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Educational manual for students and interns of higher medical institutions

Infectious Diseases Of The Digestive System In Children

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Українська медична стоматологічна академія

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Ukrainian Medical Stomatological Academy
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Навчальний посібник підготовлений співробітниками кафедри ендокринології з дитячими інфекційними хворобами (зав. кафедри проф. Л.Є. Бобирьова) Української медичної стоматологічної академії, доц., к.мед.н. К.В.Пікуль, доц., к.мед.н. В.І.Ільченко, ас., к.мед.н. К.Ю. Прилуцьким; лікарем-педіатром ПОДКЛ О.В. Чеботар.

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List of abbreviations

AII - acute intestinal infections
ABB- acid-base balance
CVP - central venous pressure
TSS - toxic shock syndrome
ELISA - enzyme-linked immuno sorbent assay
EPE - enteropathogenic Escherichia
ETE - enterotoxigenic Escherichia
CIP - complex immunoglobulin preparation
RIA - reaction of immunofluorescence analysis
RA - reaction of agglutination
RPHA - reaction of passive hemagglutination
CFR - complement fixation reaction
NR - neutralization reaction
HDR - hemadsorption delay reaction
IFR - immunofluorescence reaction
RIA - radioimmune analysis
OR - oral rehydration
VH - viral hepatitis
VHA - viral hepatitis A
VHB - viral hepatitis B
VHC - viral hepatitis C
VHD - viral hepatitis D
CHB - chronic hepatitis B

Навчальний посібник складений згідно з типовим навчальним планом та програмою для підготовки іноземних студентів медичних закладів вищої освіти для циклу «Дитячі інфекційні хвороби». Цей навчально-методичний посібник написаний англійською мовою і присвячений питанням етіології, епідеміології, клініки та диференційної діагностики найбільш частих дитячих інфекційних хвороб, наведені алгоритми діагностики та терапії, згідно з протоколами лікування, що затверджені МОЗ України. Назви розділів автори підібрали у відповідності з тематичним планом для підготовки іноземних студентів медичних закладів вищої освіти. Посібник містить запитання для самоконтролю, тести, задачі та розбір історій хвороб дітей, що лікувались у дитячому відділенні ПОКІЛ для більш глибокого засвоєння матеріалу.

The educational manual is compiled according to a typical curriculum and a program for the preparation of foreign students of medical institutions of higher education for the cycle "Children's Infectious Diseases". This teaching manual is written in English and is devoted to the issues of etiology, epidemiology, clinics and differential diagnostics of the most common infectious diseases of children, diagnostic algorithms and therapies are given in accordance with the treatment protocols approved by the Ministry of Health of Ukraine. The titles of the sections were chosen by the authors in accordance withthe thematic plan for thepreparation of foreign students of medical institutions of highereducation. The manual contains questions for self-control, tests, tasks and analysis of children's diseaserecords that were treated at the children's department for a deeper assimilation of the material.

Manual

Infectious diseases have always been dangerous, which indicated on their mass prevalence, rapid spread and high mortality rates, especially in childhood. Many infections can lead to the development of urgent conditions, which requires intensive care, taking into account the etiological factor of the pathological process.

This manual is devoted to the issues of etiology, epidemiology, clinics and differential diagnosis of the most common emergencies among pediatric infectious diseases, and provides algorithms for emergency care and intensive care. The manual contains questions, tests, self-monitoring tasks that will help students and interns learn material better.

We hope that the manual we have written will be useful in the daily practical work of pediatricians, pediatric infectious diseases doctors and family physicians.

The authors gratefully acknowledge all wishes and critical remarks

CONTENT

Anatomical and physiological features of

gastrointestinal tract in children

Knowledge of the anatomy and features of digestion, synthesis of physiologically active substances, the final decomposition of enzymes plays an important role in understanding the processes of entry, reproduction and development of pathogenic and opportunistic microorganisms in children, which is important in the treatment and prevention of acute intestinal infections.

The beginning of the formation of the human digestive system occurs in the early stages of embryogenesis and its morpho-physiological improvement lasts up to 18-25 years. After birth and in young children, the oral cavity (cavum oris) is small and filled with a relatively wide tongue (lingva). The mucous membrane is quite dry and is represented by a delicate epithelium, which is rich in blood vessels. Saliva secretion begins at 3 months of age is a consequence of physiological salivation. The following glands are located in the oral cavity: labial, parotid, buccal, submandibular, sublingual, lingual, palatal. Children's saliva contains amylase, lysozyme, immunoglobulin A and others. Its bactericidal properties are less pronounced than in adults. The saliva reaction is weakly acidic, which can cause frequent infection of the oral cavity with Candida albicans.

The esophagus in children is located higher than in adults - its upper limit is at the level of 3-4 cervical vertebrae, in 2-4 years - at the level of 4-5th, up to 12 years - at the level of 5-6- him. In adults, the esophagus is located from the 6th cervical to the 11th thoracic vertebrae. The esophagus in children is relatively longer and is S body length, which provides almost simultaneous swallowing and breathing, without interrupting the innate unconditional reflex of the act of sucking. The own plate of the mucous membrane at the level of the larynx and in the area of the transition to the stomach contains cardiac glands. The mucous membrane of the esophagus is dry, tender, the glands in infants almost do not function. Elastic fibers and muscle layer are poorly developed.

The formation of a pile of food in humans occurs in the oral cavity. The act of swallowing is divided into phases: pushing crushed food into the pharynx, the reflex phase of swallowing, passing through the esophagus into the stomach. The stomach of children has a number anatomical and physiological features, knowledge of which allows to understand possible pathological processes. In infants, the stomach can take different shapes: oval, pear-shaped, round. At the end of the first year, the stomach

changes from horizontal to vertical. These features can cause vomiting after feeding the baby, so it is recommended to hold the baby upright for the next 15 minutes.

The anatomical structure of the stomach is as follows:

- cardiac part - adjacent to the esophagus;
- bottom of the stomach;
- body of the stomach;
- pyloric part - adjacent to the duodenum.

The main secretory glands (glandula gastricae propriae) are located in the area of the bottom and body of the stomach, as well as the pyloric glands (glandula pyloricae). In children, the body of the stomach is poorly developed. The muscular membrane is poorly developed and has imperfect innervation, especially in the cardiac and pyloric parts, which sometimes leads to pylorospasm. Elastic tissue develops up to 7-12 years, which helps to improve the motor function of the stomach. Histological differentiation lasts up to 2 years.

The stomach performs the following functions:

1. Barrier function - the destruction of pathogenic microorganisms that have entered through the oral cavity.
2. Secretory function - the production of enzymes in the juice cells of gastric juice.
3. Breakdown of proteins and lipids by enzymes of gastric juice (hydrochloric acid, pepsin, chymosin, lipase, etc.).
4. Endocrine function - the production of biologically active substances: gastrin, histamine, serotonin, motilin, enteroglucagon.
5. Excretion into the lumen of the digestive tract of ammonia, urea, alcohol.
6. Mechanical (motor) function - pushing food into the duodenum.

Barrier function in the first year is low due to insufficient formation of hydrochloric acid and lysozyme, which increases the possibility occurrence of acute intestinal infections. Motor function in the first months is slowed down due to imperfect regulation. In children, food is in the stomach for 3.5-4 hours. If the baby is on artificial feeding, then the food is delayed a little longer than on natural.

The capacity of the stomach increases with age: the newborn - 35 ml, at 3 months. - 100 ml, in 8 years - up to 1000 ml, in an adult - 1.7-2 liters. Gastric juice of the newborn contains all the components, but the activity of enzymes is not high (tab. 1).

Table 1

Indicators of gastric juice in children

Age	Volume	pH	Free acidity, title. Od	General acidity, title. Od.
Newborns	0,1-0,3	6,5-8	0-0,5	3-6
1 year	0,2-1	1,5-3	5-10	10-15
2-7 years	0,5-1,5	1-2	10-15	20-25
8-10 years	1-2	1-2	12-15	25-30
11-14 years	1-5	1-2	15-20	30-35

The small and large intestines in children are almost 2 times longer than in adults. Anatomically, the small intestine (intestinum tenue) has the following sections:

- duodenum;
- hungry gut (jejunum);
- ileum.

At an early age, the duodenum is at the level of the 11th thoracic - 2nd lumbar vertebrae. In the prepubertal period is located at the level of the 1-4th lumbar vertebrae. Intestinal motility in infants is more pronounced, both peristaltic and antiperistaltic movements are clear, which can cause intussusception. The peculiarity of the small intestine is the relief of the mucous membrane, the presence of folds (plicae), villi (villi intestinales) and creep, where the ducts of the intestinal glands (glandulae intestinalis) open, which contain Penet cells with acidophilic secretory granules. In the submucosal layer of the duodenum lie the terminal secretory divisions of the duodenal glands (glandulae duodenalis), which resemble pyloric. Their secret protects the intestines from the harmful effects of gastric juice. Membrane digestion takes place in the small intestine with the help of microvilli enzymes. Between the villi are the liberkyun glands, which produce intestinal juice. The mucous membrane also contains many lymphoid cells, which are scattered throughout the intestine in infancy, which eventually turn into "Peyer's patches" that perform a barrier function. In infants, this function of lymphoid follicles is low, which leads to the ingress of microorganisms with the possible occurrence of

infectious and inflammatory processes. Food advancement, membrane digestion, absorption of nutrients occurs in the small intestine for 7-8 hours.

The large intestine (intestinum crassum) is represented by the cecum with the appendix (vermiformis), the colon (ascendens), the transverse bypass (transuersum and the ascending descents), the sigmoid colon. (rectum). The length of the colon in each age group corresponds to the length of its body, but the location and peristalsis has its own characteristics at an early age. The ascending colon is characterized by the presence of inflections and insufficiency of haustrae (haustrae coli). The position of the transverse colon is not constant: it can fall a few centimeters from the navel, and can rise to its level or even higher. The sigmoid colon up to 5-6 years of age has long ripples that help the appearance of loops, which in turn can be reflected in its position and approach to the anterior-midline of the abdomen. The rectum in children under 6 years of age is located above the entrance to the pelvis, and in older age in the pelvis. In infants, it has a poorly developed muscular layer, which can lead to rectal prolapse as a result of tenesmus in some infectious intestinal diarrhea. The physiological microflora of the colon plays a significant role in preventing the entry and reproduction of pathogenic and opportunistic microorganisms of the child. In the large intestine, a handful of food moves and nutrients are absorbed in 4-12 hours.

The physiological microflora of the large intestine in adults is up to 2.5-3 kg. The result of the joint symbiotic activity of the cells of the epithelium of the mucous membrane of the large intestine and the physiological microflora is the construction of a complex specific of the epithelial structure - **biofilms**. It is a protective mucosal barrier consisting of a layer of mucus, secretory immunoglobulin A molecules, microcolonies of indigenous bacteria and their metabolites. In the process of biofilm formation there is a more stable settlement of microflora components at specific loci.

Classification of physiological microflora

1**. Indigenous** (main, obligate, own) microflora - represented by bifidobacteria, lactobacilli proprion bacteria. These are the friendliest human bacterial symbionts. It is 95-99% of the total microflora and is located in the epithelial zone of the mucous membrane of the colon. Bacteroids (Bacteroides, Fusobacterium) also belong to the obligate microflora, but in pathological disorders they can lead to infectious diseases.

2. **Optional** (additional, accompanying) microflora - represented mainly by facultative aerobic bacteria of Escherichia coli and Strepoccus faecium species. The concentration of these bacteria does not exceed 5% and they inhabit the part of the lumen of the colon adjacent to the epithelial zone of the mucous membrane. These

are opportunistic pathogens that, despite their significant role in microbiotic processes, can cause severe infectious diseases under adverse conditions.

3. **Transient** (residual, allochthonous) microflora - represented by opportunistic pathogens of genera:
Staphylcoccus, Clostridium, Citrobacter, Enterobacter, Proteus, Klebsiella, Pseudomonas, Candida and others. She composes 0.01% and inhabits the lumen of the colon.

One of the largest glands in the body is the liver (hepar). In adults, it weighs up to 1.5-2 kg, which is up to 1/50 of body weight. In newborns, it is 1/8 of the total weight of the child. The connective tissue capsule covering the liver in infants is thin with delicate elastic fibers. Structural and functional unit - the hepatic lobe, of which there are up to 500 thousand, at an early age are vaguely demarcated. In adults, they are separated in the corners where the vessels pass (portal tracts). The hepatic lobules are built of hepatic beams, which consist of 2 rows of hepatocytes, between which are the bile capillaries. Important anatomical formations are Kupffer cells, which perform the function of macrophages.

The liver receives blood from 2 vessels that enter through its gate. The portal vein collects blood from all unpaired organs and brings to the liver all the nutrients absorbed from the intestines. The hepatic artery carries blood from the aorta rich in oxygen. In the fetus, the left side of the liver is in the best conditions of blood supply through the blood flow through the umbilical vein, which stops after birth. In infants, the right side is smaller than the left, but over time, the blood pressure in the vessels of the left side decreases and the size equalizes.

The upper edge of the liver is located in 4 intercostal spaces along the right midclavicular line, the left - in 5 intercostal spaces along the left parasternal line in all age categories. The lower edge of the liver in children under 5 years protrudes 2-3 cm from the right costal arch, then up to 7 years - 1-1.5 cm; up to 10 years - 0.5 cm.

The liver performs the following functions:

1. Formation of nitrogenous products from toxins (detoxification).
2. Inactivation of hormones, biogenic amines.
3. Synthesis of glycogen, which is a source of maintaining a constant level of glucose in the blood.
4. Formation of plasma proteins (fibrinogen, albumin, prothrombin).
5. Produces bile, which is necessary for the emulsification of fats.
6. Participates in cholesterol metabolism.

7. Accumulation of fat-soluble vitamins A, D, E, K.

8. The embryo acts as a hematopoietic organ.
9. Pituitary somatotropin mediator, which stimulates bone growth.
10. Barrier function.

The structure of the liver in young children does not have a complete structure, it is functionally immature, which can lead more quickly to the pathological process of intoxication and infection. The functional compensatory capacity of the liver of a healthy school-age child is much higher than that of an adult, because the gland has not yet been exposed to harmful substances.

The hepatobiliary system provides synthesis, thickening, preservation and secretion of bile into the duodenum. Bile begins to form in the peripheral part of the classical hepatic lobe and enters the biliary tract (vasa bilifera) and gallbladder (vesica fellea).

The biliary tract is represented by:

- interstitial bile ducts (ductuli biliferi et interlobularis);
- right and left bile ducts (ductus hepaticus dexter et sinister);
- common hepatic duct (ductus hepaticus communis) .;
- vesical duct (ductus cysticus);
- common bile duct (ductus choledochus).

The gallbladder holds 40-70 ml of bile. At an early age has funnel-shaped, but over time - oval. Per 1 kg of body bile in children produces 4 times more bile than in adults, but it contains more water, mucus and pigments, and cholesterol and bile acids are lower.

The pancreas in adulthood weighs 60-120 g and is located at level 2 of the lumbar vertebra in the extraperitoneal space on the left. In infants, its weight is 2-3.5 g. In the first years of life, the pancreas is projected at a higher level: the head - 12 thoracic vertebrae, body - from 10 thoracic to 2 lumbar vertebrae, tail - from 10 thoracic to 1 lumbar vertebrae. On the anterior wall of the abdomen, the gland is located between the xiphoid process and the navel. The position of the gland is oblique - bottom right and top left. Up to 10-12 years, its surface is smooth, with time there is a hump. The structural and functional part of the exocrine part are acinuses that produce trypsin, lipase, amylase, which enter the duodenum and participate in the breakdown of proteins, fats and carbohydrates. The structural and functional part of

the endocrine part are insulocytes, which make up the islets of Langenhars, whose cells synthesize:

- A-cells - glucagon;
- B-cells - insulin;
- D-cells - somatostatin;
- D1 - vasoactive intestinal polypeptide;
- PP - pancreatic polypeptide.

In infants, the endocrine part of the gland is more developed with a large number of islets of Langenhars. After the introduction of baby food, the exocrine part of the pancreas is gaining momentum, amylolytic activity is especially active, and lipolytic activity reaches the adult level only at 12 years of age.

These anatomical and physiological features of the digestive organs in children should be taken into account when organizing the diet of a healthy child and treatment regimen in patients with acute intestinal infections.

ACUTE INTESTINAL INFECTIONS IN CHILDREN

Acute intestinal infections (AII) are a group of infectious diseases of viral, bacterial, fungal or parasitic etiology that spread by fecal-oral route and mainly affect the gastrointestinal tract.

AII occupy one of the leading places in infectious pathology of childhood. According to the WHO, more than 1 billion people in the world suffer from acute intestinal diseases every year. persons, of whom 65-79% are children under 5 years. Every year in Ukraine, 60% of the total number of deaths from infectious diseases are mostly young children. In the structure of infant mortality, the share of GKI ranges from 50 to 70%. In countries with a high infant mortality rate, up to 15% of children under the age of 3 die each year from intestinal infections. In our country, unfortunately, the etiological structure of GCI changes every year and the tendency to increase the incidence of this pathology persists.

AII occupies a special place among nosocomial (nosocomial - from the word nosos, meaning disease, nosocomialis - hospital) infections, where their proportion reaches 60-80%.

Historical information on the study of AII tells us that all intestinal dysfunctions were once treated as "dysentery," "summer diarrhea," "enteritis," "colitis," food poisoning". As laboratory diagnostics improved, the etiology was deciphered and a large group of intestinal diseases of infectious origin was identified. Thus, in 1954 Vibrio cholerae was discovered, and in 1875. - dysenteric amoeba and protozoal infection. Later, other pathogens of intestinal infections were discovered: Escherichia coli (1880), Shigella and Salmonella (1880), Escherichia coli (1922), and rotavirus (1975). Recently, great importance is attached to opportunistic pathogens (Proteus, Klebsiella, Clostridium, Campylobacter), which are the causative agents of GCI in young children and newborns.

An essential feature of AII is polyetiology. According to the etiological principle, all acute intestinal infections can be divided into three groups:

1. **Intestinal infections caused by bacteria:**
   - Shigella;
   - Salmonella;
   - Escherichia;
   - Klebsiella;
   - Campylobacter and others.
2. **Intestinal infections of viral etiology:**

• Rotavirus;

• Adenovirus;
• Astrovirus;
• Norflokvirus;
• Coronavirus;
• Reovirus;
• Calicivirus and others.

3. **Intestinal infections caused by parasites and fungi:**
• Cryptosporidium;
• Microsporidia;
• Balantidium coli;
• Isocpora;
• Giardia Lamblia;
• Entamoeba Histolitica.

According to the WHO classification, all diarrheal diseases are divided into:

I. Non-infectious diarrhea.
II. Infectious diarrhea:
1. Invasive AII:
• inflammatory;
• bloody.
2. Secretory diarrhea:
• non-inflammatory;
• watery.
3. Diarrhea with immunodeficiency of the child's body.

Secretory diarrhea is caused mainly by viruses, bacteria and protozoa that secrete enterotoxin: Rotavirus, Adenovirus, Astrovirus, Norflokvirus, Coronavirus, Reovirus, Calicivirus, enteropathogenic, enterotoxigenic and enteroaggregative Escherichiae salts, Cryptosporidium, Microsporidia, Balantidium coli, Isocpora.

Invasive diarrhea is caused by the following bacteria: Shigella, Salmonella, enteroinvasive and enterohemorrhagic Escherichiae salts, Campylobacter, Clostridium, Stapfylococcus, Giardia Lamblia, Entamoeba Histolitica.

Secretory diarrhea is manifested by a combination of gastric lesions (frequent and repeated vomiting, pain in the stomach) and the phenomena of enteritis (the appearance of watery stools with undigested food, rapid dehydration).

In invasive diarrhea, the pathogen penetrates the intestinal wall, where it causes inflammation of the small and large intestine (loose stools with a large number of pathological impurities of mucus and blood with severe colitis and intoxication syndromes). Pathogens of invasive diarrhea can enter the bloodstream, causing bacteremia and foci of secondary infection.

The etiological structure of AIII in children of different ages is different. In young children, this pathology is caused mainly rotaviruses, enteropathogenic Escherichia coli, salmonella, staphylococci, Klebsiella, Proteus, Citrobacter and other opportunistic bacteria. Less common are shigellosis, clostridiosis, cholera. Shigellosis, salmonellosis with foodborne transmission, yersiniosis, typhoid fever and paratyphoid fever are more common in older children.

Acute intestinal infections have many common epidemiological features. The children's population is characterized by: high contagiousness, prevalence in children, fecal-oral transmission mechanism, the appearance of both sporadic and epidemic outbreaks, summer-autumn seasonality, features of immunity (species specificity and instability).

PECULIARITIES OF THE PATHOGENESIS OF DIARRHEA IN AII

1. **Osmotic** is characteristic of AII of viral etiology the mechanism of diarrhea, which is due to increased osmotic pressure in the intestine due to the accumulation of disaccharides on the affected epithelium of the villi, which leads to impaired absorption of water and electrolytes.

2. For AII pathogens which **secrete** enterotoxin (enterotoxigenic Escherichia coli, etc.), an inherent secretory mechanism that occurs during increased secretion of water and electrolytes into the intestinal cavity due to enzymatic disorders in the enterocyte membrane.

3. **Invasive** AII is characterized by an exudative mechanism of diarrhea, which is caused by direct damage to the pathogen of the intestinal wall, activation of inflammatory mediators and the release of large amounts of exudate (mucus, protein, blood), which increases the volume of fluid in the intestine.

All AII, regardless of etiology, have many similar ones manifestations. This is manifested by both general infectious (general toxic) syndrome and local symptoms

associated with damage to various parts of the digestive tract (gastritis, enteritis, colitis, gastroenteritis, gastroenterocolitis, enterocolitis). General toxic syndrome in GCI in children manifests itself in the form of intoxication or toxicosis (often toxicosis with exsiccosis), which have the character of a nonspecific reaction of the body to an infectious agent. Intoxication should be understood as a primary violation of intracellular metabolic processes in combination with the insufficiency of the functional state of physiological systems of elimination of toxic metabolic products of the liver, kidneys, lungs, reticuloendothelial system.

Clinical manifestations of intoxication are lethargy, weakness, malaise, loss of appetite, sometimes to anorexia, temperature reaction, dysfunction of various organs and systems. Intoxication is dominated by metabolic disorders and symptoms of irritation of the parasympathetic nervous system. In toxicosis with exsiccosis, metabolic disorders associated with dehydration and loss of electrolytes are leading. Depending on the amount of water lost by the body, there are three degrees of toxicosis with exicosis:

- mild dehydration - fluid loss up to 5% of body weight;
- moderate dehydration - fluid loss from 5% to 10% of body weight;
- severe dehydration - fluid loss of more than 10% body weight.

The degree of dehydration of the child is determined on the basis of two parameters: weighing the patient to determine the deficit of weight (or same in relation to the mass that was on the eve of the disease, or in relation to the one that the child should have at this age) and on the basis of clinical manifestations of dehydration.

According to the content of electrolytes in the blood of patients with exsiccosis, in particular sodium, which is part of the extracellular fluid of the body and its osmolarity, determine three types of dehydration:

- hypertensive, hyperosmolar, hypernatremic, water-deficient type;
- hypotonic, hypoosmolar, hyponatremic, salt deficiency type;
- isotonic type, without electrolyte disturbances.

Monitoring of metabolic disorders is carried out according to changes in the indicators of CBS, electrolytes and gas composition of venous blood (Table 3).

Clinically, dehydration in each type of exsiccosis is manifested by the following symptoms, which are presented in tables 4-6.

Hypertensive (water-deficient, cellular) type develops if water loss predominates, which is facilitated by vomiting and liquid watery stools on the background of hyperthermia and shortness of breath. Water loss is manifested by an increase in the concentration of electrolytes in the extracellular fluid (blood plasma and interstitial fluid), mainly due to hypernatremia, which leads to the transition of fluid into the extracellular space to equalize osmotic pressure and intracellular dehydration.

Table 2

Clinical assessment of dehydration

Indicators	**1 degree**	**2 degree**	**3 degree**
Loss of mass body	0.5%	5-10%	10-15%
Diuresis	slightly reduced	reduced	sharply reduced
Thirst	moderately	sharply expressed	is absent
Skin	not changed	sluggish	taken from fold
Turgor	saved	reduced	much reduced
Mucous shell	wet	dry	dry, hyperemic
Heart rate	standard	moderate tachycardia	embryocardia
Heart tones	loud	weakened	much weakened
Circulation	unchanged	mild acrocyanosis	"marbling"
CNS condition	unchanged	apathy, excitation	sharp lethargy, loss consciousness

Table 3

Indicators of ABB and respiratory process in children with intestinal toxicosis

Indicator	**Toxicosis I**	**Toxicosis II**	**Toxicosis III**	**Healthy children**
1	**2**	**3**	**4**	**5**
pHBE, mmol/l	7,37±0,003	7,25±0,009	7,14±0,025	7,39±0,006

1	2	3	4	5
pCO2, mmol/l	5,3±0,36	8,9±0,38	14,3±0,75	1,2±0,12
venous blood	39,1±0,18	32,3±0,86	28,1±0,63	2,5±0,79

Hypotonic (salt-deficient, extracellular) type of dehydration develops gradually, at a later date in severe forms of AII with a predominance in the clinical symptoms of repeated vomiting on the background of diarrheal syndrome. As the degree of exsiccosis and loss of electrolytes (mainly potassium) increases, vomiting becomes unrelated to food or drink, contains bile, sometimes blood (in the form of "coffee grounds"). The loss of salts is accompanied by a decrease in plasma osmolarity during the transition of water and sodium from the vascular bed into the cells - intracellular hyperhydration develops, a significant increase in the amount of sodium in the cell and a decrease in the amount of potassium, hematocrit is increased. Plasma volume decreases, blood clotting, slowing of blood circulation, hypoxemia, hypoxia, acidosis, microcirculation is disturbed.

In the presence of potassium deficiency in the serum develops lethargy, intestinal paresis, hypotension in children. The amount of potassium according to the standard is 4.5 mmol / l. Metabolic acidosis is manifested by a clinical symptom complex: "marbling" of the skin, hyperthermia, shortness of breath, lethargy, oliguria, peripheral circulatory disorders.

Isotonic (general) type of dehydration develops with extracellular and intracellular proportional loss of water and electrolytes. It is most common at the beginning of AII. Because water and electrolytes are lost in physiological proportions, this condition is compensated for faster during treatment than the two previous types of exsiccosis.

The results of the study of the level of electrolytes are of differential diagnostic significance. The degree of exsiccosis does not always correspond to the severity of the child's condition. Sometimes clinical symptoms at 2 degrees of dehydration of isotonic type of exsiccosis cause hypovolemic disturbances in an organism of the patient.

Table 4

Clinical and laboratory evaluation of the hypertensive type of dehydration

Indicator	1 degree	2 degree
Consciousness	not broken or excited	somnolence, sopor
The temperature	subfebrile	febrile
Convulsions	absent	clonic-tonic
Thirst	moderate	sharply expressed
The skin	dry, warm	pale, dry, warm, wrinkled
Mucous shell	dry	dry, bright
The tongue	wet	dry, red
The voice	not changed	hoarse
The turgor of the tissue	preserved	reduced
The eyeballs	not changed	soft
Breathing	not disturbed	tachypnea
Heart rate	moderate tachycardia	significant tachycardia
Blood pressure	according to the standard	increased
CVP	according to the standard	according to the standard
Paresis of the intestines	absent	absent
Diuresis	reduced	oliguria
Na, plasma	148,6±0,24	159,6±2,6

Table 5

Clinical and laboratory evaluation of hypotonic type of dehydration

Indicator	1 degree	2 degree	3 degree
1	**2**	**3**	**4**
Consciousness	somnolence	somnolence, coma I	coma II-III
The temperature	subfebrile	subfebrile, hypothermia	hypothermia
Convulsions	absent	clonic-tonic	clonic-tonic
Thirst	absent	absent	absent

1	2	3	4
The skin	pale, cold	acrocyanosis, cold	common cyanosis, cold
Mucous shell	dry	dry, bright	dry, bright
The tongue	besieged	dry	dry, red
The voice	not changed	attenuated	aphonia
The turgor of the tissue	preserved	reduced	sharply reduced
The eyeballs	not changed	soft	soft
Breathing	not disturbed	tachypnea	paradoxically breath
Heart rate	moderate tachycardia	significant tachycardia	bradyarrhythmia
Blood pressure	reduced	low	less than 35-40 mm Hg
CVP	according to the standard	lower standard	negative value
Paresis of the intestines	I degree	II degree	III degree
Diuresis	oliguria	oligoanuria	anury
Na, plasma	131,5±0,14	127,4±0,14	117,5±0,89

Table 6

Clinical and laboratory evaluation of isotonic type of dehydration

Indicator	1 degree	2 degree	3 degree
1	**2**	**3**	**4**
Consciousness	not broken	somnolence, coma I	coma II-III
The temperature	subfebrile	subfebrile, hypothermia	hypothermia
Convulsions	absent	clonic-tonic	clonic-tonic
Thirst	absent	absent	absent

1	2	3	4
The skin	moderately dry	dry, bright	dry, bright
Mucous shell	dry	dry, bright	dry, bright
The tongue	dry, saliva viscous	dry	dry, red
The voice	not changed	attenuated	aphonia
The turgor of the tissue	reduced	reduced	sharply reduced
The eyeballs	soft	soft	soft
Breathing	not disturbed	tachypnea	paradoxically breath
Heart rate	moderate tachycardia	significant tachycardia	bradyarrhythmia
Blood pressure	according to the standard	reduced	low
CVP	according to the standard	lower standard	negative
Paresis of the intestines	absent	I degree	I degree
Diuresis	standard	oliguria	oligoanuria
Na, plasma	standard	standard	standard

Toxicity in AII in children can manifest itself in the form of neurotoxicosis. The presence of this syndrome should be considered when the symptoms of CNS damage come to the fore. The syndrome develops very quickly. Characteristic of neurotoxicosis is a steady rise in temperature above 39 ° C. There are two phases in the development of neurotoxicosis: irritative (excitation phase) and soporous-adynamic (suppression phase). In AII, the factor that causes neurotoxicosis is the endotoxin of gram-negative microflora (bacteria of the genus Salmonella, Escherichia, Shigella and other opportunistic bacteria). Toxicosis requires the elimination of the elimination function of the reticuloendothelial system (RES), which can develop during a massive infectious invasion by virulent bacteria. Insufficiency or blockade of RES leads to avalanche-like accumulation in the body of endotoxin, biologically active amines, products and mediators of inflammation, lysosomal enzymes. They damage the vascular wall, cell membranes, cause disseminated

intravascular coagulation. The process occurs in 3 pathogenetic stages. **The first stage** is mainly intracapillary disorders, spasm of capillaries with violation of blood rheology. **The second stage** - mainly extracapillary changes with increasing permeability of the vascular wall, increasing hydrophilicity of collagen connective tissue and interstitial edema, intravascular coagulation with the appearance of the first signs of increased bleeding. **The third stage** - the predominant damage to cell membranes due to energy deficiency and disruption of membrane transport, edema and cell death.

The listed pathogenetic stages correspond to a clinical picture of toxicosis, and first of all it concerns CNS where at first there are functional frustration of blood circulation, then cerebrospinal hypertension and at last hypostasis of a brain. Thus, neurotoxicosis in an infectious disease has clinical signs of CNS damage in combination with peripheral vascular insufficiency syndrome. It should be borne in mind that in AII of bacterial etiology, as a rule, vascular insufficiency can prevail, reaching even the level of **toxic shock syndrome (TSS).**

Toxic shock syndrome develops very quickly, sometimes even suddenly. Deterioration occurs against the background of a steady rise in body temperature above 39 ° C - hyperthermic syndrome. The child is characterized by agitation, anxiety, tremors of the hands, explosion and tension of the big toe, rigidity of the occipital muscles, with increasing severity - generalized seizures of clonic-tonic nature. From the cardiovascular system there is tachycardia, pale skin (may be a slight cyanosis), increased blood pressure, intense pulse, heart sounds are clear at first, the emphasis of 2 tones on the pulmonary arteries (hypertension in the small circulation). Breathing becomes more frequent. Percussion - box shade of sound (emphysema of the lungs), auscultatory - hard breathing, dry rales. Diuresis is reduced (oliguria). This is the first phase of neurotoxicosis, followed by the second (soporous) phase. In this case, it is clinically observed: the child's consciousness is preserved, but somewhat depressed, inhibited, freezes in one position, responds only to strong stimuli. The skin is pale with a "marble" pattern, cyanosis increases. Body temperature rises to 40 ° C. Tachycardia increases, hyperventilation increases, which leads to water loss and excretion of large amounts of CO2, which in turn can cause respiratory alkalosis and be one of the causes of convulsive syndrome. With further development of toxicosis passes into the stage of **coma: midbrain**, then stem (**bulbar**), and then **terminal**. Consciousness is lost, muscular hypotension develops, motor activity disappears, and seizures of a clonic-tonic nature often appear.

Peripheral blood flow insufficiency progressively increases: the skin is gray-cyanotic, hemorrhagic elements (the result of DIC syndrome), “hypostasis” (by the type of cadaveric spots) can be observed. Blood pressure decreases, heart sounds are deaf, tachycardia is replaced by bradycardia (prognostic negative sign, which indicates edema and swelling of the brain), tachypnea changes to bradycardia, shallow breathing, periodic. There is vomiting of "coffee grounds," paresis of the intestines and sphincters. Complete areflexia develops. In the lungs a picture of edema, pink foam on the lips. Cyanosis of the skin increases, hypothermia develops, the skin becomes cold, covered with sour, sticky sweat. **The terminal phase** (coma) is characterized by complete areflexia, cessation of seizures, muscular hypotension, disappearance of the swallowing reflex, depression, and then cessation of breathing and cardiac activity.

Clinical manifestations of the "**local syndrome**" in acute intestinal infections with symptoms of gastritis, enteritis, colitis, gastroenteritis, enterocolitis, gastroenterocolitis, enterocolitis. At localization of pathological process only at the level of a stomach (**gastritis**) there are pains in epigastric area, feeling of heaviness, nausea, vomiting or vomiting, manifestations of intoxication due to absorption, into the blood of endo- or exotoxins of bacteria and toxic metabolic products.

With the defeat of the small intestine (**enteritis**), in addition to pain, flatulence often occurs, the process of digestion and absorption of food ingredients, water and electrolytes due to inflammation or hypersecretion of the epithelium, increased motility - all this leads to diarrhea. With enteritis, feces will be liquid, homogeneous, watery, in large quantities, frequent, retain fecal character and odor, in the event of a fermentation process - foamy, splashing. Impurities in the stool may contain a small amount of pieces of clear mucus and greens.

When involved in the pathological process of the large intestine (**enterocolitis**), in addition to pain, manifestations intoxication (**toxicosis**), feces can be sparse (from mushy to watery), homogeneous, often foul-smelling, when there are putrefactive processes, contain a large amount of turbid mucus, sometimes blood, green like a "swamp".

In the presence of "**distal colitis**" there are cramping pains more often in the left iliac region, spasmodic and painful sigmoid colon, there is pliability or gaping of the anus, tenesmus, ie painful urge to defecate. Stools are usually liquid, fecal, scanty, with lots of cloudy mucus, greens, streaks of blood. Often the stools lose their fecal character and fecal odor and are a clot of cloudy mucus, pus with greens and

blood ("**rectal saliva**"). Painful, often false urges to defecate - a characteristic feature of distal colitis, regardless of its etiology. Clinical criteria and paraclinical studies are used to establish the diagnosis.

Diagnostic criteria for secretory diarrhea:

• acute onset;
• vomiting;
• constant aching abdominal pain;
• liquid copious stools with water and residues of undigested food;
• possible catarrh of the upper respiratory tract in AII of viral etiology;
• development of toxicosis with exsiccosis.

Criteria for the severity of secretory diarrhea:

• severity of symptoms of intoxication;
• nature, frequency of bowel movements;
• degree of dehydration;
• the presence and nature of complications.

Paraclinical studies of secretory diarrhea:

• coprogram - mucus, lymphocytes, signs of enzymatic dysfunction;
• fecal sowing - isolation of the pathogen and clarification of its type;
• culture of blood, vomit, gastric lavage, food residues;
• virological examination of feces - virus isolation;
• determination of antibodies to pathogens by ELISA (if possible);
• determination of antibodies by passive hemagglutination - increase in antibody titer (if possible).

Diagnostic criteria for invasive diarrhea:

• acute onset;
• increase in temperature to 38-40 ° C;
• symptoms of infectious toxicosis, toxic encephalopathy (disturbance of consciousness, convulsions);
• vomiting;
• cramping abdominal pain, tenesmus or their equivalents;
• spasmodic colon, sigmoid colon;
• liquid small fecal green stools;
• impurities in the feces of manure, mucus, blood.

Criteria for the severity of invasive diarrhea:

• severity of symptoms of intoxication;
• nature, frequency of bowel movements, the presence of hemocolitis;

• intensity of abdominal pain, the presence of complications.

Paraclinical studies of invasive diarrhea:

• coprogram - mucus, leukocytes, erythrocytes, cylindrical epithelial cells;

• fecal culture - excretion of the pathogen (in salmonellosis - culture of urine, blood);

• agglutination reaction, passive hemagglutination reaction (increase in antibody shooting in dynamics).

Table 7

Differential diagnostic criteria of AII in children

Signs	Salmonellosis	Shigellosis	Escherichia coli (EPE *, ETE **)	Rotavirus infection	Infection Staphyloco-ccus
1	**2**	**3**	**4**	**5**	**6**
Age	up to 2 years	from 2 to 7 years	EPE-1-3 years, ETE - all groups	up to 3 years	3-6 months
t^0C	febrile 1-4 days max lasts until 7 days	febrile from 1 day max lasts up to 5 days	subfebrile or febrile from 5-7 days max lasts 1-2 weeks	subfebrile lasts 2-4 weeks	subfebrile or febrile lasts 3-5 weeks
Vomiting	1-2 times a day, "unmotivated", does not appear every day, lasts the whole acute period	only the first day 1-2 times	at EPE - 2-5 times, max - for 5-7 days, lasts 7-10 days; at ETE - 1-2 times a day, lasts 3-7 days	multiple, lasts 1-2 days	1-2 times a day, lasts up to 5-10 days

1	2	3	4	5	6
Emptying	from 1 day, smelly, with mucus, green, in the form of "swamp"	first fecal in nature, then liquid with admixtures of mucus, blood, pus, green, characteristic “rectal spit"	EPE - frequent yellow or orange, with plenty of water, feces with a small amount of mucus, sometimes splashing; ETE - frequent, watery "watery diarrhea"	from the first day frequent, liquid, mushy, watery, yellow, sometimes discolored, with traces of clear mucus	from the first day of the disease frequent, watery with a lot of turbid mucus, sometimes blood impurities
Distal Colitis Syndrome	in rare cases, occurs on day 3-5 of the disease	characteristic , occurs on the first day of illness, may be a gap of the anus	absent	absent	absent
Toxicoxicosis	happens often	not typical	1-2 degree	1-3 degree	possible development
Neurotoxicosis	happens often	typical	occurs in severe EPE; for ETE is not typical	not typical	not typical
Hepatolienal syndrome	typical	not typical	not typical	not typical	not typical
Flatulence	typical	in children 1 year of age	not typical	not typical	not typical

1	2	3	4	5	6
Inflammatory changes in the coprogram	characteristic in the presence of a colitis variant	typical	not typical	not typical	characteristic in the presence of a colitis variant
Changes in peripheral blood	leukocytosis, neutrophilia with a shift to the left, monocytosis, in severe forms 1 year of life - anemia, erythropenia, aneosinophi-lia	in severe forms – leukocytosis neutrophilia, increased ESR	EPE - in 1/3 of patients leukocytosis, neutrophilia, increased ESR; ETE - changes are not typical	normocytosis or leukopenia, lymphocyto-sis, normal ESR	leukocytosis, neutrophilia with a shift to the left, increased ESR, in young children is a hypochromic anemia

TREATMENT OF AII

Treatment of AII in children should be carried out according to the protocols recommended by the Ministry of Health of Ukraine, which includes: diet therapy, antibacterial therapy, rehydration therapy, detoxification therapy, adjuvant therapy, enterosorption, probiotic therapy, enzyme therapy. Therapeutic nutrition is an important component of therapy at all stages of the disease.

I. Breastfeeding children with AII

It is carried out taking into account the severity and course of intestinal infection, the presence of toxicosis (exicosis), the nature and intensity of intestinal dysfunction. It is important to remember that children, especially infants, develop secondary intestinal lactase deficiency due to acute intestinal infections.

1. In children of the first year of life, breastfeeding should be maintained, because breast lactose is well tolerated by children with diarrhea.

2. Children of the first year of life on artificial feeding are transferred to low-lactose (lactose-free) mixtures, it is possible on a soy basis (though not all authors agree with it). In the absence of these mixtures, children are fed adapted mixtures. If they cannot be used, unadapted mixtures are used: dilution of cow's milk with concentrated rice broth (CRB) in a ratio of 2: 1 (children under 2 months) or 3: 1 with 3% sugar or 5% glucose. Enveloping the mucous membrane, CRB reduces its irritation and helps to normalize peristalsis.

3. In the acute period of the disease, the tactics of diet therapy depends on the presence and degree of toxicosis (exicosis).

4. If there are no signs of toxicosis (exicosis), then the number of feedings and the interval between them is the same as before the disease. With the symptoms of enteritis, the volume of food decreases by 1/3 - 1/2, with colitis - by 1/2 - 2/3 of the daily volume of the diet. As the condition improves, the age of nutrition is restored within 3-5 days. To do this, increase the daily volume of food by 100-150 ml daily, distributing this amount evenly for each feeding.

5. At toxicosis (exicosis) of the I degree of children on natural feeding continue to be applied to a breast with the same interval, as to a disease. Children on artificial feeding are prescribed a break in feeding for 6-8 hours, during which the child is given water, tea, solutions for rehydration. After unloading, dosed feeding begins with a reduction in daily food intake by 50%. Within 4-5 days, the amount of food is restored to normal, while continuing to maintain oral rehydration.

6. At toxicosis (exicosis) of the II-III degree appoint (depending on degree of toxicosis, trophic of the child, tolerance to food) on 10-50 ml of food (expressed breast milk or milk mix) for each feeding. The number of feedings is increased to 8-10 per day, the interval between feedings is reduced to 2.5-2 hours. The volume is restored to normal carefully and not more than 100 ml of food per day. Starting on day 3 of the illness, a breastfed baby can be breastfed for 1-2 feedings.

7. From the same day, children who received supplementary feeding or supplementary feeding may receive adapted fermented milk products, and in their absence - unadapted milk formulas (kefir), first in a dilution of 2: 1 with CRB and then in whole form.

8. After the disappearance of intestinal dysfunction and the restoration of the age-old physiological volume of food, children who received complementary foods before the disease are given porridge on water, mashed potatoes on water; as well as products with a high content of pectin (baked apples, apple and carrot puree, bananas), which is especially indicated in AII with colitis. From 5-6 days appoint

cottage cheese, starting with 1 teaspoon a day, to 6 months of age are brought to 40 g per day, to 1 year of age - 80 g. In the acute period and in the period of convalescence whole milk is not prescribed, porridge and puree are prepared on water.

The diet of children older than 1 year should consist of fermented milk products (kefir, acidophilus milk), rice porridge on water, soups with vegetable, cereal broth. Homemade cheese, meat, fish and white rusks are prescribed for 3-4 days. Vegetables and cereals are boiled until soft and rubbed. Meat and fish are served in the form of puree, souffle, steam cutlets, meatballs. From the first days the products rich in pectin substances are shown. At normalization stools for 7-10 days, children are transferred to the physiological table. Exclude from the diet whole milk, foods rich in fiber and cause flatulence (rye bread, beans, sauerkraut, beets, cucumbers, radishes, pears, grapes, oranges, tangerines, plums), as well as limit fats.

II. Antibacterial therapy

Indications for antibacterial therapy
with secretory diarrhea are:

1. Children of the 1st year of life with all forms of severity.
2. Children older than 1 year of life:
 • with immunodeficiency states;
 • HIV-infected;
 • who are on immunosuppressive therapy;
 • with hemolytic anemia.
3. Children with cholera, regardless of age.

Indications for antibacterial therapy with invasive diarrhea are:

1. All severe forms of the disease, regardless of age.
2. Hemocolitis, regardless of the age of the child and the severity of the disease.
3. At moderately severe forms to children till 1 year.
4. Children older than 1 year of age with:
 • immunodeficiency states;
 • HIV-infected;
 • who are on immunosuppressive, long-term corticosteroid therapy;
 • with hemolytic anemia;
 • shigellosis, amebiasis;
 • in the presence of bacterial complications and extracellular foci of infection.

Tactics of antibacterial therapy for secretory diarrhea

Prescribe antibacterial drugs for moderate
severe and severe forms in combination with sulfonamides with trimethoprim, cephalosporins III-IV generations. In cholera, the drugs of choice are erythromycin, nalidixic acid, nitrofuran drugs, combinations of sulfonamides with trimethoprim, children older than 8 years - tetracyclines. With cholera antibacterial drugs are prescribed after the first stage of rehydration 3-6 hours after hospitalization.

The course of antibacterial therapy is 5-7 days.

Tactics of antibacterial therapy at invasive diarrhea in children

All antibacterial drugs used are conditional are divided into three rows.

Drugs of the 1st row: nitrofuran derivatives, combined preparations of sulfonamides with trimethoprim. Appointed for mild and moderate forms of the disease empirically at the first meeting with the patient.

Drugs of the 2nd row: drugs of nalidixic acid, aminoglycosides of the III generation (semi-synthetic aminoglycosides) - amikacin, netilmicin and others. They are not recommended for use in salmonellosis, because in phagolysosomes containing salmonella, a low pH level is created, at which antibiotics of this group lose activity. These drugs are prescribed in case of inefficiency of 1 row, in moderate and severe forms, in late hospitalization as starting drugs and are used mainly in the hospital.

Drugs of the 3rd row: aminopenicillins protected from I-lactamase pathogens, cephalosporins of the 3rd generation, carbopenems, fluoroquinolones (only on vital signs). Prescribed in case of ineffectiveness of drugs of 2 series, in severe forms, in moderate-severe forms in children with immunodeficiency, children from infants born to parents - drug addicts and alcoholics, in the event of secondary bacterial complications, the presence of extraintestinal foci of infection, in infection. Used only in the hospital.

Table 8

Antibiotics used in the treatment of AII in children

Drug	Daily dose mg / kg	Way introduction	Multiplicity, once / day
1	**2**	**3**	**4**
SEMI-SYNTHETIC PENICILLINS			
Aminopenicillins (penicillin-sensitive)			

1	2	3	4
Ampicillin (pentrexil, mescillin, roscillin, standardicillin, ampicide)	100-200	Inside, in/m, in/in	4-6
Amoxicillin (amoxil, apo-amoxy, oklamox, solutob, grunamox, chicontsil)	20	Inside, in/m	3
Bacampicillin (penglob, penbak)	12,5-25	Inside	2
Penicillins resistant to β-lactamases			
Ampiclox (ampicillin + cloxacillin)	50-150	Inside, in/m	4-6
Sultamycillin = ampicillin + sulbactam (unazin)	25-50 to 150	Inside, in/m, in/in	3-4
Amoxicillin clavulanate = Amoxicillin + clavulanic acid (amoxiclav, augmentin, clavocin)	40	Inside, in/m	4
Thimentine = ticarcillin + clavulanic acid	200-300	in/in	4-6
Cyclic ureidopenicillins			
Meslocillin (bypen)	75-150	in/m, in/in	3
Azlocillin (securopene)	50-150	in/m, in/in	3-4

AMINOGLYCOSIDES			
A. Streptidine			
1	**2**	**3**	**4**
Streptomycin	15-30	in/m	2
Kanamycin	30-50	in/m, in/in	2-3
Gentamicin	3-5-7	in/m, in/in	2-3
Sisomycin	3-5-7	in/m, in/in	2
Tobramycin	3-5-7	in/m, in/in	2-3
B. Semi-synthetic aminoglycosides			
Amikacin	10-15-20	in/m, in/in	2-3
Netilmicin	5-7,5	in/m, in/in	2-3
Dibecacin (nipocin)	2-4	in/m, in/in	2-3
Isepamycin (isepacin)	15	in/m, in/in	2
Paromomycin (gabbroral)	10	Inside	2-3
POLYPEPTIDES			
Polymyxins			
Polymyxin-M	100 тис. MO/kg	Inside	3-4
Polymyxin-E	100 тис. MO/kg	Inside	3
Polymixin-B (sulfate)	1,2-2,4	in/m, in/in	3-4 in/in-2
CEPHALOSPORINS			
III generation			
Cefotaxime (claforan, clafotaxime, talcef, cefantral, cefotam)	100-150	in/in	3-4
Cefoperazone (cefobid, dardum, medocef)	60-150	in/in	2-4
Ceftazidime (fortum, tasicef, kefadim)	50-75-100	in/in	2-3

1	2	3	4
Ceftriaxone (longacef, ificef, lendacin, rocephin, ofromax, torocef, cefaxone)	50-75-100	in/m, in/in	1-2
Generation III + β-lactamase inhibitor			
Cefoperazone + sulbactam (sulperazone)	40-80-160	in/m, in/in	2-4
IV generation			
Cefpirom (kaiten)	30-60-100	in/in	2
MONOBACTS			
Aztreonam (azactam)	30-50	in/m, in/in	3-4
CARBOPENEMS			
Thienam (imipenem + cilastatin)	20-40	in/m, in/in	4
Meronem (meropenem)	12-20 to 40	in/m, in/in	3
FLUOROHINOLONES			
Norfloxacin (nolicin, bactinor, girablok, negaflox, norbactin, norilet, normax, norfaxine, utibid, urobacid, spectra)	7-10 to 20	Inside	2
Ofloxacin (Zanozin, Kirol, Oflo, Oflin Tarivid)	7-10 to 20	Inside, in/m, in/in	2
Pefloxacin (abactal, peflacin, pefloxacin)	8-10 to 20	Inside, in/m, in/in	2

1	2	3	4
Lomefloxacin (maxaquine)	5-10	Inside	1
Flecloxacin (quinodis)	5-10	Inside, in/in	1
Ciprofloxacin (ciprinol, ciprobay, ciprobid, ciprolet, cipronate, ciprosan, ciprosol, tsiprotsinal, tsiteral, tsifran, ifitsipro, kvintor, quipro, cefobak, tseprova, medociprin)	7.5-15	Inside, in/in	2
Enoxacin (enaxor)	7.5-15	Inside	2
Combined preparations of sulfonamides with trimethoprim			
Sulfamethoxazole / trimethoprim (co-trimoxazole, bactrim, sepetrin, biseptol, oriprim), triseptol, soptrim, sumetrolim, groseptol	from 5-6 to 20	Inside, in/in	2
Sulfamonomethoxine / trimethoprim (sulfatone)	from 5-6 to 20	Inside	2
Sulfamerazole / trimethoprim	from 5-6 to 20	Inside	2
Nitrofuran derivatives			
Furazolidone	8-10	Inside	4

1	2	3	4
Nifuroxazide	suspension: 2-6 months - 0.5-1 tsp, 6 months-6 years-1 tsp, older than 6 years - 1 hour spoon tablets: children older than 6 years - 2 tab.	Inside	2-4

The course of antibacterial therapy is 5-7 days. With giardiasis, amebiasis - use metronidazole, entero-sediv. Indications for replacement of the antibacterial drug are its ineffectiveness for 3 days. In severe septic forms, a combination of 2-3 antibiotics is possible. The most rational combinations:

1. Semi-synthetic penicillins - with aminoglycosides, polymyxins, cephalosporins, monobactams, fluoroquinolones, metronidazole.

2. Cephalosporins - with semi-synthetic penicillins, aminoglycosides, polymyxins, monobactams, fluoroquinolones, metonidazole.

Russian specialists in the treatment of diarrhea instead of antibiotics or in parallel with them widely use immunoglobulins of enteral origin and bacteriophages.

Immunoglobulins for enteral use

1. Lactoglobulin anticolitic

Contains antibodies to pathogenic Escherichia coli serovars O26, O55, O111, O119 and Proteus (diluted at least 1: 800), which have antimicrobial and toxin-neutralizing action. It is used orally, 1 dose 2 times a day, diluted with chilled boiled water at the rate of 10 ml per 1 dose of the drug before use. The course of treatment is 7-14 days. If necessary, the dose and duration of therapy can be increased.

2. Antirotavirus immunoglobulin

Contains antibodies against rotavirus with a titer in RNA of not less than 1: 640. It is used for rotavirus and rotavirus-bacterial infections through the mouth 20-30 minutes before meals. Daily dose: children under 1 year - 1 ampoule 2 times a

day, 1-3 years 1 ampoule 3 times a day, older than 3 years - 2 ampoules 2 times a day. The dose can be increased 1.5-2 times.

3. Complex immunoglobulin preparation for oral administration.

It is characterized by a high concentration of antibodies to Shigella, Salmonella, Escherichia coli, rotavirus. Before use, dilute with chilled boiled water, apply orally 30 minutes before meals 1 dose 1-2 times a day. The course of treatment is 5 days. It is possible to increase the drug by 2-3 times (1 dose 3-4 times a day).

4. Bacteriophages: shigellosis, coliprotein, abdominal typhoid, cholera

Drugs cause selective lysis of certain microbes. Available in dry and liquid form. They are applied orally, in enemas, subcutaneously, intramuscularly, in the form of rinses, irrigations, lotions, injected into purulent cavities and moisten tampons, etc.

III. Rehydration therapy

The basis of treatment of AII in children is timely and adequate rehydration therapy - compensation for water and electrolyte losses. Proper rehydration therapy is a priority in the treatment of GCI, both in secretory and invasive diarrhea. Today, rehydration is divided into oral and parenteral. To carry out rehydration it is necessary to determine:

1. Daily fluid requirement.
2. Type and degree of dehydration.
3. Current pathological losses.
4. The total level of fluid deficiency.
5. Determine the method of rehydration.

Procedure

I. Determine the degree of exsiccosis. To do this, you need to know the fluid deficit, which is calculated by the percentage of weight loss from the time of the disease to the time of examination (I st. - weight loss up to 5%, II st. - up to 10%, III st. - up to 15%). If body weight is unknown before the disease, the degree and type of dehydration is determined by clinical signs (Table 2). Even easier - in this case, the fluid deficit is taken as 10%.

II. Determine the method of rehydration therapy. At an exicosis of the I-II centuries. In the absence of unrestrained vomiting and severe anorexia, the method of oral rehydration may be sufficient.

Oral rehydration (OR)

Oral (oral) rehydration in GCI should be the first treatment, which is carried out at home with the first symptoms of the disease. Timely PR can effectively treat most children at home, reduce the incidence of severe forms of exsiccosis.

Procedure

1. Apply solutions: "Glucosolan", "Oralit", "Gastrolit" - I generation; "Regidron" - II generation; "ORS 200 HIPP" - III generation. No need to use fruit juices, sweet carbonated drinks.
2. Rehydration is carried out in 2 stages:
• Stage I - recovery of lost fluid volume: in case of exsiccosis and the century. fluid volume - 50 ml / kg body weight, with second-degree esicosis. - 100 ml / kg body weight. The duration of the first stage is 4-6 hours. The volume of solution calculated for 1 hour of reception is poured into the graduated ware, give to the child through a pacifier, by means of a pipette, a teaspoon every 5-10 minutes. If you do not drink or vomit, the solutions are administered through a nasogastric tube drip using a system for intravenous administration at a rate of 10 ml / min. Length from ear to nose + from nose to xiphoid process of sternum.

Evaluation of effectiveness, further tactics

a. Rehydration is effective (disappearance of thirst, improvement of tissue turgor, skin elasticity, hydration of mucous membranes, increase in diuresis, disappearance of signs of microcirculation disorders)
- transition to the second stage (maintenance therapy).

b. Rehydration is not effective enough (signs of dehydration persist) - continue similar treatment for another 4-6 hours.

c. Rehydration is ineffective (dehydration increases, vomiting persists, profuse diarrhea, symptoms of toxicosis increase) - switch to infusion therapy (parenteral rehydration) by peripheral vein puncture or main vein catheterization.

• Stage II OR - maintenance therapy. If the amount of stool is not more than once every 2 hours, or not less than 5 ml of liquid feces per 1 kg of body weight for 1 hour

the volume of fluid is 5 ml / kg of body weight / hour. With severe diarrhea, fluid volume = 10 ml / kg body weight / hour. The calculated volume is distributed evenly throughout the day.

To avoid complications (tissue pastosity, decreased diuresis), especially in children with comorbidities, neurotoxicosis, expressed by colitis, it is advisable to replace half of the fluid administered with strong sweetened (3% sugar) tea, better green, with lemon, rose hip broth, raisin broth, rice, fruit (apple) broth. During the day of oral rehydration should increase the patient's body weight from 5% to 10%.

Contraindications to oral rehydration

1. Severe dehydration (weight loss of more than 10% in young children and more than 6% - older).
2. Vomiting that cannot be stopped within 2 hours of oral rehydration.
3. Paresis of the intestine.
4. Stupor, coma, infectious-toxic shock.
5. Oliguria and anuria, which do not disappear during the first stage of rehydration.
6. Metabolic alkalosis.
7. Inefficiency of oral rehydration during the day. With exicosis of the III century, unrestrained vomiting, anorexia, refusal from drinking, rehydration therapy begins with parenteral (intravenous) fluid administration (infusion therapy), combining it with oral rehydration.

Parenteral rehydration (infusion therapy)
Procedure

1. Assess the degree of exsiccosis. If the body weight before the disease is not known, then its loss as a result of dehydration is taken as 10%.
2. Determine the type of exsiccosis. Prior to laboratory confirmation should focus on clinical manifestations (Table 4,5,6). As a last resort, you can assess the situation isotonic type of exsiccosis.
3. Calculate the daily volume of fluid required by the child (according to the method of J. Dennis):
I degree of exsiccosis: up to 1 year - 140-170 ml / kg; 1-5 years - 100-125 ml / kg; older children - 75-100 ml / kg.

II degree of exsiccosis: up to 1 year - 160-180 ml / kg; 1-5 years - 130-170 ml / kg; older children – 110 ml / kg.

III degree of exsiccosis: up to 1 year - 200-220 ml / kg; 1-5 years - 170-180 ml / kg; older children – 120-150 ml / kg.

4. Calculate the daily volume of infusion:

I degree of exsiccosis - up to 40% of the total daily fluid volume;

II degree of exsiccosis - up to 60% of the total daily fluid volume;

III degree of exsiccosis - up to 80% of the total daily fluid volume.

If the child has pneumonia, the infusion volume should not exceed 50% of the required daily volume.

5. Calculate the volume and duration of the first infusion (fraction). The calculated volume of infusion should be administered during the day. However, in the absence of access to the main (subclavian, etc.), the fluid must be injected into the peripheral veins by the fractional method.

I degree of exsiccosis: duration of at least 4 hours, you can enter the entire calculated volume (per day).

II degree of exsiccosis: duration of infusion (I fraction) not less than 6 hours, enter 1/2 of the calculated daily volume of infusion; after 8-12 hours, conduct the second infusion (fraction).

III degree of exsiccosis: duration of infusion (I fraction) not less than 8 hours, enter 1 / 2-2 / 3 of the calculated daily volume of infusion; after 8-12 hours, conduct the second infusion.

6. Calculate the rate of fluid injection.

Fluid volume in ml / min. = total fluid volume (ml): (3 × number of hours (min.) of infusion).

For infants, the rate of intravenous administration is not more than 60-80 ml / h (not more than 14-16 drops / min.).

7. Choose solutions, determine their ratio and sequence of their introduction.

• The most optimal crystalloid solutions for parenteral rehydration in young children are 5% glucose solution and 0.9% sodium chloride solution. Colloidal solutions: plasma, 5-10% albumin solution, reopolyglucin, stabizol, refortan.

• Intravenous infusions are usually started with colloidal solutions equal to 1/3 of the total infusion volume. The daily volume of colloidal solutions is divided by the number of infusions. The dose of the drug per 1 injection should not exceed 15 ml / kg body weight. Rehydration of children with exicosis of I-II degree, accompanied by repeated vomiting, it is advisable to carry out glucose-saline solutions without the use of colloids.

• In the same way distribute between fractions daily solution of 5% of glucose and 0,9% of solution of sodium chloride.

• In the isotonic type of exsiccosis, the ratio of the volume of 5% glucose solution to 0.9 NaCl solution is 2: 1 (these solutions can be mixed in one vial, they can be administered simultaneously).

• In the hypertensive type of exsiccosis, the ratio of 5% glucose to 0.9% NaCl solution is 3: 1 (if these solutions are not mixed in one vial, the infusion is started with glucose solution).

• In the hypotonic type of exsiccosis, the ratio of the volume of 5% glucose solution to 0.9% NaCl solution is 1: 1 (if these solutions are not mixed in one vial, the infusion is started with NaCl solution).

• Potassium is a mandatory component of infusion therapy. In the hypertensive type of exsiccosis, potassium is administered in infusion solutions in the form of a 7.5 solution at a dose of 1-2 ml / kg per day; with isotonic and hypotonic - 3 ml / kg per day. Additionally, in all these situations, panangin (asparkam) is administered at a dose of 1 ml / kg per day. The daily dose of potassium preparations is evenly distributed between the fractions during the day. The concentration of potassium chloride in the infusion is not more than 0.5%. Contraindications for intravenous potassium - anuria or severe oliguria (less than 20 ml of urine per kg of body weight per hour).

• Add 1-2 ml / kg / day 10% solution of calcium gluconate and in exsiccosis of the III degree, expressed by micro disorders circulation, toxic, acidotic respiration, disturbances of consciousness - use 4% sodium bicarbonate solution at a dose of 4 ml / kg / day. The calculated amount of sodium bicarbonate is divided into 3-4 injections and administered intravenously drip together with glucose solution.

8. Evaluate the effectiveness of therapy. Proper infusion therapy is accompanied by:
• Elimination (reduction) of signs of exsiccosis in the first 24 hours (criterion - weight gain by 7-9% for the first day).
• Steady improvement of hemodynamics (criterion - normalization of blood pressure, heart rate).
• Restoration of microcirculation (improved color, normalizes skin temperature, stabilizes hourly diuresis).
• Improving the general condition of the child.

9. Calculate the amount of rehydration therapy in the following days: • Take into account the residual unfilled body weight deficit.
• Take into account the daily fluid requirement and its current pathological losses: with hyperthermia above 38 ° C add fluid at a rate of 10 ml / kg / day; with shortness of breath for every 10 breaths above the norm of 10 ml / kg / day; with vomiting and diarrhea - 20-30 ml / kg / day; with a decrease in urine output - 30 ml / kg / day. Age-old daily water requirements: up to 6 months - 120-100 ml / kg; 6 months - 2 years - 100-80 ml / kg, older than 2 years - 80-40 ml / kg.

In all cases, you should try to move as quickly as possible from parenteral rehydration to oral maintenance rehydration therapy.

IV. Detoxification therapy

It is performed in the presence of a pronounced intoxication syndrome and the occurrence of infectious toxicosis. In mild forms, the use of solutions for oral rehydration is sufficient. In moderate and severe forms, intravenous administration of 5% glucose solution, 0.9% sodium chloride solution, colloidal solutions (albumin, reopolyglucin, refortan, stabizol) is used in parallel with oral rehydration. The total volume of infusion is 50-100 ml / kg / day. The ratio of colloids to crystalloids is 1: 2. It is more expedient to begin infusion with introduction of colloidal solutions in a single dose to 20 ml / kg. Then enter solutions of glucose and sodium chloride in a ratio of 1: 1. The total volume of fluid, including rehydration, should not exceed 150-160 ml / day.

V. Adjuvant therapy

1. Enterosorption

• Enterodesis. The solution is prepared immediately before use (1 teaspoon of powder is dissolved in 100 ml
chilled boiled water) Used for children per os: up to 1 year - 20 ml / day, 1-3 years - 30 ml / day, older than 3 years - 50 / ml / day. The daily dose is divided into 3-4 doses.
• Polyfepan. Available in powder form, always in moist form. Infants are prescribed 1 tsp, older - 1 dessert spoon 2-3 times a day.
• Smectite. Available as a powder in sachets. Dilute with water before use. Children under 1 year are prescribed 1 sachet per day, older 2-3 sachets.
• Enterosgel. Available in powder form. Infants are prescribed 1 teaspoon, older - 1 dessert spoon 2-3 times a day, pre-dissolved in 50-100 ml of chilled boiled water.

These drugs are most often used in domestic pediatric practice, the first place is given to Smectite. The duration of enterosorption in GKI - 5-7 days. The criterion for early withdrawal is the normalization of stools or their delay within 2 days.

2. Probiotic therapy

In the treatment of AII it is necessary to correct dysbiotic disorders of the intestinal microbiocenosis using probiotics. Probiotics are drugs that contain live microorganisms. They are divided into:
• monocultures of intestinal normoflora (Bifidum-bacterin, Colibacterin, Lactobacterin);
• monocultures of normoflora enriched with vitamins (Laktovit Forte);
• self-eliminating antagonists (Bactisubtil, Enterol, Biosporin, A-Bacterin);
• combined drugs - obligate flora + optional (Yogurt, Bificol, Linex);
• complex drugs - microorganisms + components to enhance the therapeutic effect (Bifi-forms).

Dry bifidumbacterin - contains lyophilized bifidobacteria. To children till 6 months of life to appoint on 2-2,5 doses 2 times a day for 30 minutes before feeding; from 6 months up to 1 year - 5 doses 2 times a day; from 1 year to 5 years - 6-8 doses 2 times a day; older than 5 years - 8-10 - 2 times a day. The course of treatment is 14-21 days.

Lactobacterin is a mixture of lyophilized lactobacilli. Children under 6 months of age should be prescribed 1-2 doses 2 times a day 30 minutes before feeding; from

6 months up to 1 year - 2-3 doses 2 times a day; from 1 year to 5 years - 3-5 doses 2 times a day; older than 5 years - 5-6 - 2 times a day. The course of treatment is 14-21 days.

Dry Colibacterin is a dried culture of Escherichia coli. Children from 6 months. up to 1 year - 3 doses 2 times a day; from 1 year to 5 years - 3-5 doses 2 times a day; older than 5 years - 5-10 doses - 2 times a day. The course of treatment is 21-30 days.

Enterol - lyophilized Saccharamyces boulardii. Apply 1-2 capsules per day.

Dry biosporin - living cells of B. subtilis, B. licheniformis. Biobactone - contains lyophilized culture
acidophilic bacillus.

Sporbacterin is a live culture of Escherichia coli.

Bificol is a combination drug containing lyophilized Escherichia coli and bifidobacteria. Children from 6 months. up to 1 year - 3 doses 2 times a day; from 1 year to 5 years - 3-5 doses 2 times a day; older than 5 years - 5-10 doses - 2 times a day. The course of treatment is 21-30 days.

Bifi-form is a combination drug containing lyophilized bifidobacteria and enterococci. Assign 1 capsule 3 times a day for 10-14 days.

Linex - contains Bifidobacterium infantis, Lactobacillis acidophilus, Streptococcus faecius. Children from 2 to 12 years - 1-2 capsules 3 times a day.

In the acute period of AII probiotics are used as etiotropic drugs in the absence of antibacterial drugs
in appointments. The course of probiotic therapy in the acute period lasts 7-10 days. During the period of convalescence in order to restore the normal intestinal microflora, physiological probiotics are used for 3-4 weeks.

Prebiotics are drugs that contain products of microbial metabolism. They are used to stimulate the growth of intestinal microflora, both alone and in combination with probiotics. Prebiotics are divided into:

• products of microbial metabolism of bacteria that form lactic acid (Hilak Forte);
• fructooligosaccharides (Dufalak, Normaze, Portalak).

Hilak-forte - contains lactic acid, products of metabolism of the small and large intestine, forming lactose, amino acids. Assign 10 drops 4 times a day.

Normaze - lowers the pH of the large intestine, the concentration of putrefactive bacteria and stimulates the growth of bifidobacteria.

3. Enzyme therapy

Enzyme preparations are divided into:

• drugs containing components of the gastric mucosa (abomin, acidin-pepsin, pepsidil, pepsin);

• pancreatic enzymes (pancreatin, menzim-forte, creon, pancitrate, oraza, solizim, somilase, nigedase);

• preparations containing pancreatin, bile, hemicellulose (digestal, cadistal, festal, kotazim-forte, panstal, rustal, enzistal);

• combined enzyme preparations (combicin, panzinorm-forte, pancreoflet);

• drugs containing lactose (tilactase, lactrase).

Enzyme therapy should be carried out taking into account coprograms. If the amount of undigested fiber and muscle fibers in the child's stools is increased, then we observe gastric indigestion. In this case, use drugs that contain components of the gastric mucosa: Pepsidil, Abomin - 1 dessert spoon 3p. the day before meals.

If in the studied stools of the child the quantity of neutral fats is increased, then we speak about pancreatic indigestion. It is recommended to use pancreatic enzymes: pancreatin -0.1-0.15 to 1 year, 0.15-0.2 to 3 years, older 0.2 - 2-3 times a day 1 hour after meals.

If there is an excess of starch and fatty acids in the stool - enteric insufficiency of digestion. In this case, we also use pancreatic enzymes, in particular mesylate forte, creon.

At insufficient allocation of bile, in feces of the patient we observe not split fats, soap of fatty acids therefore it is desirable to apply the preparations containing pancreatin, bile, hemicellulose (digestal, kadistal, festal, kotazim-forte, panstal, rustal, enzistal).

4. With AII due to the occurrence of fermentation processes in the intestine may be excessive flatulence, so with bloating of the intestinal loops, colic, it is recommended to prescribe a decoction of dill, Espumizan 25 drops 3-4 times a day after feeding.

5. Asymptomatic therapy if necessary.

Thus, the development of AII in childhood is a complex pathological process that leads to disorders in the health of the child. Treatment should be timely, comprehensive and justified from an etiopathogenetic point of view. Primary prevention of AII in children's groups should take a prominent place in order to preserve the health of the younger generation.

VIRAL HEPATITIS

In the structure of morbidity of children infectious diseases take the 6th place, and in the structure of mortality 4-5 place. Mortality from infectious diseases among villagers has almost equalized with that in cities and exceeds by 6.2%, when in 2001 this difference was 21.3%. The situation in Ukraine with regard to viral hepatitis is complicated. The incidence of children per 100,000 children was 105.8 at the viral hepatitis A in 2004; on viral hepatitis B- 2.95; at viral hepatitis C - 0.53 (data from the Ministry of Health of Ukraine). Mass outbreaks occur in areas where sewage and water systems do not meet modern requirements.

The term viral hepatitis (VH) includes viral diseases liver, in addition to hepatitis caused by cytomegalovirus, herpes, Epstein-Barr and adenoviruses.

Classification of viral hepatitis (according to MK-10 adopted by the WHO)

1. Acute hepatitis A.
 • Hepatitis A with hepatic coma.
 • Hepatitis A without hepatic coma.
2. Acute hepatitis B.
 • Acute hepatitis B with delta agent (confection) with hepatic coma.
 • Acute hepatitis B with a delta agent (coinfection) without hepatic coma.
 • Acute hepatitis B without delta agent (coinfection) with hepatic coma.
 • Acute hepatitis B without delta agent (coinfection) and without hepatic coma.
3. Acute delta (superinfection) of the hepatitis B virus carrier
4. Acute hepatitis C.
5. Acute hepatitis E.
6. Other specified viral hepatitis.
7. Chronic viral hepatitis.
 • Chronic hepatitis B with delta agent.
 • Chronic hepatitis B without delta agent.
 • Chronic hepatitis C.
 • Chronic hepatitis unspecified.
 • Other specified chronic viral hepatitis.

Clinical classification of viral hepatitis (Ministry of Health of Ukraine)

1. Etiological species: A, B, C, D, E, F, G.

2. Forms: inapparent, subclinical, non-jaundiced, jaundiced (cytolytic, cholestatic).

3. Cyclical course: acute, subacute, chronic.

4. Severity: mild, moderate, severe.

5. Complications: acute hepatic encephalopathy (precoma, coma); exacerbations and relapses (clinical, enzymatic, morphological); functional and inflammatory diseases of the biliary tract and bladder, induction of immunocomplex and autoimmune diseases.

6. Consequences: recovery, residual effects (asthenovegetative syndrome, posthepatitis hepatomegaly and hyperbilirubinemia), chronic hepatitis, liver cirrhosis, primary liver cancer.

VIRAL HEPATITIS A

Viral hepatitis A is an acute cyclic disease caused by an RNA-containing virus and characterized by short-term symptoms of intoxication, transient liver dysfunction, and lack of chronicity.

ETIOLOGY AND EPIDEMIOLOGY

The causative agent of viral hepatitis A is an RNA-containing virus from the picornavirus family. The disease is endemic and is an anthroponotic intestinal infection. The source is a sick person or virus carriers - convalescents. Viral hepatitis A is characterized by autumn-winter seasonality and periodic rises in morbidity every 3-5 years. Transmission of the virus occurs in the following ways: fecal-oral, contact-household, food and water, theoretically possible parenteral infection. After undergoing viral hepatitis A, a person develops a stable immunity throughout life. The virus can be detected in the patient's blood, urine and feces in the highest concentration in the pre-jaundice period, and in 4-5 days after the appearance of jaundice is almost undetectable. Children 1 year of age rarely get sick because they have immunity that is received from the mother. The maximum incidence of viral hepatitis A among children is at the age of 5-14 years.

Features of the pathogenesis of viral hepatitis A

The entrance gate of infection is the gastrointestinal tract.Penetrating into the small intestine, the virus enters the bloodstream and there is a short-term viremia. With the bloodstream, the virus enters the liver, in particular hepatocytes, where it replicates, which is accompanied by disruption of intracellular metabolic processes and damage to the membrane of hepatocytes. As a result of direct cytopathogenic action of the virus on the liver parenchyma there is a syndrome of cytolysis, all types of metabolism are disturbed. The pathogen has a high degree of immunogenicity. Along with the protein complexes removed from the cells, which act as autoallergens, the virus stimulates the T- and B-systems of the immune system, causing the formation of specific antibodies, as well as enhancing the mechanisms of autoaggression. But with hepatitis A, the defense mechanisms prevail over the mechanisms of autoaggression, as a result of which viral activity is neutralized, recovery occurs.

Pathological anatomy

In the prodromal period mitosis, proteinaceous dystrophies, in particular balloon dystrophies, and also scattered necrosis of hepatocytes with disturbance of structure of a liver parenchyma are found in hepatocytes. Simultaneously with dystrophy and necrobiosis, cell infiltration regenerates and decreases. The functional state of the liver is restored within 6-8 weeks, sometimes up to 4-5 months. The formation of residual liver fibrosis is possible.

Clinic of acute viral hepatitis A

The incubation period is from 7 to 50 days.The disease begins acutely and is characterized by cyclicity.

It is conventionally assumed that the acute course of viral hepatitis A lasts up to 3 months, prolonged from 3 to 6 months, chronic for more than 6 months.

The inapparent form of viral hepatitis is indicated in cases when only specific markers of the pathogen and the corresponding immunological changes are detected.

The subclinical form is characterized by immunological, biochemical and pathohistological changes, but has no clinical signs.

In the non-jaundiced form, in addition to immunological, biochemical, pathohistological shifts, various clinical symptoms appear except for jaundice.

The jaundiced form is accompanied by jaundice which is based on hyperbilirubinemia.

The pre-jaundice period lasts from 2 to 7 days. There are several variants of this period: dyspeptic, asthenovegetative, influenza, atralgic and mixed.

The dyspeptic variant is manifested by complaints of decreased appetite, dull pain in the epigastrium and right hypochondrium, fever up to 38-390C for 3-5 days.

Asthenovegetative variant is characterized by mood swings, sleep disturbances, headaches, irritability.

For the flu-like variant is characterized by fever up to 5-7 days, headache, no catarrhal manifestations.

Arthralgic variant in children is rare and is manifested by pain in the joints, muscles and bones.

An important diagnostic sign of the initial period is hyperenzymemia, there is an increase in ALT at the end of the incubation period, 5-7 days before the onset of jaundice. By the end of the pre-jaundice period, diuresis decreases, urine becomes dark brown, feces becomes light yellow. Hepatosplenomegaly is observed in almost all patients. The appearance of a jaundiced sclera indicates a transition to the jaundice period. Jaundice increases gradually. The sclera, hard palate and sublingual mucosa turn yellow first, then - skin of the face and torso. Improvement of the patient's condition is observed from the first days after the appearance of jaundice. In children often there is an abdominal syndrome. The duration of the jaundice period lasts 1-2 weeks, and on average 7-10 days. In viral hepatitis A, the bilirubin content exceeds the norm by no more than 4-5 times, mainly due to the bound fraction. The amount of bilirubin in the urine increases in parallel with the increase in bilirubinemia. On the contrary, the content of urobilin in the urine, which was sharply increased at the beginning of jaundice, falls and even disappears at the height of jaundice, and then increases again as it decreases. At the height of jaundice, the reaction of feces to stercobilin is negative. These data on changes in bilirubin metabolism are important for the icteric form of viral hepatitis A.

The content of total protein in the blood decreases only in the midst of severe disease (decreased albumin, but the concentration of gamma globulins increases). The presence of dysproteinemia can be established using sediment samples - thymol and sulema. In viral hepatitis, the thymol test increases and the sulem test decreases. Alkaline phosphatase levels also increase. In the general analysis of blood there is

leukopenia, lymphocytosis, eosinophilia, thrombocytopenia, ESR slowing. The final diagnosis of hepatitis

And can be confirmed by specific immunochemical (ELISA, RIA) or molecular biological methods. To the main markers of HAV infection include antibodies of immunoglobulin classes M, G to the virus (anti-HAV-IgG, anti-HAV-IgM), HAV-RNA virus antigen, viral RNA (HAV-RNA).

In clinical practice, the most common definition of anti-HAV-IgM to the capsid of the virus. Their definition indicates the course or recent infection. These antibodies appear in the serum in the pre-jaundice period, 3-5 days before the onset of the first symptoms, which circulate for several months (sometimes 6-12 months, indicating a delay in convalescence in hepatitis). Anti-HAV-IgG antibodies appear later, but at much higher titers. Their appearance characterizes the end of the infectious active process, sanitation of the body and circulates for life, providing lifelong immunity. Determination of viral antigen HAV is rarely used in clinical practice. Peak excretion of the virus is observed in the incubation and pre-jaundice periods, when it can be excreted in the feces, less often in the blood. Virus RNA can be detected in the blood and feces of patients. Determination of HAV-RNA indicates not only the presence of the virus, but also its active replication. The method is highly sensitive.

The convalescence period varies from 1-2 months to a year or more. The patient's condition is characterized by a slow improvement and attenuation of clinical symptoms: appetite appears, jaundice disappears, urine lightens, feces darkens, the size of the liver and spleen return to normal. Sometimes the first sign of recovery is polyuria, when diuresis increases by 2-4 times. But many convalescents persist with asthenic syndrome, a feeling of heaviness in the right hypochondrium and epigastrium, especially after eating. Reverse dynamics of functional changes of the liver such - first hyperbilirubinemia passes, then hyperfermentemia and finally dysproteinemia.

MAIN CRITERIA OF PRIMARY DIAGNOSIS OF HEPATITIS A

- Contact with the patient, taking into account the length of incubation.
- Group morbidity with the formation of epidemic foci of infection.
- Characteristic seasonality (September - November).
- Children (3-10 years), adolescents.

• Acute onset with a pronounced temperature reaction and manifestations of intoxication.

• Enlarged liver, often in combination with intoxication.
• Short pre-jaundice period (4-6 days), mainly with dyspeptic manifestations.
• Improvement of the general condition with the appearance of jaundice.
• Low jaundice.
• No chronization of the process.
• Increase in ALT, thymol test.

Determination in the blood of anti-HAV-IgM, and in the initial period and HAV-RNA. Determination of HAV-Ag in faeces.

With viral hepatitis A, the disease can end:

• recovery with complete recovery of liver structure;
• recovery with anatomical defects (residual fibrosis);
• formation of various complications of the gastrointestinal tract and biliary tract.

Children who have been in contact are not isolated, but medical supervision is established for a period of 35 days. For this period, children are not transferred to other groups or institutions, new children are accepted only with the permission of an epidemiologist with the timely introduction of gamma globulin.

Dispensary supervision of convalescents is 6 months. 10 days after discharge from the hospital is carried out biochemical blood test for bilirubin and transaminases. Re-examination and examination are carried out in 3 and 6 months, provided normal indicators are removed from the register.

Specific prophylaxis in Europe and the United States is carried out with recombinant and plasma vaccines starting at 12 months. Hepatitis A vaccination is not carried out in Ukraine. Immunoglobulin with a high titer of antibodies to hepatitis A virus - 1: 10000 and above is used for prophylaxis according to epidemic indicators. The drug is used no later than 7-10 days after the start of contact with children older than 1 year and pregnant women.

VIRAL HEPATITIS B

Viral hepatitis B is an acute infectious disease of the liver caused by a DNA-containing virus from the family of hepadnaviruses, characterized by varying degrees of severity, from asymptomatic to fulminant forms, has a tendency to exacerbation, cholestasis with delayed recovery of liver function and possible formation cirrhosis of the liver.

Etiology and epidemiology of viral hepatitis B

The source of infection is a sick person and a virus carrier. VHB patients are contagious already in the incubation period, which is from 1.5 to 6 months. Susceptibility to VHB is almost 100%. After the disease, there is a stable lifelong immunity.

Risk groups for hepatitis B and C

- Medical staff and blood transfusion staff.
- Long-term and frequent patients who receive parenteral manipulations.
- Patients of hemodynamics departments, oncohematological profile
- Patients with a history of blood transfusion.
- Patients with a history of surgery.
- Patients and staff of psychiatric hospitals.
- Newborns from mothers who have positive markers of viral hepatitis B or viral hepatitis C
- Family members of patients with chronic viral hepatitis B and C.

All known routes and factors of viral hepatitis B transmission are divided into natural and artificial. Among the natural ways of transmitting the virus distinguish: vertical, horizontal and sexual. "Vertical transmission" is divided into prenatal (transplacental) and intranatal. According to some authors, since 1990, the frequency of detection of HBsAg among pregnant women has increased 2.5 times. Infection of the child with the virus from the mother is essential because in newborns, the greatest risk of transformation of acute infection into a carrier, with the subsequent development of chronic disease.

All known routes and factors of viral hepatitis B transmission are divided into natural and artificial. Among the natural ways of transmitting the virus distinguish: vertical, horizontal and sexual.

"Vertical transmission" is divided into prenatal (transplacental) and intranatal. According to some authors, since 1990, the frequency of detection of HBsAg among pregnant women has increased 2.5 times. Infection of the child with the virus from the mother is essential because in newborns, the greatest risk of transformation of acute infection into a carrier, with the subsequent development of chronic disease. From such carriers the population of the infected persons which is a source of an infection is formed. According to the literature, prenatal infection occurs in 5-15% of children. In HbeAg-positive mothers, the probability of perinatal infection of the child is from 50 to 90%, and in anti-Hbe-positive mothers the risk of infection is reduced to 10-30%. In addition, viral hepatitis B infection in infants during breastfeeding (in the presence of cracked nipples and oral mucosa) cannot be ruled out, as the virus may be present in small amounts in breast milk.

One of the main routes of viral hepatitis B transmission is sexual. The biological basis for the implementation of this route of transmission of the virus there is contamination of vaginal secretions and testicular fluid, it is also important to damage the genital mucosa during sexual intercourse. Horizontal pathways (hemopercutaneous contacts) of viral hepatitis B transmission occur in the environment of an active source of infection (a patient with acute and chronic VH) both in everyday life (family outbreaks, in closed children's groups) and at work. The basis of this route of transmission is a meager infectious dose of the virus in VH. Serum with viral hepatitis B has been shown to be infectious at a dilution of 10-7-10-8. Among the artificial ways of implementing the parenteral mechanism of viral hepatitis B transmission, according to many scientists, the leading infection remains in medical institutions. The frequency of detection of serological markers of VH (HBsAg and anti-HBs) in patients of medical institutions is 26.8%. Infection is possible during medical manipulations with damage to the skin and mucous membranes, transfusion of blood and its components, transplantation of organs and tissues. Among other components of the group should pay attention to the infection of drug addicts by intravenous administration of drugs. Among injecting drug users, the frequency of detection of markers of viral hepatitis B infection is 77.2%. Also important in the epidemiological situation of VH may be religious and national factors in the regions (ritual manipulations) and others that affect the behavior of the population (hairdressing and cosmetic manipulations).

The antigenic structure of the hepatitis B virus is complex. There is a surface antigen - HBsAg, which corresponds to the originally discovered "Australian antigen", nuclear, which is closely related to DNA polymerase - HBsAg, infective antigen - HBeAg, which is a transformed HBsAg. The outer shell of the virus is located in the cytoplasm of the infected hepatocyte and contains proteins-antigens: surface HBsAg and antigens pre-S1, pre-S2. HBsAg is formed in excessive amounts, part of it is used to collect the virus, the other - goes into the intercellular space and circulates in the blood. HBsAg is heterogeneous, has 4 main subtypes - adw, adr, ayw, ayr, with a common determinant - group-specific and have epidemiological significance, as different subtypes are registered in different regions. Pre-S1 and pre-S2 antigens play a leading role in the mechanism of interaction of hepatitis B virus with hepatocytes. The outer shell also contains albumin-sensitive receptors, which determines the hepatotropicity of the virus to the corresponding zones of polyalbumin receptors of human hepatocytes.

The inner part of the hepatitis B virus (nucleocapsid) penetrates in the nucleus of the hepatocyte and contains the following components:

- HBcorAg - core or nuclear antigen, contained exclusively in the nuclei of hepatocytes in free form, and its particles have proteokinase activity, which is necessary for phosphorylation of proteins; not determined in the blood serologically;
- HBeAg - has two antigenic variants of HbeAg 1 and HBeAg 2, is part of HbcorAg, representing its secretory soluble part, closely associated with DNA polymerase. Free HbeAg is isolated in the blood serum, which reflects the degree of viral replication, so it is called the antigen of infectivity;
- in addition, the nucleocapsid contains the genome of viral hepatitis B virus and DNA polymerase.

The genome of hepatitis B virus consists of approximately 3200 nucleotides. It is a double-stranded DNA molecule in which one strand is 30% shorter than the other. The part of viral DNA that is not enough is completed during the replication of the virus from the nucleocapsids of the host using DNA polymerase contained in the nucleocapsid of the virus. The genome of the virus hepatitis B consists of 4 genes:

- Gene S encodes the main protein of the shell and contains all the information about HBsAg, preS1 and preS2.
- Gene C encodes the capsid protein HbcorAg and HbeAg; contains information about the protein that affects the DNA reproduction of the virus.

- Gene X - encodes the capsid protein HbxAg, which regulates the expression of all viral genes and the process of HBV replication. HbxAg plays a role in the development of hepatocellular carcinoma.

- Gene P - encodes enzymes required for replication of the virus (DNA polymerase) and is involved in the encoding of HbcorAg.

The virus is quite stable: when frozen to a temperature of - 20 ° C, it remains infectious for 15 years, resistant to thawing and re-freezing. Retains activity at 30 ° C for 6 months. Withstands boiling for 30-40 minutes, autoclaving for 30 minutes. In a 2% solution of formalin, its neutralization occurs after 7-9 hours. Ultraviolet radiation does not affect the virus. Hepatitis B virus is related to various tissues and, although more common in liver damage, viral DNA and proteins are also found in the kidneys, spleen, pancreas, skin, bone marrow, and peripheral blood mononuclear cells, and can cause arthritis. Peripheral mononuclear cells may be the first targets of viral hepatitis B infection. These mechanisms are the basis ineffectiveness of interferon drugs in patients with fulminant hepatitis B and liver transplantation. Viral hepatitis B replication in peripheral mononuclear cells can be registered with negative results of identification of virus markers in blood serum. All this makes it difficult to diagnose viral hepatitis B infection with extrahepatic manifestations.

The peculiarity of the infectious process in viral hepatitis B is the wide fluctuations of the replicative activity of the virus, which causes its different efficiency, which indicates the genetic heterogeneity and heterogeneity of viral hepatitis B. Along with the usual "wild" strains of the virus are mutant. At defeat by pre-S mutant strain chronicity of a disease is registered mainly. Mutations are possible in other viral hepatitis B genes. The least variable is soge-gen. In recent years, it has been found that long-term therapy with nucleoside analogues can lead to mutations in the viral hepatitis B genome. At the termination of treatment there is a return of "wild" strain of viral hepatitis B as dominant. Chronic viral hepatitis B infection is divided into two phases: active viral replication and integration. The replicative phase of the infection is characterized by the reproduction of viral hepatitis B in hepatocytes and is manifested in the laboratory by the detection along with the DNA of the virus and HBs-antigen in the blood also HBe-antigen and antibodies HBsAg-IgM. The integrative phase of infection is characterized by the detection of viral hepatitis B DNA sequences and HBs antigen in many tissues and blood. This is due to the fact that the development of viral hepatitis B infection simultaneously with the replication of the virus in the cell can also be the incorporation of viral hepatitis B DNA sequences into the DNA of cells of the macroorganism, in particular,

hepatocytes. At the end of the replicative phase of infection, the manifestation of markers of only integrative infection in the form of HBs antigen and viral hepatitis B DNA without clinical manifestations corresponds to a "healthy" carrier of viral hepatitis B infection. Hepatitis B virus antigens and antibodies homologous to them are recognized by the WHO as specific serological markers of this infection. Interpretation of antigens and antibodies to them is an indicator of the infectious process. Evidence for the active course of infection is the determination of HBsAg, anti-HBc class IgM and virus-specific DNA. The appearance of anti-HBs in combination with anti-HBs during convalescence is a sign of infection. HBeAg is a companion of full-fledged viral particles, is a direct indicator of active reproduction of the virus and reflects the degree of infection. Long-term HBe and HBs-antigenemia is an unfavorable sign that indicates the formation of a chronic process. Replacement of HBeAg with appropriate antibodies to prolong HBs antigenism indicates a benign course. HBsAg disappears from the blood during the first month after the onset of jaundice. Antibodies to it appear 3-4 months after the onset of the disease. In severe forms of viral hepatitis B anti-HBsAg are detected from the first days of jaundice. To distinguish persistence (carrier) from the active form of infection, it is necessary to study the serum of anti-HBs class IgM - their absence indicates a carrier, not an active process. A negative blood test for HBsAg does not rule out a diagnosis of HBV. In such cases, the determination of anti-HBc class IgM can serve as confirmation.

Table 9

MAIN PROPERTIES OF HEPATITIS VIRUSES A AND B

Signs		**Hepatitis A**	**Hepatitis B**
1		**2**	**3**
Properties virus	**Diameter, nm**	27	42
	Type of nucleic acids	RNA	DNA
	60^0C (1 hour)	stable	stable
	100^0C (destruction)	5 minutes	10 minutes
	Carrier	no	is present
Morbidity		epidemic	sporadic

1	2	3
The main route of transmission	enteral	parenteral
Auxiliary transmission path	parenteral	household, sexual,
Age of patients	children, teenagers	transplacental
Seasonality	is present	all age categories
Incubation period	14-45 days	is absent
Hypertransferaseemia	1-3 weeks	40-180 days
Predisposition to chronicity	rarely	4-30 weeks
Mortality	0.1%	often
Protective action of normal immunoglobulin	is present	1%

FEATURES OF PATHOGENESIS OF VIRAL HEPATITIS B

The entrance gate is blood vessels, damaged skin, mucous membranes. The virus enters the body and is fixed on hepatocytes and cells of the reticuloendothelial system. After replication (reproduction) of the virus is its entry into the blood. Hepatitis B is considered a disease of the immune system. When viral hepatitis B replicates, cytolysis of hepatocytes occurs, which is carried out by T-lymphocytes and K-cells. Hepatocyte death is associated with cytotoxic and humoral immune responses that eliminate the virus from the body. The destruction of infected hepatocytes is accompanied by a mass release of viral antigens, which leads to the accumulation of antibodies primarily anti-HBs and anti-HBe. There is the formation of immune complexes, phagocytosis by macrophages and excretion by the kidneys. The majority of patients are cleansed of the body from the virus and their recovery.

Clinic of viral hepatitis B

The incubation period lasts from 40 to 180 days. The initial (pre-jaundice period) lasts from 3 days to 3 weeks, on average 7-14 days. The disease usually begins gradually. The most common variants of the pre-jaundice period of viral hepatitis B are:

• dyspeptic (abdominal) (40-60%), characterized by decreased appetite, aversion to food, heaviness in the right hypochondrium, bloating, diarrhea or constipation;

• arthralgic (20-35%), manifested by pain in large joints, often at night and in the morning, without changing their configuration and skin color over the joints;

• allergic (10-12%) - the appearance of urticarial rash on the skin, which persists for 1-2 days and is accompanied by eosinophilia in the peripheral blood.

• asthenovegetative (general malaise, weakness, lethargy, sweating, headache);

• pseudo-influenza (increased body temperature, runny nose, sore throat, dry cough) options.

The jaundice period lasts 3-4 weeks and is characterized by the severity and stability of clinical manifestations. The transition from the initial period of the disease to jaundice is accompanied by subicteric mucous membranes, primarily of the soft palate, sublingual area and sclera, with a threshold concentration of bilirubin in the blood above 25 μmol / l. After 1-3 days, the skin gradually turns yellow, urine darkens, feces becomes acholic, which occurs with hyperbilirubinemia above 30-35 μmol / liter. Jaundice reaches its maximum in the 2-3rd week and later.

The clinical picture of this period of the disease is dominated by the following syndromes: pain, dyspepsia, asthenovegetative, approximately 20% of patients develop or worsen itchy skin, may have hemorrhagic syndrome. In all patients, the liver is moderately enlarged. The liver is smooth, slightly dense, moderately sensitive to palpation. Splenomegaly is possible, there may be positive gallbladder symptoms.

In the peripheral blood in the acute period is detected leukopenia with lymphocytic and monocytosis, sometimes with a plasma reaction. ESR is reduced to 2-4 mm / h, during convalescence can accelerate to 18-24 mm / h, followed by normalization in the absence of complications.

Hyperbilirubinemia - pronounced and persistent, often in the 2-3rd week of jaundice, the level of bilirubin in the blood is higher than in the first. As a rule, there is a sufficiently pronounced increase in the activity of aminotransferases in the serum with a decrease in the sulem test and prothrombin index. Normalization of aminotransferase activity, as a rule, in mild course occurs up to 30-35th day of the disease, in moderate - up to 40-50th, in severe form - 60-65th day.

With viral hepatitis B, everyone can be observed biochemical syndromes of liver damage: cytolytic, cholestatic, mesenchymal-inflammatory, in severe cases develops insufficiency of protein-synthetic function of the liver.

At viral hepatitis B with a cholestatic syndrome against intensive cytolysis (high activity of aminotransferases, dysproteinemia, positive thymol test, low figures of a prothrombin index) in the midst of the jaundice there are signs of cholestasis

(itchy skin, severe hyperbilirubinemia, increased activity alkaline phosphatase, increase in blood bile acids, phospholipids, B-lipoproteins, cholesterol.

The convalescence period lasts 3-6 months. and more. There is a slow disappearance of clinical and biochemical symptoms of the disease. Of the functional tests, the bilirubin content in the blood normalizes the fastest. The ALT activity index normalizes slowly.

In severe viral hepatitis B infection, signs of liver failure and progression of necrotic processes in the liver increase. The severity of acute viral hepatitis B can be determined by fulminant hepatitis, cholestatic syndrome, rarely - edema-ascites syndrome, aplastic anemia.

Criteria for the severity of viral hepatitis B

• short acute prodrome, possibly with fever;

• a feature of the beginning of the jaundice period is significant intoxication, disturbed sleep inversion, characteristic anorexia, nausea, repeated vomiting;

• intensity of jaundice and rapid growth rate;

• characteristic symptom - "scissors" between the severity of intoxication and jaundice and small liver size;

• the liver is soft, small, painful (due to necrosis);

• severe hyperthermia in the jaundice period for no apparent reason;

• tachycardia, hypotension;

• early developing hemorrhagic syndrome (hemorrhagic rash, bleeding, bleeding);

• itchy skin disappears (impaired synthesis of bile acids);

• laboratory criteria: decrease in prothrombin, decrease in total protein and albumin, decrease in cholesterol and β-lipoproteins, change in the ratio of unconjugated / conjugated bilirubin (1: 1 or more), in peripheral blood neutrophilia, aplastic anemia.

With prolonged forms of viral hepatitis B clinical and biochemical manifestations of the disease and especially the period of reverse development lasts from 3 to 6 months. These forms can be a prelude to chronic hepatitis. Chronic viral hepatitis B infection in most cases has a clinically smoothed asymptomatic course. The diagnosis is often initially established on the basis of laboratory results (increased activity of blood enzymes and the presence of markers) and liver biopsy. And so the so-called chronic "virus" is a purely conditional concept and corresponds to viral hepatitis C, which is latent.

Clinical manifestations of **chronic hepatitis B** in many cases depend on the replicative activity of the virus. HBV replication is evidenced by the presence of HBeAg, or in its absence (low replication activity, mutant strains - Hbe-negative HCV) - the detection of HBV DNA in the blood. Of particular importance is the high level of HbsAg concentration (more than 100 ng / ml), the presence of anti-HBc IgM. In the absence of markers of replication and detection of HbsAg, anti-HBc IgG and anti-HBe indicate an integrative phase.

Chronic integrative hepatitis B, as a rule, has a benign course. Its course is asymptomatic with normal blood biochemical parameters and is diagnosed based on the identification of specific viral markers corresponding to this phase.

Chronic replicative hepatitis B in most patients has a jaundiced course. The first signs correspond to the patient's complaints of fatigue, weakness, headaches, decreased tolerance to exercise, fatigue in the morning, sleep is disturbed, emotional instability - hepatic intoxication gradually develops. Dyspeptic disorders (loss of appetite, intolerance to fatty foods, nausea, epigastric pain, heaviness and pain in the right hypochondrium), sometimes combined with intermittent subfibrillation and dark urine. Hepatomegaly is the most persistent, often the only objective sign of pathological changes in the liver. The liver in chronic hepatitis C is denser than in acute hepatitis, splenomegaly is less common. Signs of hypersplenism in chronic hepatitis C are rare, mainly in the course of the disease type cirrhosis. Anemia and thrombocytopenia caused by a hemorrhagic syndrome can be registered. Stable jaundice is uncommon, mainly in patients with cholestatic hGV, in combination with itchy skin. Often there extrahepatic signs (teleanhektaziyi - vascular "stars" palmarna erythema) and extrahepatic manifestations (aplastic anemia, papular akrodermit, Sjogren's syndrome, cutaneous vasculitis, nodular periarteriitis, Polyarthralgias, myalgia, myocarditis, glomerulonephritis, cryoglobulinemia, etc.).

Of the non-specific biochemical parameters that reflect the violation of the functional state of the liver, ALT will moderately increase, the prothrombin index decreases, dysproteinemia, a slight increase in ESR. The severity of the cytolytic syndrome corresponds to the activity of viral replication.

An important criterion for assessing the course of chronic hepatitis is the characteristics and frequency of exacerbations that may be due to intercurrent diseases, errors in diet. The most common sign of exacerbation is an increase in ALT ("biochemical exacerbation" in the absence of clinical signs).

Consequences of hepatitis B:

• recovery;

• residual manifestations: asthenovegetative syndrome, posthepatitis hepatomegaly (hepatosplenomegaly), posthepatitis hyperbilirubinemia (manifestation of Gilbert's syndrome), prolonged convalescence;

• prolonged hepatitis (lasting from 3 to 6 months);

• persistent chronic hepatitis;

• dyskinesia and lesions of the biliary tract;

• cirrhosis;

• primary liver cancer;

• chronic viral load.

Specific prevention

Active immunization is recombinant hepatitis B vaccine 3 times: 0, 2, 6 months, revaccination is carried out every 5 years.

Passive immunization is performed by using immunoglobulin with a high titer of antibodies to HBsAg (1: 100000 - 1: 200000). Passive immunization is recommended to carry out:

• in the first hours after infection and again in 1 month;

• if the threat of infection persists for a long time, it is desirable to carry out in 1-3 months.

All convalescents are subject to dispensary supervision mandatory clinical and laboratory examination within 12 months. Biochemical and serological testing (HBsAg) is carried out in 10 days after discharge from the hospital, then in 3, 6, 12 months.

VIRAL HEPATITIS C

Acute viral liver disease caused by an RNA-containing virus from the flavivirus family, which has a gradual onset, with a variety of clinical forms - from non-jaundiced to malignant and frequent transition to chronic hepatitis.

Features of the epidemiological process

The source of infection is a patient with acute or chronic hepatitis C. The routes of transmission are parenteral, possible perinatal infection of the newborn from the mother with HCV infection.

The main links in the pathogenesis of viral hepatitis C

1. Getting the virus through the blood to liver cells and blood mononuclear cells.

2. Reproduction and persistence of the virus in tropical organs.

3. Damage to hepatocytes with viral and immune cytolysis. Viral hepatitis C antigens have low immunogenicity, immunity is considered suboptimal, reinfection is possible.

Clinic of viral hepatitis C

The incubation period is from 2 to 52 weeks. The pre-jaundice period lasts from 5 to 14 days. The initial signs are moderate, there are signs of intoxication, heaviness in the right hypochondrium, the liver is enlarged, painful on palpation and soft. The jaundice period can last from 2 weeks to 1.5 months. At this time, mild or moderate intoxication persists, an increase in the size of the liver and spleen in 1/3 of patients, ALT increases by 5-10 times, a slight increase in AST and thymol test. Detection of HCV RNA is a reliable and significant feature. Antibodies to HCV appear at 6-8 weeks from the onset of the disease: anti-HCV-sor-IgM, HCV-sor-IgG, the latter are detected up to 6-7 years. Chronic disease is noted in 70-90% of patients.

VIRAL HEPATITIS D

Hepatitis delta is a highly active inflammatory-dystrophic process in the liver, which is characterized by moderate and severe course, often turning into a fulminant form, chronic hepatitis and cirrhosis of the liver.

Etiology and epidemiology

Viral hepatitis delta is caused by an RNA-containing virus, the outer shell of which is the surface antigen of viral hepatitis B HBsAg. It is a defective virus and its properties depend on the helper virus. It is more common in patients with chronic hepatitis B.

The source of infection is patients and carriers of delta infection. The routes of transmission are parenteral and transplacental, as well as through damaged skin.

Features of pathogenesis and clinic

The delta virus has a direct cytotoxic effect on hepatocytes. There are two variants of the disease: coinfection and superinfection. *Co-infection* is said to occur when B and D viruses are co-infected. The disease progresses in two waves: the first is hepatitis B virus replication and expression, and the second is D virus replication. Serological markers of hepatitis B and D are found in the blood serum during coinfection.

Superinfection is said to occur in patients with chronic hepatitis B or carriers of HBs Ag are infected with a delta-positive pathogen. The combined effect of hepatitis B and hepatitis D extends to the liver parenchyma, causing the formation of chronic hepatitis and liver cirrhosis. Sometimes there is a chronicity of the process, when the body has immunosuppression and T-cell deficiency.

Table 10

EPIDEMIOLOGICAL SIGNS OF VIRAL HEPATITIS

Virus	Source	Ways of transmission	Periods			
			Incubative	Premonitory	Icteric	Reparative
1	2	3	4	5	6	7
Viral hepatitis A	patient, carrier	fecal-oral, contact household	10-45d.	3-5d.	2-4w.	2-3m.
Viral hepatitis B	sick, healthy, carrier	parenteral, transplacental	6-26w.	5-7d.	3-4w.	6-10m.
Viral hepatitis C	patient, carrier	parenteral, transplacental	6-8w.	7-8d.	3-6w.	6-12m.
Viral hepatitis D	patients with viral hepatitis B, HBs carrier	parenteral, transplacental	2w.-6m.	5-7d.	2-8w.	6-12m.
Viral hepatitis E	patient, carrier	water	18-130d., 3-28w.	1-11d.	2-3w.	6-12m.

1	2	3	4	5	6	7
Viral hepatitis G	patient, carrier	parenteral	7-11d.	5-7d.	3w.	6-12m.
Viral hepatitis TT	patient, carrier	parenteral				6-12m.
Viral hepatitis F	patient, carrier	parenteral				6-12m.
Viral hepatitis SEN	patient, carrier	parenteral				6-12m.

Table 11

Clinical differential diagnostic signs of viral hepatitis

Signs	A	B	C	D	E
1	**2**	**3**	**4**	**5**	**6**
Age	older than 1 year	all periods	all periods	all periods	older than 1 year
Incubation period	14-45 days	2-6 months	2 weeks -3 months.	2 weeks - 6 months.	15-45 days
Beginning	acute	gradual	gradual	acute	acute
Intoxication in the pre-jaundice period	expressed	weakly expressed	weakly expressed	expressed	expressed
Intoxication in the jaundice period	weakly expressed	expressed	absent or weakly expressed	expressed	absent or weakly expressed
Allergic rash	is absent	may be	may be	may be	is absent
The severity of the disease	mild or moderate	moderate or severe	light and jaundiced	severe, malignant	easy
Jaundice period	1 - 1.5 weeks	3 - 5 weeks	about 1 week	2 - 8 weeks	1 - 2 weeks
Chronization	is absent	often	up to 50%	often	no

1	2	3	4	5	6
Serological markers	anti-HAV IgM	HBs Ag, HBc Ag, anti-HBc Ig M	anti HCV, HCV RNA	HBs Ag, anti Bc, anti HDV IgM	anti HBV

Complications of viral hepatitis: acute liver failure - treatment according to the appropriate treatment protocol.

LIVER FAILURE

There are acute and chronic liver failure, which are divided into three stages: initial (compensated), exacerbation of clinical manifestations (decompensated), terminal (dystrophic). The terminal stage of liver failure ends in hepatic coma. **Hepatic coma** - a state of abrupt inhibition of higher nervous activity, which is manifested by profound loss of consciousness, impaired mobility, sensitivity, reflexes, lack of reaction of the child to sound, light and other stimuli and is accompanied by widespread hepatocyte necrosis, which leads to a significant decrease in liver function with the development of general toxicosis of the body.

Clinical picture. At the initial stage there is weakness, fatigue, loss of appetite, dyspepsia, sleep disorders, headaches, digestive disorders, fever, anemia, thrombocytopenia, hemorrhagic syndrome. In the midst of the disease, signs of portal hypertension appear - ascites and edema. When hepatocytes are damaged, the liver produces plasma albumin, inactivates aldosterone, which leads to secondary hyperaldosteronism, increased reabsorption of sodium ions into the extracellular space. Of additional importance is the production of antidiuretic hormone, which causes the development of edema.

In chronic liver failure, endocrine changes gradually occur. Boys - develop testicular atrophy, gynecomastia, hair loss under the armpits, on the head, and in girls - atrophy of the uterus, mammary glands, menstrual irregularities. All these changes are due to the accumulation of estrogen due to insufficient inactivation by the liver. Disorders of inactivation of estrogens and some vasoactive substances to small telangiectasias - vascular "stars", palmar erythema, dilation of the vascular network on the face. Anorexia, significant weight loss and polyvitaminosis gradually develop. Often liver failure is also accompanied by a violation of filtration ability of the

kidneys and the development of azotemia. In the pre-comatose state, anorexia, nausea, decreased liver size, increased jaundice, hyperbilirubinemia, amino acids and lactic acid in the blood are observed. Later, neuropsychiatric disorders, depression, and sometimes euphoria, irritability, impaired memory, sleep, increased tendon reflexes, meningeal signs appear, breathing is disturbed, the pulse becomes arrhythmic, body temperature decreases, the liver smell is noticeable from the mouth. , aggravated hemorrhagic phenomena. Increased ESR, residual nitrogen and ammonia, there is hypokalemia, hyponatremia, metabolic acidosis. Hepatic coma often ends in death for the child.

Emergency care for hepatic coma

• Normalization of metabolism in hepatocytes - hyperbaric oxygenation (HBO), preventive ventilation;

• Increase in glycogen content in hepatocytes - 10-20% glucose solution 10 ml / kg with insulin;

• Stabilizers of cell membranes - prednisolone 3-5 mg / kg, essential 3-5 ml;

• Antioxidants - 5% solution of ascorbic acid, vitamin E 5 mg / kg / day;

• To relieve swelling of liver cells - Lasix 0.2-0.4 mg / kg;

• Antihypoxants - phenobarbital 1-5 mg / kg, droperidol 0.1-0.2 mg / kg;

• Drugs that protect membrane structures from peroxidation - cytochrome C, lipoic acid 70 mg / kg, 2% soda solution, cocarboxylase 5 mg / kg / day;

• Artificial detoxification - peritoneal dialysis, enterosorption, plasmapheresis, hemodialysis, forced diuresis;

• Correction of plasma protein composition and water-electrolyte metabolism;

• In hemorrhagic syndrome hemostatic drugs heparin 30 U / kg / day under the control of coagulogram, fresh-frozen plasma 10 ml / kg;

• To inhibit ammonia production, its utilization and rapid excretion from the body, the drug dufalak in a dose of 5-20 ml 3 times a day is recommended;

• Neuroleptics, antihistamines are indicated for mental disorders.

TREATMENT OF ACUTE VIRAL HEPATITIS

All patients with viral hepatitis should be treated in infectious hospitals. In acute viral hepatitis of mild and moderate severity, semi-bed rest is shown, and in severe viral hepatitis is bed rest.

All patients were shown dietary nutrition: in mild degree - table №5, in moderate and severe degree - table №5a.

Diet №5 contains 90-100 g of protein, 80-100 g of fat, 350-400 g of carbohydrates, caloric content of 2800-3000 kcal. Warm and stew dishes are allowed. It is recommended to eat 4-5 times a day, because a small portion of food helps to normalize bile secretion and eliminate stagnation in the biliary tract. To enhance the detoxification function of the body, the daily volume of fluid consumed is increased to 1.5-2 liters.

In the diet 5a dishes are ground, the amount of fat is reduced to 50-70g, table salt - to 10-15g. The patient is transferred to the diet after a significant improvement in general condition and reduction of jaundice. During the recovery period, a diet is indicated until complete recovery, at least 3-6 months after discharge from the hospital.

Most proteins are administered with dairy products, the rest - in the form of meat (krill, veal, fish). Of the fats, only vegetable and butter are shown. Carbohydrates are abundant in white bread, sugar, oatmeal, buckwheat and semolina, potatoes, honey, fruits, jams, compotes. Very useful products with a high content of vitamins A, C, K, group B, the need for which increases during reparative processes in the liver. It is necessary to prevent overeating, eating large amounts of food, which is a common cause of exacerbation of viral hepatitis and relapse. Be sure to exclude extractives (broths, fish soup), fried and fatty foods, canned food, waterfowl, lamb, pork, marinades, spices, chocolate, alcohol.

In the treatment of viral hepatitis use protocols recommended by the Ministry of Health of Ukraine.

Mild form: basic therapy 20 days, semi-bed regime - 10 days. Diet therapy - table №5.

Moderate-severe form: basic therapy for 20 days, bed - to restore the color of urine, then half-bed - 2-3 weeks, diet therapy - table №5. Oral detoxification therapy in the amount of 40-50 ml / kg (5% glucose solution, table still mineral water) with mandatory control of water balance. Enterosorbents - 2 weeks. The use of

multivitamins is recommended. During convalescence, it is advisable to use cholagogues.

Severe form: basic therapy for 30 days, mode - bed, to restore the color of urine, then - half-bed - 3 weeks; diet therapy - table №5a 3-7 days (before stool staining), then №5. Detoxification therapy - intravenous drip solutions at the rate of 50-100 ml / kg, albumin - 5 ml / kg, rheopolyglucin 5-10 ml / kg, 5% glucose solution, Ringer's solution, 0.9% sodium chloride solution; enterosorbents - 2-3 weeks; in the presence of cholestasis - a course of deoxycholic acid 10 mg / kg; enzyme therapy - 14 days; at threat of development of a fulminant form and children of 1 year of life with an unfavorable premorbid background - prednisolone 1-3 mg / kg 4 times a day for 7-10 days. VF Uchaikin in severe forms of acute viral hepatitis recommends the following regimen of hormone therapy with prednisolone:

- 2-3 mg / kg in 4 doses for 3 days;
- 1-1.5 mg / kg in 4 doses of 3 days;
- 0.5 mg / kg in 4 doses of 3 days, followed by cancellation.

In severe cases, also use a polarizing mixture - 3.7 g of potassium chloride, 6-8 U of insulin, 5% glucose solution 1 liter.

Fulminant form: strict bed rest; diet №5a with a restriction of proteins up to 40% per day; prednisolone 10-15 mg / kg a day in 4 hours in equal doses without a night break in / in is appointed; detoxification therapy: albumin, reopolyglucin, 10% glucose solution at the rate of 50-100 ml / kg per day under the control of diuresis; proteolysis inhibitors; furosemide 1-2 mg / kg and mannitol 1.5 g / kg; heparin 100-300 U / kg at risk of DIC syndrome; antibacterial therapy; lactulose preparations - dufalak in a dose of 5-20 ml 3 times a day for 14 days; ursodeoxycholic acid 10 mg / kg body weight 1 time per day; extracorporeal methods of detoxification in case of ineffective therapy - plasmapheresis in the amount of 2-3 BCC 1-2 times a day before with a coma; hyperbaric oxygenation - up to 10 sessions; with edema-ascites syndrome - correction of water-electrolyte balance and protein composition of the blood: the introduction of 10-20% solution of albumin, plasma, potassium-sparing diuretics (verospirone, triampur, spirolactones).

Interferon therapy for acute viral hepatitis

Interferon therapy for acute viral hepatitis occupies a significant place among antiviral drugs interferon. The main purpose of interferon therapy in acute viral hepatitis is to prevent its transformation into chronic. It is important to select patients

for treatment with interferon. This drug is indicated for children at high risk of chronicity of the process, in which blood continues to circulate HbeAg, HBV-DNA and hematological patients, children with viral hepatitis C, in whom HCV-RNA is detected in the blood, high viral load, increased ALT activity in 5 times and more. Today there are 3 schemes of interferon therapy in viral hepatitis C:

1. 10000000 IU every day until the normalization of ALT activity, then 3 million IU 3 times a week for 6 months .;

2. 5 million IU 3 times a week for 2 months, then 3,000,000 IU 3 times a week - 4-10 months;

3. 3 000000 IU 3 times a week for 3-6 months.

Treatment of chronic viral hepatitis

Interferon monotherapy (IFN) remains the standard of treatment for chronic viral hepatitis. **Interferons (IFNs)** are low molecular weight polypeptides (18000-25000 daltons) -

genetically determined components of the universal system of protection of an organism from foreign genetic information or products of its realization. These products enter the human or animal body in the form of microorganisms or many natural or synthetic substances (polyphosphates, polycarbonates, etc.). They are found in people with immune and interferon-induced defense systems. The interferon defense system, according to popular belief, is a system of rapid response to infection.

The mechanism of antiviral action of IFN is to inhibit the synthesis of virus-specific proteins at the level of blocking the translation of viral messenger RNA. IFNs have multiple biological effects that go beyond their antiviral action. These effects are conditional divided into 3 groups: antimicrobial, immunomodulatory and antiproliferative. In the latter case, they control the rate of mitosis: they do not change the rate of normal cell division, but suppress the high rate of division, for example in malignant neoplasms.

IFNs are divided into two categories. The first category is natural IFN, human leukocyte IFN. It is synthesized by leukocytes. Its synonyms: IFN-a, etiferon, velferon. Next - human fibroblast IFN, also known as: IFN-r, ferron; synthesized by human fibroblasts. The third type of natural IFN -human immune IFN, synthesized by T-lymphocytes; known as y-IFN.

The second main category of IFN drugs is recombinant IFN, 2nd generation IFN, mainly different subtypes of IFN. These are IFN-a-2a (reaferon, roferon),

IFN-a-2b (intron, incre), IFN-a-2c (berofor). Also obtained recombinant IFN-p (betaferon) and recombinant IFN-y (gammaferon).

Numerous results of positive use of IFN against acute and chronic viral hepatitis, a number of ARVI: influenza, flu-like infections have been obtained.

IFN inducers also belong to the group of antiviral drugs. It is a diverse and numerous set of natural and synthetic factors. They are considered a new promising group of antivirus drugs. They vary in chemical composition, classes of compounds, and source of production or isolation. These drugs are characterized by one common property - the ability to induce interferon synthesis.

So far, about ten promising IFN inducers suitable for clinical use have been identified. The most well-known of them, which belong to the class of synthetic compounds of monomers (fluarenes and / or nitrous bases), are amixin and comedone, and of the synthetic compounds of polymers (poly-A, poly-B, li-G, poly-C) - poludan and polyguacyl. From the class of natural low molecular weight inducers of IFN (for example, from polyphenols are distinguished megasin, kagocel, savrats, ragosin, gasalidone. High molecular weight inducers of this class (polymers in the form of double-stranded RNA) are larifan and ridostin.

Another class, called the official inducers of IFN, includes: methylxanthines (theophylline, theobromine, euphylline, dipyridamole, caffeine), isoquinoline derivatives (papaverine, no-spa). In the same group includes imidazole derivatives (dibazole). Drugs of the group of "official" IFN inducers are derivatives of benzofuran (cordarone) and derivatives of chromium compounds (intercordin). IFN inducers have one unique property - the ability to exhibit interferon induction of a relatively wide range of viruses.

Currently, IFN inducers, as antiviral drugs, are mostly undergoing a stage of laboratory and preclinical studies, although there is an encouraging result of the use of these drugs in influenza and rhinovirus infections. It is assumed that in the near future future areas of clinical use of interferon inducers may be expanded.

IFN contributes to biochemical improvement, reduction of viremia, but a stable response is observed only in 25-30% of patients. Currently, one of the most pressing issues of chronic hepatitis C (CHC) is the problem of rational etiotropic treatment of such patients.

The main antiviral drug for the treatment of HG is interferon-alpha (IFN-a), in particular its recombinant forms - a2a and a2b. They are created by genetic engineering using the human interferon-a2 gene and Escherichia coli as a producer.

For therapeutic effect it is necessary to achieve, and most importantly, to maintain the required dose of the drug in the patient's body. In this regard, in recent years, pegylated (prolonged) forms of IFN have been obtained. Due to the addition of a large inert molecule of polyethylene glycol to the IFN, the molecular weight of the drug increased significantly. This allows you to release its clearance from the body and maintain the concentration of the drug in the blood for a week after injection.

Indications for the appointment of □-IF in chronic viral hepatitis

1. Signs of active replication of the virus.

2. High biochemical activity (increase in ALT activity by 1.5 times or more) for 6 months.

3. Histological picture of liver biopsies with characteristic signs of inflammatory process activity.

4. No contraindications.

In addition to IFN, a synthetic analogue of the nucleoside (guanosine) ribavirin is used for the etiotropic therapy of CHC. This drug is not used as monotherapy, but only in combination with IFN-a. The goal of antiviral therapy is to achieve it inhibition of virus replication and remission of chronic viral hepatitis. The main criterion for prescribing etiotropic therapy to patients is active viral replication (presence of RNA) against the background of an active process in the liver.

In some patients on the background of IFN therapy, side effects are observed, which in 10-15% of cases require discontinuation of the drug. The most common of these is the flu-like syndrome. It is characterized by fever, malaise, headache, loss of appetite, aches in muscles and joints, sweating. This symptom complex usually develops at the beginning of therapy. As its duration increases, the brightness of these phenomena decreases. These side effects are eliminated by concomitant administration of paracetamol or other nonsteroidal anti-inflammatory drugs.

The main side effect of ribavirin is the occurrence of transient hemolytic anemia, which does not require specific treatment and disappears with a temporary reduction in the dose of the drug.

For the treatment of patients with chronic viral hepatitis C is used or monotherapy of IFN-a parenterally for 3 million. MO 3 times a week (pegylated IFN-a is administered 1 time per week, exceeds the usual efficiency) for 12 months. or a combination of IFN-a in the same doses with ribavirin orally at 800-1200 mg daily for 6-12 months.

Determination of HCV RNA is currently the most accurate method of predicting both a stable response to treatment and its ineffectiveness. Quantitative analysis of HCV RNA by PCR allows you to quickly identify patients who have not responded to 3-month therapy.

Given all the above, it should be emphasized that today almost all patients with HCG (in the presence of HCV RNA in the blood and no contraindications) are potential candidates for the combined use of pegylated IFN - PIFN-a2a - 180 mcg or PIFN-a2b -1.5 μg / kg parenterally once a week) and ribavirin, which meets the "gold standard" of treatment. The duration of therapy and the dose of ribavirin depend on prognostic factors. The genotype of the virus is a key factor influencing the antiviral effect of treatment. In this regard, with the 2nd / 3rd genotype, the recommended duration of therapy is 6 months, and the dose of ribavirin is 800 mg. Accordingly, at the 1st genotype duration treatment is increased to 1 year, and the dose of ribavirin to 1000 mg / d for body weight <75 kg and 1200 mg / d for body weight> 75 kg.

With regard to the use of IFN in mono mode to achieve antiviral effect, the drug can be prescribed for 1 year only in patients with increased ALT activity in the presence of "favorable" factors in response to treatment (2/3 genotype, low viral load, young age, short duration infection, stage of fibrosis F0-F2). In this case, if after 3 months. from the beginning of IFN-therapy in the blood is determined by HCV RNA, it is necessary to switch to a combination with ribavirin.

In the absence of severe fibrosis (F0-F2), patients should be monitored for possible re-liver biopsy after 3-5 years to determine the rate of fibrosis progression.

Doses of IF

• For chronic viral hepatitis B caused by a wild strain of the virus (HBV DNA + Hbe Ag +) - 5,000,000 IU / m2 3 times a week for 6 months.

• For chronic viral hepatitis C caused by a mutant strain of the virus (HBV DNA + Hbe Ag-, anti-Hbe +), - 5,000,000 IU / m2 once daily for 6 months to children older than 14 years.

• For chronic viral hepatitis B - 3,000,000 IU / m2 3 times a week for 12 months for all children under 7 years and patients with non-1B genotype of virus C.

• Children from 8-12 years with 1v-genotype of the virus - 6,000,000 IU / m2 1 time a day for 1 month, then 3,000,000 IU / m2 3 times a week for up to 12 months.

• Children older than 12 years with 1v genotype of the virus - 6 000000 IU / m2 1 time per day for 1 month, then 6 000000 IU / m2 every other day for 5 months, then 3 000000 IU / m2 3 times per day week for another 6 months.

Monotherapy of chronic hepatitis B and C with **interferon** against the background of traditional hepatoprotectors (thiotriazoline or carsil) is not effective enough. Due to the insufficient effectiveness of IFN monotherapy, the search for new approaches to treatment continues.

Interferonogens are high- and low-molecular compounds of natural and synthetic origin.

However, in a significant place in the therapeutic arsenal continue to occupy inducers of endogenous interferon formation, which have both certain advantages and disadvantages compared to IFN drugs. The main disadvantages of inducers are: insufficiently intense stimulation of their own synthesis of interferon in some patients; direct antitumor and antifibrotic effects have not been proven; the need for individual selection of the drug in each patient; the need for normal operation of the interferon system (sensitivity of the system to stimulation by inducers); the presence of a refractory period due to the "internalization" of the inducer-receptor complex in the cell nucleus after stimulation. During this period, the producer cell is unable to "respond" to re-stimulation by the inducer.

Among the advantages of inductors: high bioavailability; better tolerability than interferon drugs; much fewer side effects; lack of sensitization of the body by a foreign protein; lack of tachyphylaxis (no antibodies are synthesized, as to IFN drugs); long-term effect (therapeutic concentration of stimulated interferon can be maintained for up to 5-10 days after induction); relative cheapness; a significant variety of inductors, due to which it is possible to individually select these tools in each case; availability not only etiotropic (antiviral) action, but also pathogenetic (immunostimulatory). The latter is not only softer and more controlled than IFN drugs, but also more predictable due to the possibility of individual drug selection.

To date, several hundred drugs are known that can stimulate the production of endogenous interferon. Among the main classes of inducers of the greatest clinical importance are: polynucleotides; low molecular weight (up to 300-400 D) synthetic inducers (eg, acridones); nonsteroidal anti-inflammatory drugs; antiplatelet agents and peripheral vasodilators; some vitamins in large doses.

Standard circuits for inductors are not universal. First, it is impossible to accurately predict the effectiveness of the drug in a particular patient. Methods for determining the inducing activity in vitro give an approximate representation only

concerning leukocyte fractions of interferon. Secondly, and no less important, it is impossible to determine the duration of the refractory period of the inductor in each case.

The effectiveness of such interferonogens as cycloferon, amizon, amixin, isoprinosine has been established in the complex therapy of viral hepatitis.

Table 12

Modern antiviral drugs used most often in pediatric practice by infectious disease specialists

№	Name of the drug	Daily dosage, mg / kg
1.	Amizon	125
2.	Tamiflu	10
3.	Botanafton	25
4.	Arbidol-lens	100-200
5.	Metisazon	20
6.	Widex	10
7.	Virazole	10
8.	Acyclovir	10
9.	Ganciclovir	5-15
10.	Foscarnet	60
11.	Ribavirin	10
12.	Amiksin	12-25
13.	Proteflazid	15-30 drops
14.	Lamivudine	3

Dicloferon is a low molecular weight synthetic inducer of endogenous interferon. The main biological effects are: antiviral and antibacterial effects; immunomodulatory effects (regulation of antibody production of NK cells and toxic T lymphocytes). Its use does not cause side effects, is combined with other therapies.

The effectiveness of cycloferon in the treatment of acute and chronic hepatitis has been shown. The appointment of cycloferon helped to accelerate the

normalization of pigment metabolism, cytolytic syndrome, as well as reducing the immune imbalance. Analysis of long - term results of cycloferonotherapy (after 12 months after treatment) showed that the drug has high therapeutic efficacy.

Amizon is a new Ukrainian drug that belongs to the group of non-narcotic analgesics, has analgesic, anti-inflammatory, antiviral and interferonogenic effects. When studying the effectiveness of amizon and reaferon in patients with chronic hepatitis B and C, it was found that the drugs equally contributed to the positive clinical dynamics, immune parameters, gender. **Reaferon** reduced viral load (RNA titer) to a greater extent, but AMIZON was more easily tolerated by patients and had fewer side effects.

Amiksin affects mainly T cells, styling in them the synthesis of late interferon with maximum production after 12-18 hours. The drug has a wide range of antiviral activity against DNA and RNA of viruses. Amiksin causes the synthesis of interferons of I and II types in T-lymphocytes, enterocytes, hepatocytes. A more important feature of amixin is the ability to maintain therapeutic concentrations of serum interferon in the blood for a long time. The immunomodulatory effect of amixin is to stimulate bone marrow cells and T-cell immunity, normalize the immunoregulatory index, increase the activity of NK cells and cytotoxicity of lymphocytes, activation of phagocytosis. Good tolerability of amixin has been established.

Combinations of IFN and Interferonogens with drugs of other mechanism of action (combination therapy) are offered.

Treat by prescribing combination therapy with lamivudine and α-interferon. Lamivudine is prescribed once a day at a dose of 3 mg / kg for 6 months when combined with α-interferon, in isolation - for 12-18 months. α-interferon is used in a dose of HCV 5000000 U / m2; with HVGS - 3,000,000 IU / m2 3 times a week for 6-12 months.

In cholestatic liver diseases of various etiologies and pathogenesis, it is of interest to use **ursodeoxycholic acid (UDCA)** - ursofalk at a rate of 10 mg / kg. There are data on the positive therapeutic effect of UDCA in cirrhosis of the liver and chronic hepatitis, occurring both with overt cholestasis and without it. **Ursofalk** contributes to the reduction asthenic and dyspeptic syndromes, normalization of functional activity of hepatocyte biomembranes, reduction levels of alanine aminotransferase (ALT), aspartate aminotransferase and glutamyltranspeptidase. In patients with primary biliary cirrhosis, ursofalk enhances the effect of interleukin-2

and y-IFN, changing the ratio of T-lymphocytes / helpers (Tx) type 1 and 2 in favor of Tx1. Ursofalk, included in the basic therapy of CHC, also helps to improve the well-being of patients, normalize liver pigments, but does not affect viremia, compared with laferon monotherapy in patients.

Combination therapy with **laferon and ursofalk** in patients with chronic hepatitis is more effective: virological and complete remission is more often registered, the frequency of occurrence of side effects decreases and tolerability of therapy improves. A comparative analysis of the effectiveness of treatment with laferon and its combination with ursofalk showed greater activation of the immune cell in patients with CHC who received a course of combination therapy, proving the immunomodulatory properties of ursofalk, which positively affects the antiviral activity of IF.

Infectious disease scientists have also proposed a combined scheme of IFN with **proteflazide**. The newest Ukrainian natural preparation proteflazid, which is a plant extract and contains flavonoid glycosides isolated from cereals of pike and ground soldier. The drug has antiviral action by blocking thymidine kinase and DNA (RNA) polymerase, which stops the replication of viruses. In addition, proteflazid belongs to the immunocorrectors with interferonogenic effect and antioxidant properties, which gives reason to consider it as a hepatoprotector, including against hepatocytes. and finally, the phenomenon of apoptosomodulation. Effectively suppresses the activity of viruses, causes an increase in □- and □-interferons. Dosing - the drug is applied to pieces of sugar according to the following scheme from 12 years: 1 week - 5 drops 3 times a day, 2-3 weeks 10 drops 3 times a day, 4 weeks - 8 drops 3 times a day.

A complication such as cryopathy should be kept in mind. Cryopathies play an extremely important role in the pathogenesis of many diseases, in particular - chronic hepatitis C (CHC), in which the frequency of their detection reaches 80%. Today it is believed that the appearance in the blood of thermolabile proteins - cryoglobulins (CG) - is responsible for the formation of extrahepatic manifestations of HCV infection. In addition, the "masking" of the virus in the cryoprecipitate is not only difficult diagnosis of hepatitis, but also significantly reduces the effectiveness of antiviral treatment.

The use of the antiplatelet agent **dipyridamole** in a daily dose of 1-2 ml of 0.5% solution (children older than 12 years) intravenously or intravenously, reduces

the clinical and laboratory manifestations of cryoglobulinemia, regardless of its type. Prescribing ursodeoxycholic acid (10 mg / kg) has little effect on clinical and laboratory manifestations of cryopathy.

In the general scheme of treatment use hepatoprotectors (**berlithion, heptral, hepa-merz, essentiale forte H, hepabene**), membrane stabilizers, cholagogues, enterosorbents, probiotics.

The use of **berlithione** (□-lipoic or thioctic acid) provides regression of symptoms and restoration of the functional state of the liver by suppressing the inflammatory-necrotic reaction and a pronounced antioxidant effect. Dosage 2 times a day for 300 mg / m, a course of 4-6 weeks.

Heptral (ademethionine 1,4-butanedisulfonate) is a biological substance found in all tissues and fluids of the body. Its molecule is included in most biological reactions as a methyl group donor and as a precursor of physiological thiol compounds. Indications for use are intrahepatic cholestasis, precirotic and cirrhotic conditions. Dosage for intensive care - 400 mg per day intravenously or intravenously for 2-3 weeks. Maintenance therapy - 2-4 tablets a day.

Hepabene is an effective hepatoprotector of plant origin, which contains a complex of compounds: silymarin, silibin, silidianin, silicristin. This drug relieves pain, protects and restores liver cells, improves bile flow, has antioxidant properties, reduces the activity of transaminases, slows down the rate of entry of toxic metabolic products, restores the drainage function of the biliary tract, reduces the degree of fatty degeneration of the liver, has anti-allergic, diuretic, antihypertensive effect. Dosage: 1-2 capsules 3 times after meals.

Hepa-merz (ornithine) is an amino acid that reduces the concentration of ammonia in the blood plasma when hepatocytes are damaged and the development of liver failure, normalizes the acid-base balance. Appointed in a daily dose of 2-6g IV or 2-4g IV drip with 5% glucose solution

It is recommended in the treatment of chronic viral hepatitis to use **Dufalak** in a dose of 5-20 ml 3 times a day for 14 days. This drug reduces the toxic load on the liver, promoting the excretion of toxic products of hepatocyte autolysis and facilitates the course of the disease.

Indicators of the effectiveness of chronic viral hepatitis treatment

1. Normalization of ALT level.
2. Seroconversion of HBe Ag to anti-HBe antibodies.
3. Disappearance from the blood of HBV-DNA -RNA.

4. At the same time it is desirable to eliminate the signs of immune imbalance - in particular, the level of total lymphocytes in absolute terms and CD3 (mature T cells), CD4 (T-helpers), CD8 (T-suppressors), CD16 (natural killers), CD21 (B -cells) and changes in the humoral link, mainly IgA.

According to the effectiveness of HCV therapy are:

1. Primary remission - 2 consecutive normal values of ALT levels during treatment with an interval of at least 2 weeks, the disappearance of HCV RNA or HBV DNA at the end of therapy.

2. Stable remission - normal ALT values, no HCV RNA or HBV DNA for 6 months after the end of therapy.

3. Prolonged remission - normal ALT values, no HCV RNA or HBV DNA for 24 months after the end of therapy.

4. Disappearance of remission (relapse) - within 6 months of treatment after normalization of ALT levels increase its value (2 consecutive tests with an interval of 2 weeks), reappearance in the blood of HCV RNA or HBV DNA after its disappearance.

5. Partial remission - normalization of ALT levels, reduction of viral load.

6. Absence of remission - elevated ALT, the presence of HCV RNA or HBV DNA in the blood at the end of therapy, but not earlier than 3 months of treatment.

Treatment control

1. General clinical examination once every 2 weeks.

2. Determination of ALT activity 1 time in 2 weeks.

Determination of chronic viral hepatitis markers once every 3 months.

ACCESSORIES
LABORATORY RESEARCH

METHODS Microscopic method

Material: blood, bone marrow punctate, cerebrospinal fluid, feces, urine, mucosal material, duodenal ulcers, lymph node punctate.

The bacteriological method consists in isolating the pathogen in pure culture when sowing material from the patient (hemoculture, urine culture, coproculture, vomit, nasopharyngeal material, cerebrospinal fluid, etc.) on special nutrient media.

The virological method is based on the isolation of the virus, using chicken embryos, laboratory animals, cell culture.

Serological methods are based on the antigen-antibody reaction. The results are evaluated by the height of the titer and its dynamics during the disease.

At bacterial diseases apply:

- agglutination reaction (RA);
- passive hemagglutination reaction (RPGA, RNGA);
- complement fixation reaction (COMR);
- precipitation reaction (RP);

At viral diseases apply:

- hemagglutination inhibition reaction (RGGA);
- passive hemagglutination reaction (RNGA);
- complement fixation reaction (COMR);
- neutralization reaction (PH);
- hemadsorption delay reaction;
- molecular hybridization method.

For express diagnostics use:

- immunofluorescence reaction (RIF); passive hemagglutination reaction (RPGA, RNGA);
- complement fixation reaction (COMR);
- enzyme-linked immunosorbent assay (ELISA);
- radioimmunoassay (RIA).

Hematological examination

Indicator	Age of the child	Normative
1	2	3
Hemoglobin, g / l	newborn 1 day	140-200
	up to 4 days	150-200
	5-30 days	110-200
	1-2 months	100-150
	more than 2 years	110-150
Color indicator	all age groups	0,86-1,05
Erythrocytes, 10^{12} × l	up to 4 days	4,4-5,8
	5-30 days	4,1-6,4
	older than 1 month	3,8-5,5
Leukocytes, 10^{9} × l	up to 4 days	9-30
	4-7 days	5-21
	6-12 months	6-17,5
	up to 2 years	6,2-17
	more than 2 years	4,8-10,8
Lymphocytes, %	up to 4 days	31
	4-7 days	41
	6-12 months	56
	up to 2 years	61
	2-5 years	69
	more than 5 years	19-37
Myelocytes, metamyelocytes	missing	missing
Stick core, %	all age groups	1-6
Segmental, %		47-72
Eosinophils, %		0,5-5
Basophils, %		0-1
Erythrocyte diameter, µm		7,55

1	2	3
Platelets, 10^9/l	newborn 1 day	100-290
	up to 4 days	140-300
	5-30 days	150-390
	1 month -2 years	200-473
	more than 2 years	150-450
Reticulocytes, %	newborn 1 day	3-7
	up to 4 days	1,1-4,5
	5-30 days	0,1-1,5
	1 month -2 years	0,5-3,1

Biochemical blood test

Indicator	Normative
1	**2**
Glucose, g / l	3,3-5,5
Sialic acid, Unit	180-200
Lactic acid, mmol / l	0,99-1,75
Cholesterol, mmol / l	3,6-6,2
Beta-lipoproteins, g / l	3,5-6,5
Total protein, g / l	60-80
Albumin,%	50-60
α1-globulin,%	5-8
α2-globulin,%	8-12
β-globulin,%	11-17
γ-globulin,%	11-25
Residual nitrogen, mmol / l	14,3-28,6
Urea, mmol / l	2,5-8,3
Uric acid, mmol / l	0,14-0,3
Creatinine, mmol / l	0,035-0,1
Amylase, g / ml / h	12-32
ALT, mmol / l	0,1-0,7
AST, mmol / l	0,1-0,45
Thymol test, MO	0-4
Sulem test, Jr.	1,6-2,2

1	2
Total bilirubin, μmol / l	8,6-20,5
Direct bilirubin, μmol / l	0-5,1
Indirect bilirubin, μmol / l	до 16
Alkaline phosphatase, mmol / year / l	1,2-6
SRB	+++
Seromucoid, mmol / l	1,2-1,6
OZhSS, μmol / l	4,1-94,9
Iron, μmol / l	19,7-29,9
Potassium, mmol / l	4-6
Sodium, mmol / liter	130-150
Chlorine, mmol / l	95-160
Calcium, mmol / l	2,5-2,7
Phosphorus, mmol / l	1,13-1,62
Magnesium, mmol / l	0,7-1,2
Ceruloplasmin, g / l	1,5-2,3
Fibrinogen, mmol / l	2000-4000
Prothrombin index,%	80-100

Blood immunogram parameters

Normative	**0-28 days**	**1-3 months**	**4-6 months**	**7-12 months в**	**1-2 years**	**3-6 years**	**7-9 years**	**9-13 years**	**Adults**
1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Ig G, g/l	7,5-15	2,7-7,8	1,9-8,6	3,5-11,8	5,2-10,8	6,5-14,1	7,6-13,3	7,7-15,1	8-20
Ig A, g/l	<0,06	0,06-0,58	0,1-0,96	0,36-1,65	0,36-1,65	0,83-2,17	1,08-2,0	1,08-3,25	0,9-4,5
Ig M, g/l	0,11-0,35	0,12-0,87	0,25-1,2	0,36-1,04	0,72-1,60	0,55-2,10	0,55-1,60	0,7-1,50	0,6-2,5
T-limph.,%	58-64					56-62	54-59	57-63	60-66
B-imph.,%	7-16					7-15	8-14	7-14	8-14

1	2	3	4	5	6	7	8	9	10
O-cell.,%	20-35					23-37	26-38	23-36	20-32
Moluls%	9-15					7-11	6-12	7-11	8-12
Index of voltage	1,4-3,6					1,4-3,9	1,4-3,9	1,4-3,9	1,5-3,9

Cerebrospinal fluid examination

Indicators	Normative
Number	100-150ml
Relative density	1003-1008
Pressure	150-200mm water table.
Color	without color
Cytosis (lymphocytes) in 1 µl	ventricular fluid 0-1
The pH reaction	tank fluid 0-1
Total protein, g / l	lumbar fluid 2-3
Glucose, mmol / l	7.35-7.8
Chlorine ions, mmol / l	lumbar fluid 0.22-0.33
Indicators	tank fluid 0.1-0.22
Number	ventricular fluid 0.12-0.22
Relative density	2.78-3.89
Pressure	120-128

The composition of the microflora of the large intestine

Microorganisms	The number of cells per gram of colon content				
	Age from 1 to 9 months				
	Natural feeding	Mixed feeding	Artificial feeding		
Anaerobic association,%	97-99	97-99	97-99	97-99	95-97
Bifidobacterium	10^7-10^{11}	10^6-10^9	10^6-10^8	10^{10}-10^{12}	10^9-10^{12}

Lactobacillus	10^5	10^4	10^4-10^6	10^8-10^{10}	10^7-10^{10}
Propionibacterium	10^7-10^{10}	10^7-10^{10}	10^7-10^{10}	10^7-10^{10}	10^7-10^{10}
Bacteroids	10^6-10^7	10^5-10^9	10^8-10^{10}	0-10^7	10^5-10^{10}
Peptostreptococcus	0-10^5	0-10^5	0-10^5	0-10^5	10^5-10^6
Clostridium	10^1-10^3	10^2-10^4	10^3-10^6	10^3-10^5	10^2-10^5
Eubacterium	0-10^6	0-10^6	0-10^6	0-10^6	10^5-10^7
Aerobic association,%	1-3	1-3	1-3	1-3	3-5
Escherichia: **- lactose fermentation;** **- atypical**	 10^5-10^8 0	 10^6-10^9 0	 10^7-10^9 0	 10^6-10^8 0	 10^6-10^8 ‹ 10%
Group D streptococcus (enterococci)	0	10^5-10^9	10^6-10^9	10^3-10^7	10^5-10^7
Staphylococcus	10^2-10^4	10^3	10^3-10^6	‹10^3	10^3-10^5
Proteus	10^1-10^2	10^2-10^3	10^2-10^3	‹10^3	‹10^3
Yeast-like mushrooms	10^2-10^4	10^1-10^3	10^2-10^4	‹10^4	10^2-10^4

Coprological research

Indicator	Normative
Consistence	decorated
Form	cylindrical
Color	brown
Reaction	neutral, slightly alkaline
Mucus, blood	missing
Muscle fibers	absent, there are single undigested
Connective tissue	is absent
Neutral fat	missing
Fatty acids	missing
Vegetable fiber (digested)	single cells or groups
Vegetable fiber (undigested)	different number
Starch	missing
Iodophilic flora	is absent
Mucus, epithelium	missing
Leukocytes	single

Tests for self-control
Intestinal infections
Variant I

1. Antibacterial therapy for dysentery:

a) the minimum duration of the course is 5-7 days;

b) the maximum duration of the course is determined by the child's condition and the rate of disappearance of symptoms;

c) in the absence of effect from therapy within 3 days should change the antibiotic;

d) the route of administration of antibiotics depends on the severity of the child's condition and the properties of the drug used;

e) all answers are correct.

2. What factor does not determine the morphological changes in the body and the severity of dysentery?

a) bacteremia;

b) invasive properties of microorganisms;

c) infectious dose;

d) endo- or exotoxin of the pathogen;

e) immunological resistance of the microorganism;

3. Salmonella transmission factors are not:

a) water;

b) eggs;

c) milk and lactic acid products;

d) fish and fish products;

e) household items contaminated with salmonella;

g) dried manure of birds and animals;

h) blood-sucking insects;

4. The classification of Salmonella is based on;

a) pathogenicity;

b) c) antigenic structure for O-antigen;

c) H-antigen antigenic structure;

d) antigenic structure for K-antigen;

5. For etiotropic therapy of enteroinvasive Escherichia coli use:

a) cefotaxime;

b) chloramphenicol;

c) ampicillin;

d) ciprofloxacin;

e) all answers are correct.

Variant II

1. The main route of transmission of Escherichia coli caused by enteroinvasive Escherichia coli?

a) water;
b) food;
c) contact and household; d
) all answers are correct.

2. The greatest risk of developing generalized forms of salmonellosis are:
a) children with congenital T-cell immunodeficiency;
b) newborns;
c) children with AIDS;
d) children undergoing immunosuppressive therapy;
e) all answers are correct.

3. As a result of generalized salmonellosis infection in newborns often develop:
a) pyelonephritis;
b) liver abscess;
c) abscess of the spleen;
d) osteomyelitis;
e) purulent meningitis;

4. Possible complications of dysentery:
a) infectious-toxic shock;
b) intestinal bleeding;
c) peritonitis, intussusception;
d) rectal prolapse, paraproctitis;
e) all answers are correct.

5. Which Shigella mainly emit an exotoxin:
a) Grigoriev-Shiga;
b) Larja-Sachs;
c) Flexner;
d) Boyd;
e) Zone;
f) all answers are correct.

Variant III

1. Possible complications of dysentery:

a) infectious-toxic shock;

b) intestinal bleeding;

c) peritonitis, intussusception;

d) rectal prolapse, paraproctitis;

e) all answers are correct.

2. At a contact way of transfer the maximum quantity of a salmonellosis at children is registered:

a) in winter;

b) in the spring;

c) in the summer;

d) in autumn;

3. The source of salmonellosis infection is:

a) bovine animals;

b) pigs, sheep;

c) patient or carrier;

d) poultry, wild pigeons, turtles;

e) all answers are correct.

4. Crucial in the formation of chronic dysentery is:

a) functional insufficiency of the digestive tract;

b) the ability of Shigella transitions to L-shapes;

c) violation of the diet;

d) violation of immunological reactivity.

5. In young children, morphological changes in the intestine in dysentery are observed mainly in the form of:

a) catarrhal colitis;

b) fibrous colitis;

c) diphtheria colitis;

d) ulcerative colitis.

Variant IV

1. Grigoriev-Shita dysentery is not characterized by:
a) the rapid development of colitis;
b) severe severity of infectious toxicosis syndrome;
c) hyperthermia;
d) severe disease;
e) mild course of the disease;
2. Atypical forms of dysentery include:
a) erased;
b) dyspeptic;
c) hypertoxic;
d) subclinical;
e) all answers are correct.
3. What statement is not typical of salmonellosis as a nosocomial infection:
a) the number of cases of nosocomial salmonellosis increases;
b) nosocomial outbreaks of salmonellosis are different long course;
c) nosocomial salmonellosis is more often caused strains of microorganisms with low antibiotic resistance;
d) the largest number of patients occurs in children of the first year of life.
4. The main links in the pathogenesis of salmonellosis:
a) endotoxinemia;
b) bacteremia;
c) water-electrolyte losses;
d) hemodynamic disorders;
e) all answers are correct.
5. Watery diarrhea without fever, persisting for more than 2 weeks in a child of the first year of life, is most characteristic of:
a) enteropathogenic Escherichia coli;
b) enteroinvasive Escherichia coli;
c) enterotoxinogenic Escherichia coli;
d) enterohemorrhagic Escherichia coli;
e) all answers are correct.

Variant V

1. What microorganisms cause the disease, on clinical grounds similar to shigellosis:

a) enteropathogenic;
b) etheroinvasive;
c) enterotoxigenic;
d) enterohemorrhagic;
e) all answers are correct,

2. Which of the following pairs of bacteria produce toxins with the same mechanism of action?

a) Streptococcus pyogenes and Schigella dysenteriae;
b) Escherichia coli and Vibgio scholegae;
c) Vasillus antacis and Clostridium perfringes;
d) Legionella pneumoniae and Salmonella typhi;
e) Klebsiella pneumoniae and Musolacteriym tuberculosis.

3. For the purpose of express diagnostics of dysentery use:

a) bacteriological method;
b) serological method;
c) the method of fluorescent antibodies;
d) coprological research;
e) rectomanoscopy;

4. Common signs of dysentery and appendicitis in its retrocecal location are:

a) local muscle tension;
b) symptoms of peritoneal irritation;
c) increasing leukocytosis;
d) liquid stool with mucus;

5. What clinical form of salmonellosis is most common in newborns:

a) gastrointestinal;
b) generalized;
c) bacterial excretion;
d) erased.

Variant VI

1. A reliable sign that confirms the diagnosis of dysentery are:
a) the presence of hemocolitis;
b) distal colitis syndrome;
c) the degree of intoxication:
d) high content of specific antibodies in the blood;
e) isolation of the pathogen.
2. The clinical symptoms of salmonellosis in children under 3 years of age are dominated by symptoms:
a) gastroenteritis;
b) colitis;
c) generalization of infection;
d) no clinical symptoms, only sowing of microorganisms from feces is registered.
3. For uncomplicated gastrointestinal salmonellosis is not typical:
a) high fever;
b) jaundice, hemorrhagic syndrome;
c) cramp-like abdominal pain;
d) nausea, vomiting, frequent loose stools;
e) dehydration.
4. In the center of dysentery there is no need to conduct:
a) supervision of contacts;
b) control of stools in contact;
c) current disinfection;
d) bacteriological examination of children with intestinal dysfunction;
e) bacteriological examination of all contacts.
5. Salmonella virulence factors:
a) antiphagocytic activity of salmonella;
b) the mobility of salmonella;
c) the ability to produce endotoxin;
d) the ability to survive in phagocytes;
e) resistance to lysis;
f) all answers are correct.

Variant VII

1. Salmonella osteomyelitis is a common problem in children who:
a) have sickle cell anemia;
b) used unpasteurized milk in the anamnesis;
c) received long-term antibiotic therapy;
d) had contact with animals.
2. The course of dysentery can be:
a) acute;
b) protracted;
c) chronic continuous;
d) chronic recurrent;
e) all answers are correct.
3. Among the specific complications of dysentery in children the most typical are:
a) intestinal perforation, peritonitis;
b) intestinal bleeding;
c) intussusception;
d) prolapse of the rectum;
e) cracks, erosions of the anus;
4. Local symptoms that reflect the severity of dysentery are:
a) the nature of the stool;
b) frequency of bowel movements;
c) abdominal pain;
d) gaping of the anus;
e) all answers are correct.
5. For the septic variant of the generalized form of salmonellosis is not typical:
a) fever of the wrong type;
b) prolonged gastrointestinal syndrome;
c) hepatolienal syndrome;
d) the formation of secondary septicemia foci.

Variant VIII

1. In salmonellosis lesions of the nervous system have manifestations:
a) weakness and weakness;
b) headache;
c) sleep disorders;
d) meningeal symptoms;
e) all answers are correct.
2. The criterion of severity in salmonellosis can not be:
a) fever;
b) the severity of intoxication;
c) frequency of bowel movements;
d) the duration of the incubation period;
e) toxic myocardial damage;
3. To reduce the diarrhea syndrome should not be used:
a) enterosorbents;
b) imodium;
c) biological products;
d) enzyme preparations:
e) binders.
4. The diagnosis of Escherichia coli can be established on the basis of:
a) fever, intoxication;
b) the presence of gastroenteritis syndrome;
c) epidemiological data;
d) bacteriological confirmation;
e) coprological research.
5. What group of Escherichia coli causes the most common disease in children of the first year of life:
a) enteropathogenic;
b) enteroinvasive;
c) enterotoxigenic;
d) enterohemorrhagic;
e) all answers are correct.

Variant IX

1. Clinically salmonellosis is a little like:
a) dysentery;
b) cholera;
c) yersiniosis;
d) botulism;
e) food poisoning.
2. The material for bacteriological research in salmonella is:
a) defecation;
b) vomit and gastric lavage;
c) felling;
d) blood;
e) all answers are correct.
3. For the treatment of patients with generalized forms of salmonellosis antibiotics are administered:
a) orally;
b) intramuscular;
c) intravenously;
d) all answers are correct.
4. The main link in the pathogenesis of Escherichia coli:
a) bacteremia;
b) the effect of enterotoxin;
c) allergic reactions.
5. To confirm the diagnosis of Escherichia coli the most reliable method is:
a) bacteriological method;
b) agglutination reaction;
c) indirect agglutination reaction;
d) immunofluorescent method.

Variant X

1. In the treatment of severe forms of salmonellosis in young children is not crucial:

a) oral and parenteral rehydration;

b) oral rehydration;

c) parenteral rehydration;

d) therapy with enzymes, sorbents and eubiotics.

2. A typical breeding ground for Shigella is:

a) stomach;

b) small intestine;

c) mesenteric lymph nodes;

d) large intestine;

e) all answers are correct.

3. Given the growth of antibiotic resistance of salmonella at the present stage, in children under one year of treatment of severe salmonellosis should begin with:

a) cephalosporins of the 3rd generation;

b) 3-generation aminoglycosides;

c) nitrofurans;

d) chloramphenicol;

e) ampicillin;

4. Acute febrile bloody diarrhea with the development of hemolytic anemia, thrombocytopenia and renal failure is most characteristic of:

a) enteropathogenic Escherichia coli;

b) enteroinvasive Escherichia coli;

c) enterotoxic Escherichia coli:

d) enterohemorrhagic Escherichia coli;

e) all answers are correct.

5. For gastroenteritis syndrome with salmonellosis is not typical:

a) persistent nausea, vomiting;

b) cramp-like abdominal pain, which intensifies before defecation;

c) watery, foul-smelling stools;

d) pain on palpation in the epigastric, umbilical, iliocecal areas;

e) frequent bowel movements in small portions in the form of cloudy mucus.

Viral hepatitis

Variant I

1. The gate of the liver includes:
1) hepatic artery,
2) common hepatic duct,
3) portal vein,
4) lymphatic vessels.
a) all answers are correct;
b) correct answers 1.3;
c) correct answers 2,4;
d) correct answers 1.4.
2. The main sign of cytolysis of hepatocytes is:
a) increase in bilirubin in the blood;
b) increasing the activity of ALT and AST;
c) hypoalbuminemia;
d) hypoprothrombinemia;
e) reducing the amount of beta-lipoproteins.
3. For viral hepatitis A is characterized by the following epidemiological patterns, except:
a) high resistance of the pathogen in the environment;
b) the highest incidence among children 1 year of age;
c) the incidence is seasonal;
d) the highest incidence among children 5-9 years.
4. The average duration of the incubation period for viral hepatitis A:
a) 7-40 days;
b) 1.5-6 months;
c) up to 14 days;
d) 1-2 months;
e) 14-50 days.
5. In the peripheral blood in the acute period of viral hepatitis B is not detected:
a) neutrophilia;
b) leukopenia;
c) reduction of ESR;
d) plasma reaction;

e) monocytic reaction.

Variant II

1. What is the symptom of viral hepatitis is a clinical sign of cholestasis:
a) nausea, vomiting;
b) itchy skin;
c) reduction of daily diuresis;
d) edema, ascites;
e) asthenic manifestations.
2. Hepatitis can be caused by all these viruses except:
a) human cytomegalovirus;
b) infectious mononucleosis virus;
c) influenza virus;
d) herpes simplex virus;
e) hemorrhagic fever virus.

3. Hepatitis A virus refers to:
a) enteroviruses (picornoviruses);
b) hepadnaviruses;
c) arboviruses;
d) retroviruses;
e) mixoviruses;
4. The mechanism of action of drugs:
a) stimulates the production of proteins that inhibit virus replication;
b) immuno-modulating effect: promotes the recognition of cytotoxic T-lymphocytes of hepatocytes affected by the virus;
c) antiproliferative action: stimulates cell proliferation, collagen-producing delays the development of cirrhosis;
d) all answers are correct.
5. Hepatitis C virus is not characteristic:
a) the genome of the virus is represented by RNA;
b) distinguish 6 genotypes of the virus;
c) pronounced mutagenicity of the virus, which allows it to avoid an immune response;
d) is rapidly eliminated in the infected organism.

Variant III

1. The most common variant of the pre-jaundice period in viral hepatitis A:
a) arthralgic;
b) flu-like;
c) astheno-vegetative;
d) dyspeptic;
e) latent.
2. The biological signs of cholestasis do not include an increase in blood:
a) cholesterol;
b) phospholipids;
c) bile acids;
d) AST activities;
e) LF activity.
3. The main mechanism of transmission of viral hepatitis A:
a) drip;
b) fecal-oral;
c) parenteral;
d) sexual;
e) perinatal.
4. What pathology is due to the increased formation of immune complexes in viral hepatitis B:
a) skin lesions in the form of urticaria, maculopapular rash;
b) arthralgia;
c) nodular periarteritis;
d) membranous glomerulonephritis;
e) cryoglobulinemia;
f) all answers are correct.
5. Hepatitis D is not characterized by:
a) diseases in those risk groups as hepatitis B;
b) the most common cause of posttransfusion hepatitis;
c) the virus contains RNA;
d) may exacerbate the infection in carriers of hepatitis B;
e) hepatitis D antigen is rarely detected in the blood due to the short circulation period.

Variant IV

1. Hepatitis A virus is not characteristic:
a) contains 4 structural polypeptides;
b) the genome of the virus-RNA;
c) has a reverse transcriptase;
d) stable in the pH range of 3.0 to 9.0;
e) relatively resistant to chlorine.
2. With viral hepatitis A, one of the following routes of infection is not observed:
a) water;
b) food;
c) contact;
d) transmissible.
3. Transmission of hepatitis B virus from mother to fetus occurs:
a) prenatally;
b) intranatally;
c) postnatally;
d) all answers are correct.
4. After the appearance of jaundice in viral hepatitis B, the patient's condition:
a) improves;
b) deteriorating;
c) does not change.
5. Non-typical biochemical parameters of blood serum in mixed infection of viral hepatitis B and viral hepatitis D are:
a) severe cytolysis syndrome;
b) persistent hyperbilirubinemia, which lasts longer than in viral hepatitis B;
c) indicators of aminotransferase activity are higher than in viral hepatitis B;
d) reduced prothrombin index;
e) indirect bilirubin predominates.

Variant V

1. The most effective drug for the treatment of chronic viral hepatitis C:
a) acyclovir;
b) ribavirin;
c) corticosteroids;
d) immunoglobulin;
e) interferon-alpha.
2. The growth of immunity frees the body from the hepatitis A virus:
a) in the first week of jaundice;
b) for 2-3 weeks of jaundice;
c) at 4-5 weeks of jaundice;
d) during the period of convalescence;
e) after reduction of jaundice.
3. The total duration of the incubation period for viral hepatitis B:
a) 7-40 days;
b) 1.5-6 months;
c) up to 11 weeks;
d) 1-2 months;
e) 14-50 days
4. The most common variants of the pre-jaundice period in viral hepatitis B:
A) arthralgic;
B) flu-like;
C) dyspeptic;
D) latent;
E) astheno-vegetative:

a) A + B;
b) B + D;
c) D + D;
d) A + D;
e) B + D.
5. What correctly characterizes Hbe Ag:
a) indicator of recently transferred viral hepatitis B;
b) serological marker indicating recovery and formation of immunity;

c) is found in Hbs-Ag-positive individuals, which indicates a high replicative activity of the virus and a high level of infectivity.

Variant VI

I. Serological markers of chronic hepatitis B do not include:
a) HbsAg;
b) anti Hbe;
c) anti Hbcor IgM;
d) Hbe Ag;
e) anti-CAA IgM.
2. Differential diagnosis of chronic viral hepatitis B is performed with:
a) toxic hepatitis;
b) autoimmune hepatitis;
c) Wilson-Konovalov disease;
d) liver damage in cystic fibrosis;
e) all answers are correct.
3. The main principle of treatment of patients with chronic hepatitis with minimal activity:
a) corticosteroid drugs;
b) immunomodulators;
c) hepatoprotectors;
d) refusal of drug therapy, adherence to diet and regimen.
4. A sign of intoxication with viral hepatitis B can not be:
a) general weakness;
b) nausea, vomiting;
c) headache;
d) acholia of feces;
e) tachycardia.
5. The duration of the incubation period in viral hepatitis C:
a) 7-40 days;
b) 1.5-6 months;
c) up to 14 weeks;
d) 1-2 months;
e) 14-50 days

Variant VII

1. The greatest epidemiological danger at viral hepatitis A is:
a) virus carriers;
b) patients at the end of the incubation period and in the pre-jaundice period;
c) patients in the jaundice period;
d) patients with subclinical forms of the disease;
e) convalescents.
2. The growth of immunity frees the body from the hepatitis A virus:
a) in the first week of jaundice;
b) for 2-3 weeks of jaundice;
c) at 4-5 weeks of jaundice;
d) during convalescence;
e) after reduction of jaundice.
3. The mechanism of viral hepatitis B transmission:
a) sexual;
b) parenteral;
c) parenatal;
d) all answers are correct.
4. The total duration of the incubation period for viral hepatitis E
a) 7-40 days;
b) 1.5-6 months;
c) up to 14 weeks;
d) 1-2 months;
e) 14-50 days
5. The total duration of the pre-jaundice period of viral hepatitis C:
a) 3-7 days;
b) 3 days - 3 weeks;
c) 7-8 days;
d) 3-7 weeks;
e) 3-9 weeks.

Variant VIII

1. After the appearance of jaundice in viral hepatitis A, the patient's condition:
a) improves;
b) remains unchanged;
c) deteriorating.
2. The total duration of the pre-jaundice period of viral hepatitis A:
a) 3-7 days;
b) 3 days - 3 weeks;
c) 7-8 days;
d) 3-7 weeks;
e) 3-9 weeks.
3. Hepatitis C virus belongs to the group:
a) enteroviruses;
b) herpesviruses;
c) flaviviruses;
d) caliciviruses.
4. The most unfavorable for the patient viral hepatitis are:
a) mixed acute viral hepatitis B and delta;
b) hepatitis B without joining the delta virus;
c) combination of chronic hepatitis B with acute delta superinfection.
5. For the epidemiological characteristics of viral hepatitis E is not typical:
a) more often than others, children aged 2-5 years;
b) mostly sick adults 20-35 years;
c) transmitted by water;
d) is characterized by epidemiological spread;
e) there is a severe course of the disease with high mortality among pregnant women.

Variant IX

1. Epidemiological features of viral hepatitis C, except:

a) the most common cause of posttransfusion hepatitis;

b) is widespread among injecting drug users;

c) transmission can be carried out sexually;

d) perinatal transmission of viral hepatitis C is more likely in HIV-infected pregnant women;

e) possible transmissible transmission of the virus;

2. Which indicator is not significant as a criterion for the severity of viral hepatitis B:

a) the degree of hyperbilirubinemia;

b) the severity of general intoxication;

c) hemorrhagic syndrome;

d) increase of thymol test;

e) decrease in the prothrombin index.

3. The signs of hemorrhagic syndrome in viral hepatitis B do not include:

a) bleeding gums;

b) hypoalbuminemia;

c) nosebleeds;

d) microhematuria;

e) decrease in prothrombin in the blood.

4. Which of the following is correct about the action of glucocorticoids in acute viral hepatitis B:

a) stimulate virus replication;

b) increase the frequency of relapses;

c) contribute to the chronicity of viral hepatitis;

d) lead to a temporary decrease in bilirubin;

e) all answers are correct.

5. Confirmation of the diagnosis of viral hepatitis A is the detection of:

a) hepatitis virus in the stool;

b) hepatitis A virus antigen in the blood;

c) antibodies to hepatitis A virus (anti-HAV IgM) in the blood.

Variant X

1. The consequence of viral hepatitis A is usually:
a) recovery;
b) prolonged course;
c) development of chronic active hepatitis;
d) cirrhosis of the liver.
2. Not related to the specific prevention of viral hepatitis A:
a) intramuscular injection of hemoglobin;
b) active immunization with hepatitis A vaccine;
c) the introduction of interferon;
d) there is no correct answer.
3. Hepatitis B virus refers to:
a) enteroviruses;
b) hepadnoviruses;
c) arboviruses;
d) retroviruses;
e) caliciviruses;
4. Which of the characteristics of the hepatitis B virus is not true:
a) resistant to low temperatures;
b) resistant to high temperatures;
c) dies quickly under the action of blood preservatives;
d) resistant to disinfectants;
e) resistant to drying.
5. Peculiarities of viral hepatitis B in children of the 1st year of life are not typical:
a) frequent development of severe fulminant forms;
b) indistinct manifestations of the pre-jaundice period;
c) with the same severity, the intensity of jaundice is greater than in older children;
d) acute onset of the disease, often with high fever.

Tests for differential diagnosis

Variant I

1. What factor is leading in the pathogenesis of pseudotuberculosis?
a) Infectious-toxic-allergic
b) Infectious
c) Toxic
d) Allergic
e) Coagulation
2. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic
3. What temperature is optimal for the growth of the pathogen of pseudotuberculosis
(т° С)?
a) 23-32
b) up to 15
c) 15-20
d) 30-35
e) 36-45
4. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
5. Is the musculoskeletal system damaged by pseudotuberculosis?
a) Very often
b) Always
c) Very rarely
d) Never
e) In the long term of the disease

Variant II

1. The nature of jaundice in pseudotuberculosis:
a) Parenchymal
b) Mechanical
c) Hemolytic
d) Functional hyperbilirubinemia
e) Parenchymal-hemolytic
2. Through which foods is pseudotuberculosis most often transmitted?
a) Salads from raw vegetables
b) Hot dishes
c) Boiled, cold dishes
d) Compotes
e) Conservation
3. The causative agent of yersiniosis:
a) Bacteria
b) Virus
c) The simplest
d) Coke
e) Rickettsia
4. Carriers of yersiniosis:
a) Animals, birds, rodents
b) Even-toed ungulates
c) Insects
d) Fish
e) Ticks
5. Gate of infection in yersiniosis:
a) gastrointestinal tract
b) Damaged skin
c) The mucous membrane of the respiratory tract
d) The mucous membrane of the genitourinary system
e) Rodent bite

Variant III

1. What diagnostic methods can be used not to confirm the diagnosis of intestinal yersiniosis?
a) Allergic tests
b) Serological reactions
c) Finding the pathogen in the blood
d) Finding the pathogen in the feces
e) PCR
2. In what form of intestinal yersiniosis dehydration most often develops?
a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
3. Is it possible to formulate a diagnosis of intestinal yersiniosis on the basis of the clinic?
a) No, bacteriological or serological confirmation is required
b) Yes
c) Not always
d) Yes, but a lumbar puncture is required
e) No, but a urine test is required
4. Which drugs are most effective in the treatment of yersiniosis?
a) Antibiotics of the tetracycline group
b) Immunostimulants
c) Sulfanilamides
d) Desensitizing agents
e) Hepatoprotectors
5. Specific prevention of yersiniosis:
a) There is no specific prevention
b) Live inactivated vaccine
c) Tetracycline antibiotics
d) Aminoquinoline series drugs
e) Deratization

Variant IV

1. The source of infection in pseudotuberculosis is:
a) insects;
b) wild and domestic animals;
c) a sick person;
d) birds.
2. In the pathogenesis of pseudotuberculosis there are the following phases:
a) infection, enteral, regional infection, generalization of infection;
b) infection, regional infection, recovery;
c) enteral, regional infection, generalization of infection;
d) infection, generalization of infection, recovery.
3. The most pronounced changes in pseudotuberculosis are observed in:
a) the cardiovascular system;
b) lungs;
c) joints;
d) the brain.
4. At what age the disease of pseudotuberculosis is almost non-existent:
a) 7-12 years;
b) 13-45 years;
c) 7 months. - 7 years;
d) up to 6 months.
5. The causative agent of pseudotuberculosis is:
a) gram positive bacteria;
b) gram-negative bacteria;
c) diplococci;
d) streptococci;
e) staphylococci.

Variant V

1. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic
2. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
3. Carriers of yersiniosis:
a) Animals, birds, rodents
b) Even-toed ungulates
c) Insects
d) Fish
e) Ticks
4. In what form of intestinal yersiniosis dehydration most often develops?
a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
5. Specific prevention of yersiniosis:
a) There is no specific prevention
b) Live inactivated vaccine
c) Tetracycline antibiotics
d) Aminoquinoline series drugs
e) Deratization

Variant VI

1. What drugs are most effective in the treatment of yersiniosis?
a) Antibiotics of the tetracycline group
b) Immunostimulants
c) Sulfanilamides
d) Desensitizing agents
e) Hepatoprotectors
2. What diagnostic methods can be used not to confirm the diagnosis of intestinal
yersiniosis?
a) Allergic tests
b) Serological reactions
c) Finding the pathogen in the blood
d) Finding the pathogen in the feces
e) PCR
3. Gate of infection in yersiniosis:
a) gastrointestinal tract
b) Damaged skin
c) The mucous membrane of the respiratory tract
d) The mucous membrane of the genitourinary system
e) Rodent bite
4. Through which foods is pseudotuberculosis most often transmitted?
a) Salads from raw vegetables
b) Hot dishes
c) Boiled, cold dishes
d) Compotes
5. In what form of intestinal yersiniosis dehydration most often develops?
a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice

Variant VII

1. At what form of intestinal yersiniosis dehydration most often develops?
a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
2. Which drugs are most effective in the treatment of yersiniosis?
a) Antibiotics of the tetracycline group
b) Immunostimulants
c) Sulfanilamides
d) Desensitizing agents
e) Hepatoprotectors
3. At what age the disease of pseudotuberculosis is almost non-existent:
a) 7-12 years;
b) 13-45 years;
c) 7 months. - 7 years;
d) up to 6 months.
4. In the pathogenesis of pseudotuberculosis there are the following phases:
a) infection, enteral, regional infection, generalization of infection;
b) infection, regional infection, recovery;
c) enteral, regional infection, generalization of infection;
d) infection, generalization of infection, recovery.
5. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic

Variant VIII

1. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
2. The most pronounced changes in pseudotuberculosis are observed in:
a) the cardiovascular system;
b) lungs;
c) joints;
d) the brain
3. Which drugs are most effective in the treatment of yersiniosis?
a) Antibiotics of the tetracycline group
b) Immunostimulants
c) Sulfanilamides
d) Desensitizing agents
e) Hepatoprotectors
4. In what form of intestinal yersiniosis dehydration most often develops?
a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
5. Gate of infection in yersiniosis:
a) gastrointestinal tract
b) Damaged skin
c) The mucous membrane of the respiratory tract
d) The mucous membrane of the genitourinary system
e) Rodent bite

Variant IX

1. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic
2. Is the musculoskeletal system damaged by pseudotuberculosis?
a) Very often
b) Always
c) Very rarely
d) Never
e) In the long term of the disease
3. The nature of jaundice in pseudotuberculosis:
a) Parenchymal
b) Mechanical
c) Hemolytic
d) Functional hyperbilirubinemia
e) Parenchymal-hemolytic
4. Carriers of Yersinia:
a) Animals, birds, rodents
b) Even-toed ungulates
c) Insects
d) Fish
e) Ticks
5. Is it possible to formulate a diagnosis of intestinal yersiniosis on the basis of the clinic?
a) No, bacteriological or serological confirmation is required
b) Yes
c) Not always
d) Yes, but a lumbar puncture is required
e) No, but a urine test is required

Variant X

1. In the pathogenesis of pseudotuberculosis there are the following phases:
a) infection, enteral, regional infection, generalization of infection;
b) infection, regional infection, recovery;
c) enteral, regional infection, generalization of infection;
d) infection, generalization of infection, recovery.
2. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic
3. The causative agent of pseudotuberculosis is:
a) gram positive stick;
b) gram-negative stick;
c) diplococci;
d) streptococci;
e) staphylococci.
4. At what age the disease of pseudotuberculosis is almost non-existent:
a) 7-12 years;
b) 13-45 years;
c) 7 months. - 7 years;
d) up to 6 months.
5. Specific prevention of yersiniosis:
a) There is no specific prevention
b) Live inactivated vaccine
c) Tetracycline antibiotics
d) Aminoquinoline series drugs
e) Deratization

Variant XI

1. What drugs are most effective in the treatment of yersiniosis?
a) Antibiotics of the tetracycline group
b) Immunostimulants
c) Sulfanilamides
d) Desensitizing agents
e) Hepatoprotectors
2. In what form of intestinal yeriniosis dehydration most often develops? a) Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
3. In the pathogenesis of pseudotuberculosis there are the following phases:
a) infection, enteral, regional infection, generalization of infection;
b) infection, regional infection, recovery;
c) enteral, regional infection, generalization of infection;
d) infection, generalization of infection, recovery.
4. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
5. Gate of infection in yersiniosis: a) gastrointestinal tract
b) Damaged skin
c) The mucous membrane of the respiratory tract
d) The mucous membrane of the genitourinary system
e) Rodent bite.

Variant XII

1. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
2. In what form of intestinal yersiniosis dehydration most often develops?
a)Gastroenterocolitis
b) Septic
c) Mesenteric lymphadenitis
d) Acute appendicitis
e) Jaundice
3. The most pronounced changes in pseudotuberculosis are observed in:
a) cardiovascular system; b) lungs; c) joints; d) the brain.
4. In which age, the disease of pseudotuberculosis is not found:
a) 7-12 years;
b) 13-45 years;
c) 7 months. - 7 years;
d) up to 6 months.
5. In the pathogenesis of pseudotuberculosis distinguish the following phases:
a) infection, enteral, regional infection, generalization infection;
b) infection, regional infection, recovery;
c) enteral, regional infection, generalization infection;
d) infection, generalization infection, recovery.

Variant XIII

1. What factor is leading in the pathogenesis of pseudotuberculosis?
a) Infectious-toxic-allergic
b) Infectious
c) Toxic
d) Allergic
e) Coagulation
2. What diseases are pseudotuberculosis?
a) Zoonotic
b) Anthroponotic
c) Zooanthroponoses
d) Anthropozoonotic
e) Parasitic
3. What temperature is optimal for the growth of the pathogen of pseudotuberculosis
(т° С)?
a) 23-32
b) up to 15
c) 15-20
d) 30-35
e) 36-45
4. The causative agent of pseudotuberculosis:
a) Bacteria
b) Viruses
c) The simplest
d) Chlamydia
e) Rickettsia
5. Is the musculoskeletal system damaged by pseudotuberculosis?
a) Very often
b) Always
c) Very rarely
d) Never
e) In the long term of the disease

Variant XIV

1. Which of the following changes is not characteristic of the vagus phase of scarlet fever?

a) bradycardia;

b) lowering blood pressure;

c) white dermographism appears quickly (reduction of the latent period, №-7-8) and slowly disappears (lengthening of the obvious period, №-2,5-3);

d) dry skin.

2. Which of the following is not characteristic of severe toxic scarlet fever?

a) hyperthermia;

b) darkening of consciousness, delirium, meningeal symptoms;

c) collapse;

d) necrotic angina, periadenitis, adenophlegmon.

3. Which of the following antibiotics should not be used to treat scarlet fever?

a) gentamicin;

b) phenoxymethylpenicillin;

c) ceftriaxone;

d) erythromycin.

4. What disease does not occur in a convalescent of typical scarlet fever after new contact with β-hemolytic group A streptococcus?

a) recurrence of scarlet fever;

b) sore throat;

c) were;

d) pyoderma.

5. Which of the following is not characteristic of scarlet fever?

a) lamellar exfoliation of the skin;

b) pigmentation after the rash;

c) a clean nasolabial triangle;

d) "papillary" tongue.

Variant XV

1. Which of the following is characteristic of the septic form of scarlet fever?
a) hyperthermia;
b) darkening of consciousness, delirium, meningeal symptoms;
c) collapse;
d) necrotic angina, periadenitis
2. Which route of transmission is not characteristic of scarlet fever?
a) contact;
b) airborne;
c) alimentary;
d) water.
3. Which of the following complications is not characteristic of scarlet fever?
a) mastoiditis;
b) glomerulonephritis;
c) paresis of the soft palate;
d) sinusitis.
4. What localization of the rash is not characteristic of scarlet fever?
a) on the lateral surfaces of the chest;
b) in the lower abdomen;
c) in the lumbar region;
d) in the groin area.
5. Which antibiotic is the drug of choice in the treatment of scarlet fever?
a) benzylpenicillin;
b) gentamicin;
c) tetracycline;
d) polymyxin.

Variant XVI

1. What causes autonomic disorders at the beginning of scarlet fever?
a) sympathicotonia;
b) vagotonia;
c) do not appear;
d) sympathicoparesis.
2. Which of the following statements about the etiopathogenesis of scarlet fever is
incorrect?
a) scarlet fever pathogens produce exotoxins;
b) antitoxic immunity after the disease is general (for all serovars), stable;
c) antibacterial immunity type-specific, unstable;
d) recurrent diseases of scarlet fever are often possible.
3. Which of the following patients can not be a source of scarlet fever?
a) patients with typical scarlet fever;
b) patients with erased forms of scarlet fever;
c) patients with erysipelas;
d) patients with tonsillitis caused by B-hemolytic group B streptococcus.
4. What is the contagiousness index characteristic of scarlet fever?
a) 0.5%;
b) 40%;
c) 75%;
d) 90%.
5. Which of the following is not the gateway to scarlet fever?
a) in girls, the mucous membranes of the external genitalia;
b) mucous membrane of the oropharynx;
c) morning surface;
d) burn surface.

Variant XVII

1. What rash is characteristic of scarlet fever?

a) vesicular;

b) vesiculopustular;

c) petechial;

d) fine-spotted.

2. Which of the following symptoms is not characteristic of extrapharyngeal scarlet fever?

a) sore throat;

b) small-spot rash;

c) fever;

d) intoxication.

3. Which of the following is a constant symptom of typical scarlet fever?

a) small-spot rash on a hyperemic background;

b) intoxication;

c) sore throat;

d) "crimson" tongue.

4. Which of the following symptoms is not characteristic of scarlet fever?

a) white dermographism with prolonged latent and shortened explicit period;

b) limited hyperemia of the throat;

c) rash with a predominant localization on the hands and feet;

d) dry skin.

5. Which of the characteristics of scarlet fever rash is incorrect?

a) fine-spotted;

b) petechial;

c) miliary;

d) urticarial.

Variant XVIII

1. Which of the following antibiotics will be most effective in treating severe scarlet fever?

a) polymyxin B (15-20 thousand IU / kg / day intravenously or intravenously in 3-4 injections);

b) lincomycin (10-20 mg / kg / day intravenously in two injections);

c) ciprofloxacin (20-30 mg / kg / day intravenously in 2 injections);

d) gentamicin (5-10 mg / kg / day intravenously or intravenously in 3 injections);

2. Which of the following factors of pathogenicity of B-hemolytic streptococcus group A is the main one?

a) protein T

b) capsule

c) exotoxin;

d) hemolysin (streptolysin) O.

3. Which of the following causes prevents the formation of intense antitoxic immunity in scarlet fever, allows recurrence of scarlet fever?

a) rickets in a child;

b) large doses of penicillin;

c) artificial feeding in the first year of life in the anamnesis;

d) frequent ARI in the anamnesis.

4. In how many days from the beginning of scarlet fever convalescents can be accepted in children's collective?

a) 5 days after the appearance of the last element of the rash;

b) on the 5th day;

c) in 22 days;

d) in 17 days.

5. What immunity is developed after the transferred scarlet fever?

a) stable antitoxic;

6) unstable antibacterial;

c) resistant antibacterial;

d) unstable antitoxic.

Variant XIX

1. Which of the following factors of pathogenicity of B-hemolytic streptococcus
group A causes scarlet fever rash?
a) protein T;
b) streptokinase;
c) hemolysin (streptolysin) O;
d) exotoxin (erythrogenic toxin, Dick's toxin).
2. What causes autonomic disorders after an acute period of scarlet fever?
a) are not conditioned by anything;
b) vagotonia;
c) sympathicotonia;
d) sympathicoparesis.
3. For how long from the beginning of the disease it is necessary to isolate a patient
with scarlet fever?
a) for 30 days;
b) for 5 days; at complications - on 10;
c) for 10 days;
d) for 9 days.
4. What course of scarlet fever is not typical for young children?
a) children under one year of age suffer from scarlet fever very rarely;
b) in non-immune children, scarlet fever often occurs in the septic type with severe
necrotic angina and numerous purulent-necrotic complications;
c) in non-immune children, scarlet fever often occurs in a toxic type with darkening of
consciousness, convulsions, meningeal symptoms;
d) very rarely there are manifestations of allergies and complications of infectious-
allergic nature,
5. What pathogen causes scarlet fever?
a) Staphylococcus aureus;
b) the B-2 virus;
c) K1. Pneumonie;

d) B-hemolytic group A streptococcus.

Variant XX

1. Which of the following symptoms is not characteristic of scarlet fever?
a) severe intoxication;
b) small-spot rash on a hyperemic skin background;
c) limited hyperemia of the throat;
d) significant swelling of the tonsils, subcutaneous tissue of the neck.
2. Which of the following symptoms of scarlet fever is incorrect?
a) small-spotted rash on a background of pale skin;
b) there is no rash in the area of the nasolabial triangle;
c) the symptom of Pastia is characteristic;
d) some elements of the rash are miliary in nature.
3. What component of the pathogenesis of scarlet fever is associated with significant
hemodynamic disorders, especially in severe forms?
a) toxic;
b) septic;
c) allergic;
d) toxic-septic.
4. What component of the pathogenesis of scarlet fever is associated with the occurrence of myocarditis, glomerulonephritis?
a) toxic;
b) allergic;
c) septic;
d) toxic-septic.
5. What disease occurs in a child at the first contact with B-hemolytic group A streptococcus?
a) were;
b) sore throat;
c) scarlet fever;
d) pneumonia.

Variant XXI

1. Scarlet fever is caused by the following pathogen:
a) staphylococcus;
b) B-hemolytic streptococcus group A;
c) group B streptococcus;
d) all streptococci.
2. Which of the following fractions of streptococcal toxin causes skin scarlet fever?
a) allergen;
b) erythrogenic toxin;
c) hyaluronidase;
d) leukocidin;
e) hemolysin.
3. Scarlet fever toxin has tropism mainly to:
a) sympathetic autonomic nervous system;
6) parasympathetic autonomic nervous system;
c) the cardiovascular system;
d) lymphatic system;
e) all answers are correct.
4. The minimum incubation period for scarlet fever:
a) 1 day;
b) 2 days;
c) 3 days;
d) 5 days;
e) 10 days.
5. The source of infection with scarlet fever is:
a) patients with scarlet fever;
b) patients with streptococcal infection;
c) patients with nasopharyngitis;
d) convalescents of scarlet fever;
e) all answers are correct.

Variant XXII

1. The maximum incubation period for scarlet fever:
a) 1 day;
b) 3 days;
c) 5 days;
d) 12 days;
e) 14 days.
2. Erythrogenic toxin of streptococcus in patients with scarlet fever is not involved in
the formation of:
a) rash;
b) dry skin;
c) changes in dermographism;
d) tachycardia;
e) arthralgia.
3. Group A of B-hemolytic streptococci is characterized by:
a) the presence of a common group-specific toxin;
b) resistance to p-lactam antibiotics;
c) thermal stability;
d) the ability to induce stable antimicrobial immunity;
e) resistance to disinfectants.
4. The entrance gate for scarlet fever can not be:
a) pharynx;
b) upper respiratory tract;
c) lungs;
d) gastrointestinal tract;
e) intact skin.
5. Ways of transmission in scarlet fever:
a) drip;
b) transmissible;
c) parenteral;
d) all answers are correct.

Variant XXIII

1. The main symptoms in the early diagnosis of scarlet fever are, in addition to:
a) acute onset of the disease;
b) significant intoxication;
c) swelling of the tonsils, subcutaneous tissue of the neck;
d) there is no correct answer.
2. With scarlet fever, the following cardiovascular changes are possible, except:
a) tachycardia;
b) increase in blood pressure;
c) systolic murmur;
d) the rhythm of the "gallop";
e) muffled heart tones.
3. Miliary rash with scarlet fever indicates:
a) unfavorable prognosis;
b) the possibility of developing allergic complications;
c) the possibility of developing septic complications;
d) favorable prognosis of the disease;
e) there is no prognostic value.
4. In the blood of patients with scarlet fever are not found:
a) leukocytosis;
b) neutrophilia;
c) shift the formula to the left;
d) early eosinophilia;
e) anemia.
5. Scarlet fever rash is absent on the following parts of the body:
a) bending surfaces of the extremities; .
b) skin folds;
c) lateral surfaces of the chest;
d) chin;
e) cheeks.

Variant XXIV

1. Which of the following is not characteristic of scarlet fever?
a) lamellar exfoliation of the skin;
6) pigmentation after the rash;
c) a clean nasolabial triangle;
d) "papillary" tongue.
2. What localization of the rash is not characteristic of scarlet fever?
a) on the lateral surfaces of the chest;
6) in the lower abdomen;
c) in the lumbar region;
d) in the groin area.
3. Which of the following statements about the etiopathogenesis of scarlet fever is
incorrect?
a) scarlet fever pathogens produce exotoxins;
6) antitoxic immunity after the disease is general (for all serovars), stable;
c) antibacterial immunity type-specific, unstable;
d) recurrent diseases of scarlet fever are often possible.
4. What rash is characteristic of scarlet fever?
a) vesicular;
6) vesiculopustular;
c) petechial;
d) fine-spotted.
5. Which of the following causes prevents the formation of intense antitoxic immunity
in scarlet fever, allows recurrence of scarlet fever?
a) rickets in a child;
6) large doses of penicillin;
c) artificial feeding in the first year of life in the anamnesis;
d) frequent ARI in the anamnesis.

Variant XXV

1. In how many days from the beginning of scarlet fever convalescents can be accepted in children's collective?

a) 5 days after the appearance of the last element of the rash;

6) on the 5th day;

c) in 22 days;

d) in 17 days.

2. What causes autonomic disorders after an acute period of scarlet fever?

a) are not conditioned by anything;

6) vagotonia;

c) sympathicotonia;

d) sympathicoparesis.

3. What component of the pathogenesis of scarlet fever is associated with significant

hemodynamic disorders, especially in severe forms?

a) toxic;

6) septic;

c) allergic;

d) toxic-septic.

4. The minimum incubation period for scarlet fever:

a) 1 day;

6) 2 days;

c) 3 days;

d) 5 days;

e) 10 days.

5. Ways of transmission in scarlet fever:

a) drip;

6) transmissible;

c) parenteral;

d) all answers are correct.

Variant XXVI

1. Which of the following indicates in favor of the bulbar form of polio?

a) disturbances of consciousness and pyramidal signs;

b) disturbance of consciousness and convulsive syndrome;

c) symptoms of lesions of IX and X pairs of cranial nerves with flaccid paresis;

d) all of the above.

2. Which of the following statements about the polio vaccine used in Ukraine is incorrect?

a) low-reactogenic vaccine;

b) inactivated (killed) vaccine;

c) causes both humoral and cellular immunity;

d) is administered orally.

3. What antibodies (immunoglobulins) cause the formation of local immunity after the

penetration of polio vaccine into the intestine?

a) Ig M;

b) Ig G;

c) IgA;

d) Ig E.

4. What will be the doctor's tactics in case of polio in the children's team?

a) emergency vaccination of children not vaccinated against polio;

b) emergency vaccination is not carried out at all;

c) emergency one-time vaccination of all contact children;

d) emergency vaccination is carried out only in children with low titers of specific

antibodies.

5. What does not happen in the paralytic period of polio?

a) normal body temperature;

b) areflexia;

c) seizures of clonic-tonic nature;

d) the affected extremities are cold, pale, cyanotic.

Variant XXVII

1. What disorders are characteristic of the paralytic period of the spinal cord forms of polio?
a) impaired sensitivity;
b) pyramid signs;
c) loss of pelvic functions;
d) everything is not typical.
2.Which of these symptoms is not characteristic of the pontine form of polio?
a) facial asymmetry;
b) lack of active movements in the palms of the hands;
c) one-sided laughter;
d) incomplete closing of the eyelids.
3.Which of the symptoms does not occur in the paralytic period?
a) oliguria;
b) vomiting;
c) diarrhea;
d) anorexia.
4. Which of the following forms of polio is paralytic?
a) Pontinha;
b) "small illness";
c) meningeal;
d) non-transparent.
5. Which of the following forms of polio is the mildest?
a) Pontinha;
b) bulbar;
c) meningeal;
d) non-transparent.

Variant XXVIII

1. Which of the following forms of polio is the most dangerous?
a) meningeal;
b) bulbar;
c) spinal;
d) "small illness".
2. Which parts of the CNS are most often affected by the polio pathogen?
a) the nucleus of the motor cranial nerves;
b) meninges;
c) motor cells of the anterior horns of the spinal cord;
d) all these departments.
3. Which of the following glucocorticoids is the drug of choice in the treatment of
cerebral edema?
a) prednisolone;
b) prednisone;
c) hydrocortisone;
d) dexamethasone.
4.Which of these corticosteroids has a pronounced mineralocorticoid activity (contributes to fluid retention in tissues)?
a) prednisolone;
b) methylprednisolone;
c) dexamethasone;
d) triamcinolone.
5. What form of polio is most common?
a) spinal;
b) bulbar;
c) Pontinha;
d) non-transparent.

Variant XXIX

1. Which of the following is characteristic of the development of paralysis in polio?

a) acute, rapid onset from several hours to 1-2 days;
b) asymmetric placement of paralysis;
c) the absence of sensitivity disorders, pelvic disorders;
d) all of the above is typical.

2. What clinical manifestations are characteristic of the spinal form of polio?

a) hyperesthesia of the skin on the damaged extremities;
b) hyperreflexia on the affected extremities;
c) clonic muscle spasms on damaged limbs;
d) all of the above is not typical.

3. What is not typical for the paralytic period of spinal polio?

a) central paralysis;
b) peripheral paralysis;
c) sudden development of paralysis;
d) asymmetric spread of paralysis.

4. What indicators of cytosis are characteristic of normal cerebrospinal fluid?

a) 0.001-0.01-109 / l, neutrophils;
b) 0.1-1, MO9 / l, neutrophils;
c) 0.001-0.01-109 / l, lymphocytes;
d) 0.03-0.06-109 / l, lymphocytes.

5. What is the normal pressure of the cerebrospinal fluid (mm.vod.st.)?

a) 200-300;
b) 400-500;
c) 120-180;
d) 120-440.

Variant XXX

1. The etiology and epidemiology of poliomyelitis are characterized by the following

signs:

a) the pathogen belongs to the genus of intestinal infections;

b) diseases become more frequent in the summer-autumn period; V-.

c) source of infection - a sick person;

d) all answers are correct.

2. For the preparalytic stage of polio is not typical:

a) lethargy, drowsiness;

b) the appearance of pain in the spine and extremities;

c) the appearance of meningeal syndrome;

d) development by the court;

e) increase in body temperature.

3. One of the following symptoms is not typical for polio:

a) the disease is biphasic with fever, which precedes the appearance of paralysis;

b) in the cerebrospinal fluid is sharply reduced sugar content;

c) may begin acutely with headache and vomiting;

d) the appearance of pain in skeletal muscles, a decrease in their tone;

e) the appearance of flaccid paralysis.

4. Differential diagnosis of poliomyelitis is performed with;

a) diphtheria polyneuritis;

b) polyradiculoneuropathy;

c) encephalitis;

d) myelitis;

e) all answers are correct.

5. For laboratory diagnosis of polio use:

a) virological (virus excretion from feces, blood, mucus);

b) serological;

c) all the answers are correct.

Variant XXXI

1. What is uncharacteristic of polio?
a) infection occurs through dirty hands;
b) the virus replicates in the intestine and CNS;
c) the virus is transmitted by airborne droplets;
d) the lesion is determined mainly in ganglion cells and cells of the anterior horns of
the spinal cord;
e) all answers are correct.
2. The paralytic stage of polio is characterized by the following symptoms:
a) improving the patient's well-being;
b) decreased muscle tone and tendon reflexes;
c) the development of flaccid paralysis;
d) normalization of body temperature;
e) all answers are correct.
3. The polio virus is not detected in;
a) if;
b) blood;
c) urine;
d) nasopharyngeal lavage and cerebrospinal fluid;
e) cadaveric material.
4.Prophylactic measures for polio:
a) isolation of the patient;
b) routine vaccination with a live vaccine;
c) all answers are correct;
d) contact care for 21 days;
e) disinfection of the hearth.
5. A child of 3 years - fever, sudden weakness of the left leg, the child is not vaccinated. What additional symptoms will help diagnose polio?
a) lack of reflexes in the affected leg;
b) lowering the temperature before the development of paralysis;
c) normal cerebrospinal fluid;
d) the development of the disease in January;
e) symmetrical ascending paralysis.

Variant XXXII

1. What is the percentage of paralytic forms of all forms
polio?
a) more than 1%;
b) 10% or more;
c) 20% or more;
d) up to 50%.
2. How many types of polio viruses do you know?
a) one;
b) two;
at three o'clock;
d) four.
3. What forms of polio do you know?
a) asymptomatic;
b) abortive;
c) Non-paralytic;
d) paralytic;
e) all answers are correct.
4. Duration of the incubation period:
a) 2-10 days;
b) 4-20 days;
c) 2-35 days;
d) 5-60 days;
e) more than 60 days.
5. What complications are associated with respiratory failure and pulmonary
Ventilation acute period?
a) respiratory failure, hypoxia;
b) asphyxia due to inspiration of food, saliva;
c) pulmonary edema;
d) pulmonary embolism (against the background of venous stasis);
e) sudden cessation of breathing;
e) all answers are correct.

Variant XXXIII

1. Meningeal form of polio refers to:

a) abortive form;

b) visceral form;

c) non-paralytic;

d) paralytic.

2. At what age do you start polio vaccination?

a) on the 5th day of life;

b) in the 1st month;

c) in 3 months;

d) at 6 months;

e) at 12 months.

3. Which of the following speaks in favor of the bulbar form of polio?

a) disturbances of consciousness and pyramidal signs;

b) disturbance of consciousness and convulsive syndrome;

c) symptoms of lesions of IX and X pairs of cranial nerves with flaccid paresis;

d) all of the above is paravil.

4. What will be the doctor's tactics in case of polio in a children's team?

a) emergency vaccination of children not vaccinated against polio

b) emergency vaccination is not carried out at all;

c) emergency vaccination is given to all contact children;

d) emergency vaccination is given to children with low titers of specific antibodies.

5. What does not happen in the paralytic period of polio?

a) seizures of clonic-tonic nature;

b) normal body temperature;

c) areflexia;

d) the affected extremities are pale, cold, cyanotic.

Variant XXXIV

1. What disorders are characteristic of the paralytic period of the spinal form of polio?

a) impaired sensitivity;
b) pyramid signs;
c) loss of pelvic functions;
d) everything is uncharacteristic.

2. Which of the following forms of polio is the most dangerous?

a) meningeal;
b) bulbar;
c) spinal;
d) small illness".

3. Which of the following glucocorticoids is the drug of choice in the treatment of

cerebral edema?

a) prednisolone;
b) prednisone;
c) hydrocortisone;
d) dexamethasone.

4. What age children most often get polio?

a) up to 3 months of life;
b) from 4 months to a year;
c) 2-7 years;
d) 8-15 years.

5. How is there no period in the paralytic form of polio?

a) the initial period;
b) preparalytic period;
c) paralytic period;
d) recovery period;
e) residual period.

Variant XXXV

1. What is not typical for the paralytic period of the spinal cord polio?
a) central paralysis;
b) peripheral paralysis;
c) sudden development of paralysis;
d) asymmetric spread of paralysis.
2. Which parts of the CNS are most often affected by the polio pathogen?
a) the nucleus of the motor cranial nerves;
b) meninges;
c) motor cells of the anterior horns of the spinal cord;
d) all these departments.
3.Which of these symptoms is not characteristic of the pontine form polio?
a) facial asymmetry;
b) lack of active movements in the palms of the hands;
c) one-sided laughter;
d) incomplete closing of the eyelids.
4. At what time after vaccination can develop polio vaccine-associated?
a) up to 30 days;
b) up to 40 days;
c) up to 60 days;
d) never.
5.Which drug is considered etiotropic in the treatment of polio?
a) hemoglobin;
b) interferon;
c) antibiotics;
d) glucocorticoids;
e) etiotropic treatment has not been developed.

Tasks

1. Boy K., 9 years old, was hospitalized in the infectious department for viral hepatitis (type B). On the seventh day after hospitalization, the patient developed lethargy, apathy, anxiety, sleep disturbances (drowsiness during the day, insomnia at night), speech became slow, sometimes confused, lost appetite, joined by nausea, vomiting, abdominal pain. A day later, jaundice increased significantly, there were hallucinations and a kind of trembling hands ("slapping" tremor). On examination: the patient is stunned, daydreaming, body temperature 38 ° C, "liver odor" from the mouth, skin hemorrhages, convulsive twitching of certain muscle groups. Expressed yellowing of the skin and sclera. Pulse 108 per 1 min, rhythmic, weak filling, soft. BP 80/50 mm Hg. Art. Heart tones are weakened, rhythmic activity, tachycardia. Vesicular respiration in the lungs. The abdomen is soft, painful in the epigastrium and right hypochondrium. The liver is soft, its transverse size along the midclavicular line has decreased by 3 cm. The spleen is not palpable. Urination is infrequent, urine is dark yellow.

Blood test: leukocytes 13.0-10^9 / l, ESR 36 mm / h; bilirubin 230 μmol / l, total protein 60 g / l, albumin 34.2%, globulins: “i - 6%, <x2; - 8%, P - 12.5%, y - 38.3%, prothrombin 50 %, alkaline phosphatase 50 IU (norm up to 90 IU), cholinesterase 820 IU (norm 1800-3800 IU), ALT 0.38 mmol / l, AST 0.42 mmol / l. ECG: dystrophic changes of the myocardium. EEG: increase in amplitude and decrease in frequency of rhythmic activity waves.

2. Patient Sh., 11 years old, was admitted to the hospital with complaints of "bloody" vomiting, heaviness in the epigastric region and in both hypochondria, loss of appetite, weakness, itchy skin, mostly at night.

Ill since the age of 5, when jaundice appeared. He was treated in the infectious department.

On examination: the condition is severe, the patient is disoriented in time and space, apathetic, drowsy, impaired coordination, hand tremor, fibrillar muscle twitching, pupils moderately narrowed, deep tendon reflexes are suppressed.

The skin and visible mucous membranes are jaundiced. Numerous bright vascular "stars" on the back, shoulders, chest and face. Raspberry tongue, "liver" palms, subcutaneous fat and the muscular system is underdeveloped. Pulse 92 per 1 min, rhythmic, weak filling, soft. BP 90/60 mm Hg. Art. heart sounds are weakened,

systolic murmur is heard at all points. Breathing is accelerated, periods like Kusmaul. The abdomen is enlarged, the veins of the anterior abdominal wall are dilated, the navel is bulging. The liver protrudes 11 cm from under the costal arch, dense, pointed edge. The spleen is enlarged. Free fluid in the abdominal cavity is determined, there is a symptom of "floating ice". Dark feces.

In the blood: anemia (erythrocytes 2,4-10^{I2} / l, hemoglobin 90 g / l, anisocytosis, poikilocytosis), ESR 58 mm / h; bilirubin 71.8 μmol / l, the reaction is direct; total protein 53 g / l, albumin 31.6%, y-globulin 41%, ALT 0.65 mmol / l, AST 0.87 mmol / l.

Task. Determine the amount of differentiated care for patients with various forms of hepatic coma

taking into account etiological, provoking factors and pathogenetic mechanisms.

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Age	Vaccination aginst					
1st day		Hepatitis B				
3-5 day	Tuberculosis					
1st month		Hepatitis B				
2 months			Diphteria, whooping cough, tetanus	Polyomielitis IPV	Hemophilic infection	
4 months			Diphteria, whooping cough, tetanus	Polyomielitis IPV	Hemophilic infection	
6 months		Hepatitis B	Diphteria, tetanus	Polyomielitis OPV	Hemophilic infection	
12 months						Measles, rubella, parotitis
18 months			Diphteria, whooping cough, tetanus	Polyomielitis OPV	Hemophilic infection	
6 years			Diphteria, tetanus	Polyomielitis OPV		Measles, rubella, parotitis
14 years			Diphteria, tetanus	Polyomielitis OPV		
18 years			Diphteria, tetanus			
Adults			Diphteria, tetanus			

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