in the form of uncontrolled synthesis of autoantibodies and the formation of antigen-antibody immune complexes that circulate in the blood and are fixed in the tissues, with the development of a severe inflammatory reaction (especially in the microcirculatory channel, joints, kidneys, heart, etc.). In addition to the proximity of pathogenesis, the following features are characteristic of all systemic diseases of connective tissue: multifactorial type of predisposition with a certain role of immunogenetic factors associated with the sixth chromosome; single morphological changes (disorganization of connective tissue, fibrinoid changes of the main substance of connective tissue, generalized damage to the vascular bed - vasculitis, lymphoid

and plasma cell infiltrates, etc.); the similarity of certain clinical signs, especially in the early stage of the disease (for example, Raynaud's syndrome); systemic, multiorgan damage (joints, skin, muscles, kidneys, serous membranes, heart, lungs); general laboratory indicators of inflammatory activity; general group and immunological markers specific to each disease; close principles of treatment (anti-inflammatory drugs, immunosuppression, extracorporeal cleansing methods and pulse corticosteroid therapy in crisis situations).

The etiology of systemic diseases of the connective tissue is considered from the standpoint of the multifactorial concept of autoimmunity, according to which the development of these diseases is caused by the interaction of infectious, genetic, endocrine, and environmental factors (i.e., genetic predisposition + environmental factors, such as stress, infection, hypothermia, insolation, trauma, as well as the effect of sex hormones, mainly female, pregnancy, abortions contribute to the emergence of systemic connective tissue diseases). Most often, environmental factors either exacerbate the hidden disease or, in the presence of a genetic predisposition, are the starting points for systemic diseases of the connective tissue. Currently, indirect data on the possible role of chronic viral infection have been accumulated. The role of picornaviruses, RNA viruses - in measles, rubella, parainfluenza, mumps, as well as DNA-containing herpes viruses - Epstein-Barr, cytomegalovirus, herpes simplex virus is studied. The chronicity of a viral infection is associated with certain genetic features of the body, which allows us to talk about the often familial - genetic nature of systemic diseases of the connective tissue. In patients' families, compared to healthy families and the general population, various systemic diseases of the connective tissue are more often observed, especially among relatives of the first degree of consanguinity (sisters and brothers), as well as more frequent lesions of monozygotic twins than dizygotic ones. Numerous studies have shown an association between the carrier of certain HLA-antigens (which are located on the short arm of the sixth chromosome) and the development of a specific systemic connective tissue

disease. For the development of systemic connective tissue diseases, the carrier of class II HLA-D genes, which are localized on the surface of B-lymphocytes, epithelial cells, bone marrow cells, etc., is of the greatest importance. For example, systemic lupus erythematosus is associated with DR3 histocompatibility antigen. Accumulation of A1, B8, DR3-antigens in combination with DR5-antigen is noted in systemic scleroderma, and in primary Sjogren's syndrome - a high correlation with HLA-B8 and DR3. Thus, the mechanism of development of such complex and multifaceted diseases, which are systemic diseases of the connective tissue, has not been fully studied. However, the practical application of diagnostic immunological markers of the disease and determination of its activity will improve the prognosis of these diseases.

Systemic lupus erythematosus (SLE)

Systemic lupus erythematosus (SLE) is an autoimmune disease. The reason for its appearance is not completely clear. However, it is assumed that the disease is genetically determined. The essence of the lesion in SLE is reduced to the formation of antibodies in the body to its own tissues. The immune system ceases to correctly recognize the proteins of the body and, perceiving them as "foreign", attacks them.

Clinical manifestations of systemic lupus erythematosus are extremely diverse, and correct diagnosis is possible taking into account all manifestations, as well as a number of laboratory tests.

The onset of the disease is often characterized by weakness, weight loss, and some increase in body temperature; all these manifestations are non-specific and may resemble a common cold.

In the future, typical signs of systemic lupus erythematosus develop:

- skin lesions

- damage to muscles and joints

- damage to the cardiovascular system

- damage to the nervous system

- other internal organs.

Skin lesions often have primary diagnostic value, despite the fact that skin manifestations are very diverse. Thus, there are 28 variants of skin changes in SLE. The most typical are reddish spots on the face, neck, chest and in the area of large joints of the hands and feet. These spots are quite bright, can merge with each other, but are sharply delineated from normal skin. A characteristic manifestation is the "butterfly" symptom - the presence of such spots on the cheeks and bridge of the nose that merge with each other as if forming a butterfly shape. Along with the spots, the appearance of rashes in the form of red rings with paler skin inside the ring is possible. Skin manifestations are accompanied by dry skin, diffuse hair loss, brittle nails. Whitish rashes and ulcers on the mucous membrane of the mouth are also possible.

In the vast majority of cases of systemic lupus erythematosus, pain in the joints appears. Pain in one or more joints can last from a few minutes to several days. With high activity of the disease, the pain can be persistent, with the development of inflammation and swelling of the joint. The phalanges and wrists of the hands, knee joints are especially often affected. Joint lesions are usually symmetrical. Damage to 2 or more joints is characteristic.

In case of a lupus-like lesion of the heart, all its membranes are involved in the pathological process, but most often the outer one is the pericardium, or "heart bag". There is pain behind the sternum, which intensifies when laughing and sneezing, shortness of breath, the appearance of cardiac edema on the legs, front

wall of the abdomen is possible. It can be combined with another serositis - pleurisy. Characteristic for SLE is kidney damage - "lupus nephritis".

SLE is also characterized by neurological symptoms in the form of seizures or psychoses.

Systemic lupus erythematosus is characterized by Haserik's diagnostic triad: 1) lupus corpuscles (deep) - free groups of nuclear substance with neutrophils damaged by nuclear antibodies; 2) rosette phenomenon - lupus corpuscles with neutrophils all around; 3) LE cells are neutrophils that have engulfed lupus corpuscles.

Laboratory - immunological changes: hematological - leukopenia, lymphopenia, thrombocytopenia, anemia, immunological are LE - cells, antibodies to DNA, false-positive Wasserman test, antinuclear antibodies, reduced activity of complement fractions.

Kidney damage is characterized by persistent proteinuria (more than 0.5 g/l), hematuria, cylinduria with granular cylinders.

Treatment of systemic lupus erythematosus must be started as early as possible to avoid irreversible organ damage. The main drugs in therapy are anti-inflammatory drugs, corticosteroids and agents that suppress the activity of the immune system.

Scleroderma

Scleroderma is a chronic disease with the characteristic development of local or generalized fibrosis of the skin, underlying tissues and visceral organs.

Focal and systemic scleroderma are distinguished.

Focal scleroderma is divided into

- plaque

- linear

- deep nodular subcutaneous.

The plaque form of scleroderma is characterized by the appearance of one or multiple foci of whitish color with a purple rim around the periphery. The foci can increase, thicken or dissolve, leaving behind atrophy of the skin, the underlying tissues, as a rule, are not changed.

In the linear form of scleroderma, cells are located on the limbs along the course of the vascular-nerve bundle and can spread deep, affecting the subcutaneous tissue, muscles and bones and leaving behind areas of lipodystrophy, amyotrophy, growth disorders of the affected limb. When the focus of scleroderma is localized on the scalp and gradually spreads to the skin of the forehead, the back of the nose, it is called scleroderma of the "saber stroke" type. This form is quite difficult to distinguish from facial trophoneurosis - Romberg's atrophy.

With the deep subcutaneous nodular form of scleroderma, the cells are most often localized on the thighs and buttocks, the skin above them is unchanged or has the appearance of "orange peel", which indicates the involvement of the muscle fascia in the process. There are changes in the deep muscle fascia in the area of the forearms and lower legs, which causes the development of flexion contractures.

Systemic scleroderma is a progressive disease with characteristic induration of the skin, vasospastic reactions, pseudoarthritis, acroosteolysis and the possible development of calcifications and a number of visceritis. The most characteristic of visceritis is esophagitis, in the form of diffuse expansion of the esophagus with narrowing of the lower part and weakening of peristalsis, duodenitis, malabsorption syndrome, as well as diffuse basal fibrosis of the lungs, large focal cardiosclerosis, scleroderma nephropathy.

Therapy of scleroderma should solve the following tasks: elimination of systemic and local inflammation, normalization of microcirculation, reduction of fibrosis processes, restoration of impaired body functions.

Dermatomyositis

Dermatomyositis is a diffuse connective tissue disease. Its occurrence is associated with Coxsackie viruses, a genetic predisposition. Dermatomyositis syndrome can develop with primary blood pathology, toxoplasmosis, trichinellosis and can accompany scleroderma. Girls get sick more often, the peak incidence occurs at the age of 8-10.

Clinic of dermatomyositis. In approximately 25% of children, the disease begins acutely with fever, swelling and muscle damage, bright dermatitis and rapidly progressive muscle weakness. There are intense muscle pains, sweating, general dystrophy progresses rapidly. In subacute variants of the disease, the body temperature is subfebrile, the growth of all symptoms is gradual from 2-3 weeks to 3 months. The disease often develops following respiratory infections, angina, cooling, stress. Skin lesions in dermatomyositis are polymorphic, but are a constant symptom of the disease and are manifested in the form of erythema with a cyanotic shade, located on the face in the form of periorbital purple erythema ("purple disease"), on the auricles, over the joints. Damage to the mucous membranes is manifested by cheilitis, gingivitis, stomatitis, as well as damage to the gastrointestinal tract. In the case of muscle damage, edema, testiness and woody density, pain on palpation and increasing weakness are noted. Myogenic paresis or paralysis develops, including pseudobulbar syndrome with dysphagia, dysarthria, and dysphonia. Breathing disorder is possible. When joints are affected, arthralgias or arthritis are observed. Lymph nodes are often affected, which leads to swelling of the neck and face. In dermatomyositis, all the layers of the heart are involved, most often the myocardium. Pneumonia, glomerulonephritis may be observed. General and muscular dystrophy, febrile reaction, and salivation are among the

general symptoms. The course of the disease can be favorable, dermatomyositis can manifest itself in the form of separate episodes, or with frequent exacerbations, or continuously recur. A severe condition (observed in 10% of cases) is a myopathic crisis with exclusion of respiratory, larynx, pharyngeal and skeletal muscles against the background of severe intoxication, fever, decreased cardiac and respiratory functions, requiring immediate intensive therapy.

Corticosteroid hormones remain the drugs of choice in the treatment of dermatomyositis.

Nodular periarteritis

Periarteritis nodosa is a systemic vascular disease in which the walls of medium and small arteries are affected. It occurs most often in men, but it is also possible in children. The etiology is not exactly established. Sensitizing factors are infection, especially viral, intoxication, vaccination, administration of serums and drugs. The pathogenetic role is attributed to immune complexes circulating in the blood and fixed in the vessel walls. Thickening of an inflammatory nature, small aneurysms, hemorrhage, and necrosis were noted in the walls of small vessels. Blood clots are often found in the affected vessels. Histological changes resemble those found in serum sickness. When blood circulation is disturbed, the tissues where the damaged vessels are located are affected (kidneys, nervous system, cardiovascular system, lungs, gastrointestinal tract).

The clinical picture of the disease begins gradually, the body temperature rises, the fever has an irregular character. Patients complain of weight loss, pain in muscles and joints, weakness, sweating, lack of appetite. Kidneys with glomerulonephritis syndrome and hypertension are often affected. When heart vessels are damaged, angina occurs, myocardial infarction may occur. If the vessels of the abdominal cavity are damaged, abdominal pain, nausea, diarrhea, vomiting, and blood in the stool occur. There may be a picture of gastritis,

enteritis, appendicitis. Violation of blood supply to nerves leads to polyneuritis. Focal lesions of the brain and its membranes are possible. The disease can be acute or chronic. In acute forms, death occurs with the phenomena of renal failure with hypertension, hemorrhages, intestinal infarction may occur. In chronic forms, death is a consequence of renal sclerosis, heart failure, cachexia, and secondary infection. Treatment consists in the appointment of hormone therapy in high doses (2-3 mg/kg) in the early stages of the disease. Sometimes hormones cause the formation of multiple heart attacks, the progression of kidney failure. If the treatment is ineffective, immunosuppressants are prescribed.

Rheumatoid arthritis

Rheumatoid arthritis is a disease that is based on immunopathological processes and is characterized in most patients by a chronic course with systemic damage to the connective tissue, mainly of the musculoskeletal system.

There are two main forms of rheumatoid arthritis:

- articular

- articular and visceral

- several options for each of them.

The articular form develops gradually, is accompanied by minor flare-ups without a pronounced temperature reaction, without signs or with weakly expressed signs of damage to internal organs, with the rare appearance of an allergic rash, symptoms of an allergic condition. Changes in the joints in this form have the character of mono or oligoarthritis. Knee, then ankle, small hand, wrist, elbow, and relatively rarely all other joints are more often involved in the pathological process. Asymmetry is typical for joint damage. Local phenomena are characterized by a change in the shape of the joint, an increase in temperature

above the joint, limitation of function, hypotrophy of the muscles of the corresponding limb.

Rheumatoid uveitis develops relatively often (20%) in the articular form of rheumatoid arthritis. Eye capsules are involved in the pathological process, iritis or iridocyclitis occurs, less often choroiditis or damage to all three parts of the choroid - uveitis. Ribbon-like dystrophy of the cornea and opacification of the lens are added as secondary manifestations.

The triad of symptoms (iridocyclitis, cataract, and corneal dystrophy) is considered typical for childhood rheumatoid arthritis. The course of the articular form (with the exception of rheumatoid uveitis) is relatively favorable.

The articular-visceral form (Still's disease) is the most severe form of rheumatoid arthritis in children. It is characterized by an acute onset and violent development, high fever, massive joint syndrome, pronounced reaction of lymphoid organs and liver, visceritis (myocarditis, pericarditis, pleurisy, etc.). Joint damage is multiple, symmetrical, sometimes all joints are affected. Characteristic morning stiffness, which, depending on the duration, can characterize the activity of juvenile rheumatoid arthritis. Peculiarities in children - oligoarthritis in combination with chronic iridocyclitis is described only in children. Oligoarthritis with damage to large joints is more common in younger children.

Laboratory signs. Positive rheumatoid factor in the diagnostic titer. Positive results of biopsy of the synovial membrane.

Treatment begins with non-steroidal anti-inflammatory drugs (brufen, meloxicam, nurofen, indomethacin), if they are ineffective, hormonal drugs are used, even in a dose of 3-4 mg/kg (to obtain an immunosuppressive effect). In patients with low activity, chloroquine, plaquenil, D - penicillinamine (cuprenil, methylcaptase) are preferable. Cyclosporine is used in severe disease. Massage and physiotherapy methods are also used in complex treatment.

TESTS

1. LE cells are characteristic of the following disease

A. Dermatomyositis

B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

2. Which disease is characterized by the "saber stroke" symptom

A. Dermatomyositis

B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

3. In which disease purple erythema is observed on the face

A. Dermatomyositis
B. Systemic lupus erythematosus
C. Rheumatism
D. Scleroderma
E. Juvenile rheumatoid arthritis

4. Morning stiffness is characteristic of the disease
A. Dermatomyositis
B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

5. What disease is characterized by joint deformation

A. Dermatomyositis

B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

6. Which disease is characterized by Hazerik's triad

A. Dermatomyositis

B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

7. Juvenile rheumatoid arthritis is characterized by heart defects

A. Aortic valve defect

B. Mitral valve defect

C. There is no defect

D. Tricuspid valve defect

E. Combined aortic and mitral valve defect

8. Skin rashes in the form of a butterfly are characteristic of the disease

A. Dermatomyositis

B. Systemic lupus erythematosus

C. Rheumatism

D. Scleroderma

E. Juvenile rheumatoid arthritis

9. Treatment of the allergoseptic variant of rheumatoid arthritis begins with

A. Nonsteroidal anti-inflammatory drugs

B. High doses of hormones

C. Suprastina

D. Chloroquine

E. D – penicillinamine

10. It is used to treat juvenile rheumatoid arthritis with low activity

A. Ibuprofen

B. Prednisolone

C. Suprastin

D. Chloroquine

E. D – penicillinamine

Answers. 1. - B, 2. – D, 3. - A , 4. - E, 5. -E ,6. - B, 7. - C, 8. - B, 9. - B, 10. – A

Clinical task 1. A 12-year-old girl has been complaining of knee pain for the past 3 weeks. She turned to the pediatrician due to the fact that, against the background of a runny nose, pain appeared in the second knee joint. During the examination, an increase in temperature to subfebrile figures was established, both knee joints were moderately enlarged and swollen. On the skin of the face, closer to the bridge of the nose, there are mildly pronounced rashes that look like a butterfly. On auscultation, the heart sounds are muffled, a soft blowing noise at the top of the heart. Palpation of the abdominal cavity revealed an increase in the size of the liver by 2 cm, the edge was soft, rounded. The spleen is not palpable. Pasternacki's symptom is weakly positive on both sides. In the blood test - ESR - 50 mm/h, leukocytopenia with lymphopenia. In the urine analysis, the urine is cloudy, protein up to 1 g/l, erythrocytes in a small amount, granular cylinders.

What is your previous diagnosis?

What laboratory methods of examination will confirm the diagnosis?

What immunological data will confirm the diagnosis?

What additional methods of examination should be performed in a sick child?

With what other diseases should a differential diagnosis be made?

Answer standard. Systemic lupus erythematosus, acute course with skin lesions, polyarthritis, myocarditis, lupus nephritis.

Laboratory methods of examination: general blood analysis in the dynamics of observation, general analysis of urine in the dynamics of observation, detection of antinuclear factor, LE cells.

Immunological: antibodies to DNA, antinuclear antibodies, decrease in the activity of complement fractions.

Additional examination methods: x-ray of knee joints, electrocardiogram, ultrasound examination of internal organs (heart, kidneys, liver).

Differential diagnosis is provided with dermatomyositis, juvenile rheumatoid arthritis, rheumatism, scleroderma, nodular periarteritis.

Pyelonephritis

Pyelonephritis is a non-specific microbial-inflammatory disease of the calyceal system and tubulointerstitial tissue of the kidneys with predominant damage to the tubulointerstitium. Acute pyelonephritis is an inflammatory disease of the renal parenchyma and pelvis that arose as a result of a bacterial infection. Chronic pyelonephritis is kidney damage, which is manifested by scar changes (fibrosis) and deformation of the calico-pelvic system, as a result of repeated infections of the genitourinary system, as a rule, against the background of anatomical anomalies of the urinary tract or obstruction, which are confirmed by imaging methods of diagnosis.

Etiology. In most cases, pyelonephritis is caused by a single uropathogen, but with frequent relapses and against the background of defects in the development of the urinary system, microbial associations are found in up to 62% of cases. Currently, Gram-negative flora predominates among the causative agents in children, while about 90% is due to infection with Escherichia coli bacteria. Among other pathogens, Proteus mirabilis (in boys - about 30%), Klebsiella spp. (Mostly in young children), Enterobacter spp. and Pseudomonas spp. - are detected in less than 2% of cases. Nosocomial infections with strains of Klebsiella, Serratia and Pseudomonas spp. Gram-positive microorganisms are mainly represented by enterococci and staphylococci (5-7%). In newborns, streptococci of groups A and B are a common cause. Recently, an increase in the detection of Staphylococcus saprophyticus has been noted, although its role remains controversial. Intrauterine viral infections are considered as a contributing factor to bacterial infection. The development of pyelonephritis can be caused by urogenital chlamydia, ureaplasmosis, mycoplasmosis, especially in children with nonspecific inflammatory diseases of the external genitalia. In children with immunodeficiency conditions (premature, with hypotrophy, intrauterine infection, developmental defects, children who received immunosuppressive therapy for a long time), associations of bacteria with fungi are characteristic. Actinomyces species,

Brucella spp., Mycobacterium tuberculosis are registered with the hematogenous route of infection.

Pathogenesis. Conditions that lead to the development of pyelonephritis: pyelonephritis refers to diseases with a hereditary predisposition; dysembryogenesis in the kidney, immaturity of nephrons that create ischemic foci; the role of metabolic disorders (in primary and secondary tubulopathies with a change in urine pH and crystalluria, often as a result of enzymopathy) they create favorable conditions for the fixation of microorganisms in the kidney tissue; microbes penetrate into the capillary network, from there into the interstitial tissue, where inflammatory foci appear. In Ukraine, the main pathogenetic theory of the development of pyelonephritis is the block-cascade theory, which was proposed by Academician V.G. Maidannyk.

Favorable conditions in young children: congenital abnormalities of the genitourinary system, early transition to mixed or artificial feeding, frequent diseases of early age: intracranial birth trauma, dystrophy, rickets, atopic diathesis; diseases of other organs leading to urodynamic disorders and kidney hemodynamic disorders.

Leading risk factors for the development of pyelonephritis in children: children aged the first 2 years of life; anomalies of the development of the genitourinary system, urogenital area - organic disorders of the passage of urine; neurogenic dysfunction of the urinary bladder: rare or frequent urination, urinary incontinence - functional disorders of the urinary passage; violation of the composition of the urine itself (for example, diabetes, tubulopathy); constipation; worm infestations; inflammatory diseases of external genital organs (vulvovaginitis, balanitis, balanoposthitis); transurethral medical manipulations (bladder catheterization); masturbation, early sexual life; an important cold factor - there is a spasm of the muscles and a violation of the hemodynamics of the kidney.

Ways of infection. Ascending - the most frequent. Anatomical features determine the high frequency of infections of the urinary system and pyelonephritis in girls. The reservoir of uropathogenic bacteria is the rectum, perineum, and lower parts of the urinary tract. After the bacteria overcome the vesicoureteral barrier, their rapid reproduction occurs with the release of endotoxins. In response - activation of the local immunity of the macroorganism (macrophages, lymphocytes, endothelial cells) with the production of inflammatory cytokines (IL1, IL2, IL6, tumor necrosis factor), lysosomal enzymes, inflammatory mediators; activation of lipid peroxidation, which leads to damage to kidney tissue, primarily tubules. This view of the pathogenetic mechanism of the occurrence of pyelonephritis was called the block - cascade theory of the development of this disease. The hematogenous path of development of pyelonephritis occurs mainly in the development of septicemia in the newborn period and in the first months of life, especially in the presence of immune defects; this pathway also occurs during infection with Actinomyces species, Brucella spp., Mycobacterium tuberculosis. The lymphogenic pathway is less significant, possible in the presence of damaged urinary tract mucosa, not recognized by all nephrologists; there is a hypothesis about the lymphogenic migration of microorganisms from the intestine.

Classification. In Ukraine, the modern classification of A.F. Voziyanov, V.G. Maidannyk, and I.V. Baghdasarova is used

Clinical form	Characteristic of the process	Activity	Stage	Kidney function status
Non-obstructive	Acute	I, II, III degree of activity	Infiltrative	No kidney function disturbance
Obstructive	Chronic with wave-like or latent course		Sclerotic	With kidney function disturbance
				Chronic renal failure

Clinical manifastation. The clinic of pyelonephritis depends on 1) the age of the child, 2) the general previous condition, 3) ways of infection. Symptoms are divided into: general typical symptoms of a severe infectious process with pronounced intoxication; symptoms of a local nature (in the case of an ascending path of infection, these symptoms may first appear). General symptoms with intoxication: mainly febrile temperature without catarrhal phenomena or subfebrile, lethargy, fatigue, headache, decreased appetite to anorexia, vomiting, pain in the abdomen, lumbar region, pathognomonic symptom of throbbing on one or both sides, paleness, earthy complexion , changes on the part of the blood (leukocytosis, increased ESR), in case of impaired kidney function, arterial hypertension, anemia, osteodystrophy. Symptoms of a local nature: often minor dysuria, including enuresis (symptoms of variants of urinary tract infection, which can be concurrent with pyelonephritis or precede it), in case of impaired kidney function, nocturia, polyuria and oliguria in the final stage, changes in urine.

The course of pyelonephritis in newborns and infants is characterized by a non-specific clinical picture and general symptoms of intoxication. Among them, the following symptoms should be highlighted: fever or hypothermia (more often in premature babies and with hypotrophy) - may be the only symptom in newborns and young children; paleness or marbling of the skin, lethargy, vomiting and retching, decreased appetite, insufficient weight gain or loss; symptoms can persist for a long time, subfebrility is possible in the dynamics; the equivalent of dysuria in children of the first year of life can be anxiety or crying during and after urination, facial redness, croaking, urination in small portions, weakness or intermittent urine stream; dysuria in the form of accelerated and painful urination is not typical for children younger than 1.5-2 years and usually does not occur with pyelonephritis; an alternative to the symptom of tingling in young children is to press with a finger in the area of the 12th rib and spine. Approximately 60% of infants have impaired kidney function. Hyperazotemia develops, which is associated with hypercatabolism of own tissues as a result of the intoxication

process; this is confirmed by an increase in blood urea, and an increase in creatinine with a decrease in glomerular filtration - the main criteria of kidney function. In older children, the main symptoms of pyelonephritis are presented in the form of the following symptoms: febrile fever, often short-term, symptoms of intoxication, then there may be prolonged subfebrility without catarrhal phenomena; vomiting, diarrhea (rarely), pain in the abdomen and / or lower back, a positive symptom of Pasternacki's palpation (this symptom can be negative if the kidney is not located in a typical place (dystopia, nephroptosis), then the pain can be in the meso- or hypogastrium), sharp smell of urine, dysuria are symptoms of other options for urinary tract infection, they sometimes contribute to the diagnosis of pyelonephritis, for example, febrile fever with dysuria).

Diagnostics. The main laboratory criteria of pyelonephritis: leukocyturia, bacteriuria (bacteriological examination of urine), minor proteinuria. A violation of the urine concentration process is possible, especially in the acute period. Neutrophilic leukocytosis with a shift to the left, an increase in ESR (more than 20 mm / h). A high level of CRP and procalcitonin (helps in diagnosis in the absence of typical signs of inflammation). Pathognomonic leukocyturia and bacteriuria; conduct a clinical analysis of urine with a count of the number of leukocytes. Quantitative urine tests: Addis-Kakovsky test (the most reliable) - urine is collected during the day, excretion per day is estimated. Normal indicators: up to 2,000,000 leukocytes, up to 1,000,000 erythrocytes, up to 20,000 cylinders. Nechyporenko's test: a portion of morning urine obtained from the middle stream, the secretion is estimated at 1 ml. Normative values for girls are up to 4,000 leukocytes, boys up to 2,000 leukocytes, erythrocytes up to 1,000, up to 250 cylinders. Other laboratory signs: glucosuria (a sign of tubulopathy, acute or chronic impairment of tubular functions of the kidneys); crystalluria is considered normal with unstable changes - up to (++); specific gravity of urine - a sign of tubular functions of the kidneys, the norm > 1018 - 1020 in older children, > 1015 - in infants, > 1010 - in newborns; decrease - hypoisostenuria can be in renal

failure; protein in the urine is not normally detected, with pyelonephritis proteinuria is insignificant, as a rule, < 1 g / day; hematuria in pyelonephritis is rare, standards depending on the method: hematuria > 5 erythrocytes/μl by test strips, microscopically > 3 in the field of view of non-centrifuged urine or the presence of > 5 erythrocytes in the field of vision at x40-microscopy of centrifuged urine. Important urocytogram (selectivity of leukocyturia): in pyelonephritis, neutrophilic leukocyturia > 90%; in addition, the mycelium of mushrooms is excluded microscopically.

Extraction of culture from urine. When a microorganism is isolated in a monoculture from an average portion of urine in a titer > 105 CFU/ml, the causative agent can be considered etiologically significant. It has been proven that urine can be collected during free urination in a clean container after a thorough perineal toilet for culture isolation. The disadvantage of the method of free urination is the high risk of contamination, especially in children of the first months of life. Non-golden staphylococcus, greenish streptococcus, micrococci, corynebacteria and lactobacilli are considered typical contaminants. Other diagnostic methods: ECG, blood pressure profile. Biochemical examination of blood: CRP / procalcitonin, urea, creatinine. Clearance of endogenous creatinine or calculation of GFR according to Schwartz. Zimnytsky's test (Reiselman in younger children) + daily proteinuria. Quantitative urine samples (according to Nechyporenko) with urocytogram. Two-glass urine sample. Accounting for the rhythm and volume of spontaneous urination - to exclude the collective concept of urodynamic disorders.

Treatment. The main principles of pyelonephritis therapy in children: elimination of the acute inflammatory process, prevention of recurrent infection. Features of the appointment of antibacterial drugs are: targeting the sensitivity of microorganisms, reducing the dose of the antibacterial drug depending on creatinine clearance, timely detection and correction of urodynamic disorders, long-term antimicrobial prophylaxis in the presence of reflux and recurrent urinary

tract infection, control of the functional capacity of the intestine. Antibacterial drugs used for the treatment of pyelonephritis in outpatients: amoxicillin + clavulanic acid 50 mg / kg / day (according to amoxicillin) 3 times a day, cefixime 8 mg / kg /day 1once a day, cefuroxime axetil 50-75 mg / kg / day two times a day, ceftibuten 9 mg / kg / day once a day. In hospitalized patients, especially infants, when it is difficult to give the drug orally, antibacterial therapy is started parenterally in the first three days, followed by the transition to oral administration. In the absence of pronounced intoxication and the preserved ability of the child to receive the drug by mouth, oral administration is possible from the first day. Antibacterial drugs for parenteral use: amoxicillin + clavulanic acid 90 mg / kg / day 3 times a day, ceftriaxone 50-80 mg / kg / day once a day, cefotaxime 150 mg / kg / day four times a day, cefazolin 50 mg / kg / day thre times a day. Aminoglycosides can be used as reserve drugs (amikacin 20 mg/kg/day once a day, tobramycin 5 mg/kg/day thre times a day, gentamicin 5-7.5 mg/kg/day thre times a day), carbapenems . With pseudomonas infection - ticarcillin / clavulanate (250 mg / kg / day) or ceftazidime (100 mg / kg / day) + tobramycin (6 mg / kg / day).

Today, the appointment of cephalosporins of the III generation should be considered as the starting empirical antibacterial therapy. The effectiveness of the treatment is assessed after 24-48 hours based on clinical signs and results of a urine test. If the treatment is ineffective, anatomical defects or kidney abscess should be suspected. Antibacterial therapy for 7-10 days usually eliminates the infection, regardless of its localization, with longer courses, resistance of the flora is produced. After a course of antibacterial therapy, uroantiseptics should be prescribed - furagin, furadonin, at a dose of 5-8 mg/kg, 5-NOK, nitroxoline at 5-10 mg/kg, sulfonamides - biseptol, pipemidine acid.

Prevention (primary): regular emptying of the bladder and bowels, sufficient fluid intake, hygiene of the external genitalia. Indications for preventive treatment (secondary): presence of reflux; severe anomalies of the development of the urinary system before surgical correction. The duration of prophylaxis is

individual, usually at least 6 months. Preparations for long-term antimicrobial prophylaxis: furazidin 1 mg / kg once at night, co-trimoxazole 2 mg / kg (after sulfamethoxazole) Only once at night, amoxicillin + clavulanic acid 10 mg / kg once at night. In addition, phytotherapy with bactericidal effect can be used for therapeutic and preventive purposes. From the point of view of evidence-based medicine, preference should be given to canefron-type phytocollections made using the phytoniering mechanism.

Prognosis. Focal shrinkage of the kidneys is found in 10-20% of patients who have undergone pyelonephritis, especially with relapses of infection and the presence of reflux. More active diagnosis and treatment at an early age reduce the risk of progression to the stage of chronic renal failure, arterial hypertension develops in 10% of children with pyelonephritis.

TESTS

1. Name the main etiological factor of acute pyelonephritis in young children?

A. Golden staphylococcus

B. Escherichia coli

C. Streptococcus

D. Adenovirus

E. Candida albicans

2. Name the main route of exposure of the pathogen in the case of pyelonephritis:

A. Hematogenous

B. Lymphogenic

C. Descending

D. Ascending

E. Combined

3. What theoretical justification of the pathogenesis of pyelonephritis do you know?

A. Block-cascade theory

B. Immune inflammation

C. Direct microbial damage

D. Virus-mediated kidney damage

E. Autoimmune process in the kidneys

4. The leading clinical syndrome in young children with pyelonephritis is:

A. Intoxicating

B. Dysuric

C. Painful

D. Vegetative

E. Dyspeptic

5. The leading clinical syndrome in older children with pyelonephritis is:

A. Intoxicating

B. Dysuric

C. Painful

D. Vegetative

E. Dyspeptic

6. Which method of counting formed elements in urine belongs to express methods?

A. Method of urine collection according to Zimnytskyi

B. Method of collecting urine according to Nechiporenko

C. Urine collection method according to Addis-Kakovsky

D. General analysis of urine

E. Urine culture for pathogen culture

7. Which method of counting formed elements in urine is one of the most accurate methods?

A. Method of urine collection according to Zimnytskyi

B. Method of collecting urine according to Nechiporenko

C. Urine collection method according to Addis-Kakovsky

D. General analysis of urine

E. Urine culture for pathogen culture

8. Which group of antibiotics is the starting point for children with acute pyelonephritis?

A. Group of penicillins

B. group of cephalosporins of the III generation

C. Group of aminoglycosides

D. Group of macroliths

E. Group of fluoroquinolones

9. Your further treatment tactics after the end of the course of antibiotic therapy for pyelonephritis:

A. Prescribe uroantiseptics

B. Prescribe nonsteroidal anti-inflammatory drugs

C. Prescribe drinking of renal collections of herbs

D. Prescribe a course of vitamins

E. Cancel drug therapy

10. From the standpoint of evidence-based medicine, which herbal medicine should be preferred in the treatment of pyelonephritis in children?

A. Renal collection No. 1

B. Renal collection No. 2

C. To kidney weapon No. 3

D. Kanefron

E. Collecting daisies

Answers. 1. - B, 2. - D, 3. - A , 4. - A , 5. - C, 6. - B, 7. - C, 8. - B, 9. - A , 10. - D

Clinical task. A 3-year-old girl was admitted to the hospital with complaints of nausea, lethargy, decreased diuresis, and change in urine color. Child from 2nd pregnancy, which occurred with toxicosis of 2nd half, urgent uncomplicated delivery. She was born with a weight of 3400, a length of 51 cm. Early

development without peculiarities; suffered from pain in the abdominal cavity for a year. During the last two days, a high fever, nausea, loss of appetite appeared, urination began to decrease, urine was cloudy. In connection with these complaints, she was referred for hospitalization. During examination, the child is pale, lethargic, complains of pain in the abdominal cavity. The child's temperature is 38.9◦C, the child's weight is 14 kg. Blood pressure 95/65 mmHg. Art. In the lungs, breathing is weakened in the lower parts, there are no wheezing. The borders of the heart on percussion are not expanded to the left, the tones are muffled, the heart rate is 32/min. The abdomen is soft, painless, the liver +2.5 cm, the edge is soft. During the day, the girl released 350 ml of urine; urine is cloudy. General blood analysis: Hemoglobin - 111 g / l, erythrocytes - 4.2x1012 / l, leukocytes - 10.9x109 / l, p / i - 4, s / i - 64, l. - 20, e - 4, m. - 8, ESR 25 mm / h. General analysis of urine: color - white-yellow, transparency - cloudy, R.N. - 6.0, specific gravity - 1025, protein - 0.5 g / l, acetone - negative, glucose - negative, leukocytes - 30 in p / vision, erythrocytes - single in the field of vision, flat epithelium -0-1 in p / with polymorphic 1-2 in n / vision, cylinders: absent in the field of vision. Urine culture revealed Escherichia coli. Biochemical analysis of blood: total protein 60 g/l, albumin 32 g/l, cholesterol 4.6 mmol/l, urea 8.5 mol/l, creatinine 112 μmol/l, endogenous creatinine clearance - 48 ml/min. Ultrasound of the kidneys - the right kidney is enlarged, the contours are even, the location is typical. The differentiation of the parenchyma layers is disturbed, the echogenicity of the parenchyma is moderately increased. The cup-bowl system is deformed on the right.

Your diagnosis?

Plan for additional examination?

What will be the initial antibiotic therapy?

What can be prescribed from uroantiseptics?

Answer standard. Acute obstructive pyelonephritis of the right kidney. Acute renal failure. Start antibiotic therapy ceftriaxone 500 thousand. × 2 times/day, nitroxoline 200 mg per 1 tablet. × 4 times a day.

Glomerulonephritis

Glomerulonephritis is an acute diffuse immune-inflammatory lesion of the kidneys, mainly glomeruli, which occurs after a bacterial, viral or parasitic disease, after a certain latent period (a period of sensitization). Most often, glomerulonephritis occurs with nephritic syndrome, has a cyclical course. Glomerulonephritis is often equated with post-streptococcal glomerulonephritis.

Epidemiology. According to endemic research, the frequency of glomerulonephritis in the pediatric population is 33:100,000. Children of both sexes are affected, more often at the age of 6-12 years, mostly boys, the disease occurs, as a rule, in a sporadic form, the number of patients with glomerulonephritis is increasing. Factors in the development of glomerulonephritis are: burdened heredity in relation to infectious and allergic diseases; increased family susceptibility to streptococcal infection; presence of chronic foci of infection in the child; hypovitaminosis, helminthiasis; frequent ARVI; cooling and meteorological factors; vaccinations; reception of allergens; presence of HLA antigens, DRW4, DRW6, B12.

Etiology.The etiology of glomerulonephritis is infectious. Diseases are caused by viral diseases (Australian antigen, infectious mononucleosis, cytomegalovirus, Coxsackie B4 virus); bacterial diseases (subacute bacterial endocarditis, streptococcal, staphylococcal infection, typhoid); parasitic diseases (malaria, schistosomiasis, toxoplasmosis). Glomerulonephritis of streptococcal nature occurs after streptococcal diseases (angina, impetigo, scarlet fever, erysipelas, lymphadenitis). The disease is caused by nephritogenic strains of β hemolytic streptococcus group A (types 1, 2, 4, 12, 18, 25, 49). The etiology of streptococcal glomerulonephritis is confirmed by culture from the streptococcal focus, detection of antigens and antibodies in the blood - ASO, antihyaluronidase, antistreptokinase.

Pathogenesis. The pathogenesis of glomerulonephritis of streptococcal etiology involves the formation of immune complexes consisting of anti-streptococcal antibodies and streptococci. Complement, properdin, inflammatory mediators, factors of cellular immunity are involved in immune reactions, anti-GBM (glomerular basement membrane) antibodies are also formed. As a result of the activation of the coagulation system, a local disseminated intravascular coagulation syndrome develops. The main consequences of a developed inflammatory process in the kidneys are a decrease in glomerular filtration, and the formation of the main syndromes of glomerulonephritis - urinary, edematous and hypertensive. Macrohematuria develops due to an increase in vascular, capillary and tissue permeability, activation of hyaluronidase, which leads to depolymerization of hyaluronic acid, which is part of the main substance of connective tissue and the intercellular substance of the vessel wall - per diaridesum, erythrocytes penetrate into the urine, which involves the clotting system of platelets (their aggregation) and plasma factors (factor XII, Hageman), a local disseminated intravascular coagulation syndrome develops, leading to macrohematuria. An increase in blood pressure is based on the activation of the renin-angiotensin-aldosterone system, which leads to an increase in the secretion of the antidiuretic hormone of the pituitary gland, which is the basis of the increase in the volume of circulating blood. Edemas in glomerulonephritis develop due to the stimulation of aldosterone, which increases the reabsorption and retention of sodium and water in the body. An increase in vascular and tissue permeability due to inflammation is also important in the development of edema. The accumulation of sodium in the vascular bed increases the osmolality of the plasma, which contributes to the increase in the secretion of antidiuretic hormone and the increase in the sensitivity of the distal tubules to it, and even greater retention of water and the development of hypervolemia. An additional increase in the sodium content in the body contributes to an increase in the content of angiotensin II and aldosterone. The activation of the kinin-kallikrein system is also important, which leads to an increase in vascular permeability and the exit of fluid from the blood

into the tissue space, with the redistribution of fluid and its accumulation in loose tissue. The pathogenesis of rapidly occurring nephrotic edema includes primary retention of sodium and water, activation of the kinin-kallikrein system and hyaluronidase with a total increase in vascular permeability, followed by the release of the liquid part of the blood into the interstitial space.

Classification of primary glomerulonephritis

Forms of glomerulonephritis	Activity of the renal process	Kidney function status
Acute: With acute nephritic syndrome; With nephrotic syndrome; With isolated urinary syndrome; With nephrotic syndrome with hematuria, arterial hypertension.	Period of initial manifestations. The period of extensive clinical manifestations. The period of reverse development. Transition to chronic glomerulonephritis.	No kidney function disturbance With kidney function disturbance. Acute renal failure
Chronic: Nephrotic form Hematuric form Mixed form	Period of exacerbation. A period of partial remission. Period of complete clinical and laboratory remission	No kidney function disturbance With kidney function disturbance. Chronic renal failure
Subacute (malignant)		With kidney function disturbance. Chronic renal failure

There are two clinical variants of the course of glomerulonephritis - cyclic (typical) and acyclic (monosymptomatic). In typical cases, the anamnesis reveals a previous streptococcal lesion of the throat and skin. 2-4 weeks after the transferred infection (latent period), there is a deterioration of the general condition, a decrease in diuresis, darkening of urine, the appearance of a headache, swelling on the face, lower legs, sometimes on the abdomen, lower back. There may be a short-term increase in temperature, nausea, less often vomiting, pain in the kidney area, sometimes signs of eclampsia. Objectively: the child is pale (due to angiospasm), edema localized on the face and lower legs. Swellings are "hidden" (revealed by a positive McClure Aldrich test). From the side of the cardiovascular system, tachycardia, less often bradycardia, muffled tones, expansion of the limits of relative cardiac dullness, weakening of the 1st tone at the apex, strengthening of the 2nd tone at the aorta and pulmonary artery, expansion of the borders of the heart (due to an increase in blood pressure) are detected. In some cases, circulatory failure develops. Urine the color of "meat slops" (macrohematuria). Oliguria is diagnosed in half of the children, recovery of diuresis is noted after 3-7 days. During the period of reverse development of symptoms, polyuria appears, edema disappears, arterial hypertension disappears, extrarenal manifestations of the disease, headaches, and malaise. Finally, hematuria disappears. Complete restoration of morphological changes in the kidneys occurs after 1-2 years. Acyclic variant of glomerulonephritis occurs, often with an isolated urinary syndrome. The disease is characterized by a gradual onset in the absence of subjective symptoms and extrarenal manifestations. After several years, chronic glomerulonephritis can develop in various forms. The main clinical variants of glomerulonephritis are: nephrotic and nephritic syndromesДіагноз glomerulonephritis грунтується на виявленні перенесеної стрептококової інфекції і латентного періоду після неї. Характерна тріада симптомів (гематурія, помірні набряки, підвищення артеріального тиску) дає підставу запідозрити glomerulonephritis.

Nephritic syndrome is a symptom complex that includes extrarenal symptoms (edema or pastiness, increased blood pressure, changes in the heart, central nervous system) and real (oliguria, hematuria, proteinuria up to 1 g per day, cylindruria) manifestations. In the period of the initial manifestations of glomerulonephritis, a violation of kidney function can be observed, sometimes acute renal failure develops.

Nephrotic syndrome is a symptom complex characterized by oliguria up to anuria, massive proteinuria (more than 50 mg / kg / 24 g or more than 3 g / 24 h), hypo- and dysproteinemia (a decrease in albumin below 25 g / l, as well as γ-globulins, an increase α2 and β-globulins), hyperlipidemia and hypercholesterolemia, which corresponds to the concept of complete nephrotic syndrome (NS). "Incomplete" NS occurs without swelling. NS occurs in two forms of glomerulonephritis: nephrotic and mixed.

Features of the course of glomerulonephritis in children. 1. The initial period of the disease is often combined with acute renal failure. 2. A combination of glomerulonephritis and pyelonephritis is more often observed. 3. In children with nephrotic syndrome, when treated with glucocorticoids, good results are more often observed. 4. In young children, the morphological diagnosis of glomerulonephritis is complicated, since hereditary and congenital kidney pathologies and their combination are often detected at this age. 5. Mono and oligosymptomatic manifestations of the disease are often observed. 6. Abdominal syndrome is observed more often than in adults, but arterial hypertension is less often detected.

Diagnostic. Laboratory studies General analysis of urine. With glomerulonephritis, hematuria is most often detected, and at the beginning of the disease, leukocyturia. Leukocyturia is a marker of the immune-inflammatory process in the kidneys. Cylindruria (hyaline, granular, erythrocytic) is a characteristic finding for glomerulonephritis. Many patients have proteinuria up to 1-2 g/l/day. In the blood test: increased ESR, neutrophilia; in the presence of an

infectious focus - leukocytosis, moderate anemia. Biochemical research. Dysproteinemia due to moderate hypoalbuminemia, hyper-α2 and γ-globulinemia is observed. With the development of pronounced oliguria in some patients, the level of urea and creatinine in the blood increases, which is considered as a violation of kidney function in the acute period, the development of acute renal failure is possible. During a serological examination, antistreptolysin O titers increase in 60-90% of patients. Anti-M-protein antibodies appear 4-6 weeks after a streptococcal infection and persist for a long time. In 90% of patients, circulating immune complexes increase in the blood. During the first two weeks of the disease, the level of C2, C3, C4, complement fractions decreases, which lasts 4-6 weeks. Disorders in the coagulation system are characterized by: an increase in the prothrombin index, a decrease in the level of antithrombin III, inhibition of fibrinolytic activity, and the appearance of fibrinogen degradation products in the blood. With ultrasound, it is possible to detect a slight increase in the size of the kidneys, an increase in their echogenicity.

Kidney biopsy - intravital morphological examination of kidney tissue using a percutaneous puncture (closed biopsy) or an operative method (open biopsy). Indications for biopsy in glomerulonephriti: atypical manifestations of glomerulonephritis, age up to 3 years, anuria, high azotemia, inconsistent with the clinical picture; growth disorders, glomerulonephritis in the family, long-term persistence of symptoms (delayed remission), signs of a systemic disease that persist for a long time, hematuria and proteinuria.

Differential diagnosis. The differential diagnosis of glomerulonephritis is carried out with: chronic glomerulonephritis, hereditary nephritis, hemorrhagic vasculitis, transient urinary syndrome on the background of an acute infectious disease, acute pyelonephritis, acute rheumatic fever, systemic lupus erythematosus, rheumatoid arthritis, infectious endocarditis, interstitial nephritis, rapidly progressive glomerulonephritis, IgA nephropathy (disease Berge), hemolytic uremic syndrome.

Treatment. Tasks of treatment: eradication of streptococcal infection, elimination of renal and extrarenal manifestations of glomerulonephritis. Scheme of treatment. Mandatory measures: bed rest, diet, antibacterial therapy. Auxiliary treatment: means of symptomatic therapy (diuretic, hypotensive), vitamin therapy, antihistamines, euphilin, disaggregants, membrane-stabilizing drugs, antioxidants. Indications for hospitalization: treatment of glomerulonephritis is carried out in a hospital. Regime. Strict bed rest is indicated for extrarenal symptoms and macrohematuria. Extending the regimen is indicated when hypertension, edema, and reduction of hematuria are eliminated. The possibility of transfer to an extended regimen is decided by the absence of complaints, hypertension and improvement of urinary sediment. Diet. The fluid is prescribed based on the previous day's diuresis and perspiration losses (15ml/(kg/day) or 400ml/(m2/day)) for schoolchildren. With an increase in diuresis, the amount of fluid drunk increases. Restriction of sodium chloride (without table salt) is prescribed for oliguria and hypertension. When blood pressure is normalized and diuresis is increased, salting of food is allowed (0.5-1.0 g/day). The normal amount of sodium chloride (50 mg / (kg/day)) with a favorable course of the disease, the child can receive from the 4th-5th week. At the onset of glomerulonephritis, table 7a (according to Pevzner) is prescribed for 3-5 days. On the 3-5-7 day, a transitional table 7b is appointed. The amount of protein and fat in the diet increases. Later, the patient is transferred to table 7c. Salt is added to ready meals. Protein restriction is indicated for oliguria and hypertension. For a period of 5-7 days, reduce protein consumption (to 1.0-0.5 g / (kg/day)). Some restriction of animal proteins for 2-3 weeks is also advisable. Caloric content is maintained due to an increase in carbohydrates and fats in the diet. With oliguria, potassium restriction is indicated. Due to the danger of hyperkalemia, exclude fruit or vegetable juices. Potassium-sparing drugs are contraindicated. After swelling subsides, it is advisable to enrich the diet with potassium (baked potatoes, fruits, etc.). Patients with glomerulonephritis need antibacterial therapy mainly with antibiotics of the penicillin series (semi-synthetic penicillins such as amoxicillin or macrolides in

usual doses). In the absence of foci of infection, the duration of antibacterial therapy is 7 - 10 days. If there are foci of chronic infection after the end of the course of antibacterial therapy (4 - 6 weeks), you can use Bicilin - 5. The duration of bicillinotherapy is up to 6 months. Pathogenetic therapy - consists in the appointment of glucocorticoids (prednisolone, polcortolone) - for nephrotic syndrome; nephrotic syndrome with hematuria and arterial hypertension; with the addition of acute renal failure. Anti-inflammatory nonsteroidal drugs are used in glomerulonephritis with isolated urinary syndrome. Cytostatics (leukeran, chlorbutin) are used for chronic glomerulonephritis, plaquenil, delagil for the hematuric form of chronic glomerulonephritis. To improve renal blood flow, antiaggregants (curantyl, persantil) are used, which are prescribed for 3-4 weeks 2-3 times a day in a daily dose of 1.5-3.0 mg/kg/day. Heparin therapy is indicated for: presence of signs of hypercoagulation; symptoms of intrarenal (local) intravascular blood coagulation (a rapid decrease in kidney function with a decrease in the content of fibrinogen and an increase in the content of fibrin degradation products in blood serum); the presence of disseminated intravascular coagulation syndrome; severe edematous syndrome; pronounced hyperlipidemia. Methods of heparin therapy: parenteral administration (subcutaneous, intramuscular) by the electrophoresis method (300 units/kg) and by the aerosol method (500 units/kg), 100-200 units/kg daily dose in 4 injections. The drug should be withdrawn gradually under the control of coagulogram indicators. Nicotinic acid activates the fibrinolytic system of the blood, prevents the aggregation of platelets, and has a vasodilating effect. Electrophoresis of 1% nicotinic acid solution is used on the kidney area. Procedures are carried out daily, the number of procedures is 7-10. Euphilin increases the lumen of kidney vessels, gives a mild diuretic effect, and reduces total peripheral resistance. The drug is prescribed for 1-2 weeks in powders or tablets 3 times a day in a daily dose: up to 9 years - 15-18 mg / (kg/day); 9-12 years - 10 - 12.5 mg / (kg/day); older than 12 years - 10 mg / (kg/day); you can use trental, nicospan. Prescribe vitamins A, groups B, E in age-related doses; membrane stabilizers - xidifon, dimephosphon,

karsil. Diuretics are rarely used in glomerulonephritis. Diuretics are indicated for massive edema, arterial hypertension, hypertensive encephalopathy. In order to increase diuresis, furosemide (Lasix) is prescribed at a dose of 1.5 - 2.0 mg / kg intramuscularly or intravenously 1-2 times a day, then for another 3 days the drug is administered orally in 1 dose. Hypotensive drugs are indicated at the level of diastolic pressure above 95 mm Hg. Art. and with hypertensive encephalopathy. For high hypertension, the drugs of choice are captopril (kapoten). Captopril is prescribed in a daily dose of 0.3 mg/kg/day. The dose can be increased to 2.0 mg/kg/day for 3-5 days. Intravenous administration of a 2.4% solution of Euphilin in physiological saline together with Lasix (1-4 mg/kg) is possible. the basis of treatment of arterial hypertension - diuretics and calcium antagonists. At the threat of eclampsia, a 1% solution of dibazole and a 5% solution of papaverine (0.1 mg/kg/day) and a 1% solution of furosemide (1.0 - 2.0 mg/kg) are used intramuscularly. In case of eclampsia, to obtain a rapid hypotensive effect, diazoxide is injected intravenously as soon as possible in a dose of 2 - 5 mg / kg (maximum dose of 100 mg) or methyldopa intravenously in a dose of 5 - 10 mg / kg (administration of methyldopa can be repeated after 20-60 minutes). To relieve convulsions, prescribe a 0.5% solution of seduxen (relanium) in a dose of 0.3-0.5 mg/kg intramuscularly or sodium oxybutyrate in a dose of 100-150 mg/kg, 10% calcium gluconate intravenously. Treatment is carried out against the background of oxygen therapy. Hemodialysis for glomerulonephritis is indicated in the absence of a reaction to lasix, an increase in urea > 20-24 mmol / l, potassium > 7 mmol / l, phosphorus > 2 mmol / l, sodium < 130 mmol / l, blood pH > 7.25. In glomerulonephritis with isolated urinary syndrome, the basis of treatment is antibiotics of the penicillin series, macrolides in combination with antiplatelet agents. In case of glomerulonephritis with nephritic syndrome, the basis of treatment is antibiotics of the penicillin series, macrolides in combination with antiplatelets and direct anticoagulants.

Complication. With acute glomerulonephritis, the development of: angiospastic encephalopathy, acute renal failure, acute cardiovascular failure is possible.

Prognosis. Recovery occurs in 85-90%. Fatality is rare (< 1%). In 10-15% of cases, glomerulonephritis transforms into chronic glomerulonephritis. The near and distant forecast is favorable.

TESTS

1. What is the most common cause of acute glomerulonephritis in children?

A. Nephrogenic strains of beta hemolytic streptococcus group A

B. Staphylococcus aureus

C. Allergic factor

D. Adenovirus

E. Worm infection

2. A lesion is characteristic of acute glomerulonephritis

A. Cup-bowl system

B. Kidney glomeruli

C. Kidney tubules

D. Kidney interstitium

E. All together

3. The main damage to the kidneys occurs due to which immunological changes?

A. Immunocoplex damage of the kidneys

B. Immunological reactions according to the Artyus phenomenon

C. Cytotoxic damage to the kidney

D. Reactions of the immediate type with the formation of immunoglobulin E

E. All of the above immunological reactions take part in kidney damage

4. What syndromes are characteristic of acute glomerulonephritis?

A. Nephritic

B. Nephrotic

C. Isolated urinary

D. Nephrotic with hypertension and hematuria

E. All of the above

5. What complication is characteristic of the initial period of acute glomerulonephritis?

A. Acute heart failure

B. Angiospastic encephalopathy

C. Acute renal failure

D. Hypertensive crisis

E. Convulsions

6. Pain syndrome in acute glomerulonephritis is observed in the form of:

A. Pain in the lumbar region of the spine

B. Headache

C. Abdominal pain

D. Pain in the heart area

E. Pain in the lower third of the abdominal cavity

7. Intense edematous syndrome is characteristic of

A. Nephritic syndrome

B. Nephrotic syndrome

C. Hematuric syndrome

D. Hypertensive syndrome

E. For all the above-mentioned syndromes

8. Dysproteinemia with hypercholesterolemia is characteristic of the following syndrome:

A. Nephritic syndrome

B. Nephrotic syndrome

C. Hematuric syndrome

D. Hypertensive syndrome

E. For all the above-mentioned syndromes

9. Glucocorticoid therapy is used in case of which syndrome?

A. Nephritic syndrome

B. Nephrotic syndrome

C. Hematuric syndrome

D. Hypertensive syndrome

E. For all the above-mentioned syndromes

10. Anti-inflammatory nonsteroidal medicinal products are used in the following syndrome:

A. Nephritic syndrome

B. Nephrotic syndrome

C. Isolated urinary

D. Hypertensive syndrome

E. With pain syndrome

Answers. 1. - A, 2. - B, 3. - A, 4. - E, 5. - B, 6. - C, 7. - B, 8. - B, 9. - B, 10. - C

Clinical task 1. An 11-year-old boy was admitted to the hospital with complaints of headache, nausea, lethargy, decreased diuresis, and change in urine color. Child from 2nd pregnancy, which occurred with toxicosis of 2nd half, urgent uncomplicated delivery. He was born with a weight of 3400, a length of 51 cm. Early development without features; before a year he suffered from atopic dermatitis, after a year he often suffered from acute respiratory infections, three times from angina. A week ago, he suffered from angina. During the last two days, a headache, nausea, loss of appetite appeared, he began to urinate less, the urine was dark brown and cloudy. In connection with these complaints, he was referred for hospitalization. During the examination, the child is pale, lethargic, complains of a headache, the child's bloat, swelling on the lower legs are noted. Blood pressure 125/90 mmHg. Art. The temperature is 37.5◦C. In the lungs, breathing is weakened in the lower parts, there are no wheezing. The borders of the heart on percussion are expanded to the left, tones are muffled, systolic murmur at the apex, heart rate 68 / in min. The abdomen is soft, painless, the liver +2.5 cm, the edge is soft. During the day, the boy released 350 ml of urine; urine is red-brown, cloudy. General blood analysis: Hemoglobin - 111 g / l, erythrocytes - 4.2x1012 / l, leukocytes - 10.9x109 / l, p / i - 4, s / i - 64, l. - 20, e - 4, m. - 8, ESR 25 mm / h.

General analysis of urine: color - brown, transparency - cloudy, R.N. - 6.0, specific gravity - 1026, protein - 1.5 g / l, acetone - negative, glucose - negative, leukocytes - 10 in p / vision, erythrocytes - completely cover all fields of vision, flat epithelium -0-1 in field of vision, cylinders: hyaline - 10, erythrocyte - 25-30 in the field of vision. Urine culture - the result is negative. Biochemical blood analysis: total protein 60 g/l, albumins 32 g/l, cholesterol 4.6 mmol/l, urea 15 mol/l, creatinine 140 μmol/l, seromucoid 0.38, ASL: About 1:1000, CRP 0.012 (norm 0.0001), potassium 6.1 mmol / l, sodium 140 meq / l. Clearance according to endogenous creatinine - 52 ml / min.. Ultrasound of the kidneys - the kidneys are enlarged, the contours are even, the location is typical. The echogenicity of the parenchyma is moderately increased. Cup-bowl system without deformations and ectasias.

Your diagnosis? Treatment tactics?

Answer standard. Acute glomerulonephritis, nephritic syndrome. Acute renal failure. Treatment is bed rest, diet - restriction of salt and proteins of animal origin, amoxacillin 1.0 x 2 p/day, nurofen 0.4 x 2 p/day, lespenefril 1t. × 3 p/day, black rowan tablets

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