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**VIRAL HEPATITES. HIV-INFECTION.  
AIDS-ASSOCIATED INFECTIONS AND INVASIONS.  
HERPESVIRUS INFECTIONS**

Manual for practical training and independent work  
of students for the 5th year of the Medical Faculty  
on Infectious Diseases to the module №2

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## Introduction

Viral hepatitis and HIV infection is an acute problem of modern infectious diseases, both in our country and abroad, as number of patients with HCV infection and HIV-infected people is increasing from year to year. The incidence of VH and HIV infection are comparable in terms of growth. The epidemic process is characterized by ubiquitous and high intensity in all age groups. The steady growth of the second harmonic and HIV is inextricably linked with the spread of drug addiction. With special intensity in the epidemic process involved persons aged 15 to 35 years.

Viral hepatitis (VH), HIV-infection, Herpesvirus infections - anthroponosis groups, for the most part, ubiquitous infections, including currently about 10 independent nozoform. Despite the presence of these diseases etiopathogenetical, epidemiological and clinical differences they all share expressed hepatotropic pathogens and related biochemical and clinical manifestations of liver disease. According to WHO estimates infection pathogens SH in the world is about one billion people. Currently, the group consists of SH: viral hepatitis A (HA), B (HB), C (HC), D (HD), E (HE), G (HG) and hepatitis caused by viruses and TTV SEN. Due to the increasingly widespread introduction of modern virology, immunochemical and molecular biological methods of diagnosis in the moment of practitioners is possible to verify the HA, HB, HC, HD. In hepatology centers there are test systems for the laboratory diagnosis of GE, r g et al. Modern methods of laboratory diagnosis of HS based on specific antigens and antibody markers of these diseases. However, the clinical interpretation of the results of these studies presents certain difficulties for practitioners.

This manual presents modern data on the etiology, epidemiology, pathogenesis, clinical manifestations, diagnosis and treatment of acute viral hepatitis, HIV-infection, AIDS-associated infections and invasions, Herpesvirus infections as well as the indicative amount of the scheme, the timing of examination of patients and interpretation of the results. The annex presents the algorithms of diagnostics, treatment of these diseases and SH diagnostic test systems permitted for use.

## **The Viral hepatitis with fecal-oral mechanism of transmission**

### **Actuality of theme**

Among the infectious diseases of man special place viral hepatitis. In 1973, the WHO agreed on the need of separate registration serum hepatitis and fecal-oral, and diseases that they cause, called hepatitis B and hepatitis A (HAV). Earlier have seen mainly in children, as was part of the group of so-called "children's" disease. The children observed mainly mild illness without jaundice symptoms in 75-80% of all cases of the disease. But lately there is a significant increase in the incidence of HAV among adults with more severe forms of domination, accompanied by jaundice, long disease and generally more severe course. The incidence of HEV recorded mainly in regions with hot climate and poor sanitation habits - is, first of all, Asia, Africa, South America, where HEV is the cause of 50% of all cases of acute viral hepatitis. Ill mostly men between 15 and 40 years. In cases of Hepatitis E (HEV) pregnant mortality reaches 20-40%.

The lack of cross-immunity allows a person to get sick more than once in their lifetime SH different etiologies.

**Viral hepatitis** - a group of infectious diseases in humans, characterized by a primary lesion of the liver. Diseases have similar clinical picture, but differ in etiology, epidemiology, pathogenesis, outcomes.

### **Viral hepatitis A**

Etiology: Viral hepatitis A (picornavirus family).

Epidemiology: the source of infection - with all forms of acute infectious process (of particular importance anicteric patients and asymptomatic forms). The mechanism of transmission: fecal-oral, infection occurs when consuming contaminated food and water, sometimes by contact-household. The greatest susceptibility to hepatitis A is typical for children.

Pathogenesis: Viral hepatitis A is introduced into the human body through the lining of the gastrointestinal tract, multiplies in the endothelium of the small intestine, mesenteric lymph nodes, then haematogenously enters the liver, where penetrates retikulogistiotsitarnye Kupffer cells in the liver parenchymal cells (hepatocytes) and damages them. In the future, the pathogen enters the bile into the intestine and excreted in the feces of the patient.

Clinic:

A) The minimum incubation period of 7 days, the maximum - 50 days, more usually 15 to 30 days.

B) initial (preicteric) period - 4-7 days, several variants of the clinic:

1) flu - a disease begins acutely, the body temperature rises rapidly to 38-39 ° C, often with chills, and kept on these figures 2-3 days. Patients concerned about a headache, aching muscles and joints, and sometimes a little runny nose, pain in the oropharynx. Smokers reduced or no desire to smoke. Asthenic and dyspeptic symptoms are mild.

2) dyspeptic - characterized by a decrease or disappearance of appetite, pain and heaviness in the epigastrium or right upper quadrant, nausea and vomiting, sometimes - increased stool 2-5 times a day.

3) asthenovegetative - disease begins gradually, the body temperature remains normal. Predominant weakness, reduced working capacity, there is irritability, drowsiness, headache, dizziness.

4) Mixed - features several syndromes.

On palpation of the abdomen marked increase, compaction and increase the sensitivity of the liver, and often enlarged spleen. For 2-3 days before onset of jaundice of the sclera and skin, patients notice that they darkened urine (acquired a dark brown color), and bowel movements, by contrast, have become lighter.

C) the icteric period - manifested yellowness of the sclera, mucous membranes of the oropharynx, and then the skin. The intensity of jaundice increases rapidly and in the coming weeks reaches its maximum. The color of urine becomes darker stool - discolored. With the emergence of a number of symptoms of jaundice

predzheltushnogo period weakens and a substantial portion of patients disappears (the longest preserved general weakness and loss of appetite, sometimes - a feeling of heaviness in the right upper quadrant). The body temperature is normal.

Revealed an increase, compaction and increased sensitivity of the edge of the liver, positive symptom Other. Characteristically slowing of the pulse. Blood pressure is normal or slightly reduced. The first heart sound at the apex is weakened. In the blood, elevated levels of total bilirubin, mainly due to the direct (related) increases sharply transaminases, especially alanine aminotransferase (ALT), increased performance thymol, reduced prothrombin index. Characterized hematological changes: leukopenia, neutropenia, and relative lymph monocytosis, normal or slow sedimentation rate. When serological tests determined the blood of anti-HAV IgM.

D) the period of convalescence - general condition improves, weaker signs of pigment metabolism, comes "pigment crisis." Reduced yellowness of the skin and mucous membranes, brightens urine, bowel movements become normal color, there is a clear trend towards normalization of biochemical parameters (bilirubin and prothrombin).

Bilirubinemia at the GA often less than 100 mmol / l. Marked reduction in blood bilirubin level occurs most often in the 2nd week of jaundice, while falls aminotransferases, and by 20-25 th day of the onset of jaundice, these figures usually reach standards.

By severity distinguished:

a) Easily - mild symptoms of intoxication, or their absence, low severity of jaundice. Bilirubinemia less than 100 mmol / L, and prothrombin index is over 60%.

b) moderately - intoxication symptoms (anorexia, fatigue, insomnia, nausea, vomiting, etc.), a moderate increase in liver. Bilirubinemia from 100 to 200 mmol / l, prothrombin index - from 50 to 60%.

c) heavy - pronounced symptoms of intoxication (progressive weakness, drowsiness, dizziness, anorexia until aversion to food, repeated vomiting, bright yellow skin, hemorrhagic syndrome, etc.). Bilirubinemia exceeds 200 mmol / l, prothrombin index is less than 50%.

On the severity of the isolated form of the HAV:

a) light - a general satisfactory condition at the height of the disease, the rapid disappearance of the yellow skin (2-3 weeks), rapid normalization of ALT (for 1 month.).

b) Moderate - moderate severity of the patient during the height of the disease, duration of jaundice of the skin up to 3-4 weeks, increased ALT to 1.5 months.

c) severe - in the midst of heavy disease state of the patient, duration of jaundice than 4 weeks, increased ALT activity - more than 1.5 months.

d) fulminant (lightning) - fast, in a matter of hours - days, the development of acute hepatic encephalopathy. In most patients, injection site haemorrhage, epistaxis, vomiting contents like "coffee grounds". High rates of aminotransferase activity, while AST ALT dominates. The development of the coma is fatal.

Cyclic observed within 90-95% of cases, 5% or more infectious process becomes wavy character as one or two exacerbations (usually within 1-3 months. From the beginning of the disease, sometimes later.) Exacerbations occur enhancement features for the height of hepatitis. In this general condition deteriorates again after improvement, appetite disappears, amplified discomfort in the liver, dark urine, discolored stools, jaundice increases the intensity of the skin, increased transaminases. When the GA, even with prolonged convalescence phase, the disease usually ends in complete recovery.

Diagnosis: epidemiological anamnesis (stay in the hearth of the GA for 15-40 days before the disease), acute onset, short initial period (usually by flu-like type), diarrheal manifestations (anorexia, nausea, vomiting, abdominal discomfort) with 3-5- day of illness, the rapid development of jaundice, the KLA (characteristic lymphocytosis, slowing ESR), BAC (early and prolonged elevation of transaminase activity still in the incubation period), a positive qualitative reaction of urine for urobilin and bile pigments, ELISA (detection of serum anti-HAV IgM during the first 2-3 weeks of illness and / or a fourfold and a more pronounced increase in the titer of anti-HAV IgG, taken in the icteric period of illness and convalescence period).



a) RNA virus (HAV RNA) - is detected in blood serum, feces, water and food by molecular hybridization PCR. Detection of HAV RNA in serum correlates with the maximum level of antiviral antibodies of class IgM; in faeces indicates the "infectivity" of the patient; in water and food - their viruses.

b) virus antigen (HAV Ag) - ELISA detected in the feces of a patient within 10-20 days after infection (early in the crisis period - only 20-50%)

c) antibodies to the class IgM (antiHAV IgM) - identified by enzyme immunoassay and radioimmunoassay techniques in serum / plasma levels early in the disease, regardless of the presence or absence of clinical symptoms. Antibody titer increases rapidly; maximum concentration is maintained for 1.5-6 months. 1 year after the recovery of the antibody in the blood are not detected. Detection of IgM antibodies indicates acute disease and allows serological differentiation of acute infection (presence antiHAV IgM and antiHAV IgG) and transferred to the medical history (no antiHAV IgM and presence antiHAV IgG).

d) an antibody to a virus class IgG (antiHAV IgG) - detected by ELISA in the serum / plasma levels early in the disease; their concentration reaches a maximum within a few weeks. Lack antiHAV IgG during the height of hepatitis avoids communication with hepatitis A virus; availability antiHAV IgG in the absence antiHAV IgM indicates the fact of past hepatitis A. After recovery antiHAV IgG detected in the blood for life and are considered as an indicator of immune protection against the virus. Level antiHAV IgG may be quantified to assess the dynamics of the immune response postvaccination vaccination against hepatitis A.

#### Treatment:

1) under light and moderate forms - polupostelny regime under heavy - bed; adherence obshchegigienicheskikh rules, including oral hygiene and skin; itching of the skin - wiping it with a solution of edible vinegar (1: 2), a 1% solution of menthol alcohol, hot showers at night.

2) control of the daily fluid balance, regular bowel movements - constipation laxatives herbal magnesium sulfate.

3) Therapeutic diets №5, food in the form of heat, food fractional without extractives.

4) when the GA benign course antivirals are not shown.

5) in the absence of pigment crisis within a week from the beginning of the crisis period - chelators (Polyphepanum, bilignin, coal granular sorbents type SKN-P, KAU, SUGS et al.).

6) enzyme preparations to strengthen the digestive function of the stomach and pancreas (pancreatin, Creon, likreaza, mezim fort pantsitrat, festal, enzistal, panzinorm, yunienzaym, zimopleks, Pankreoflat, abomin et al.)

7) infusion-detoxification therapy (5% glucose solution, gemodez).

8) with a deterioration of the patient on the background ongoing pathogenetic therapy - oral and parenteral corticosteroids, and in their inefficiency within 2-3 days - extracorporeal detoxification (hemisorbtion, plasmapheresis with partial plazmoobmenom, plasmadsorption, ultrafiltration).

9) with edematous ascitic syndrome: concentrated (10-20%) solutions of albumin, plasma; restriction of sodium chloride and 5 g / day; kalisodergaszczycie solutions; aldosterone antagonists (veroshpiron) triampur, with no effect - furosemide 40 mg / day 2-4 times a week

10) at 1-3 months hepatoprotectors .: derivatives silymarin (legal, Kars leprotek, silegon, Silimar, siromin), preparations of plant extracts (gepaliv, gepatofalk, gepabene) essentielle, Riboxinum, potassium orotate.

11) immunotherapy: drugs thymus (timalin, timogen, taktivin, leukinferon), interleukin-2 (roncoleukin).

12) phenobarbital (if prolonged postgepatitnoy hyperbilirubinemia)

**Hepatitis E** - called HEV RNA-containing viruses.

Epidemiology: the mechanism of infection - enteral (fecal-oral).

Pathogenesis: HEV has a cytopathic effect.

Clinical picture: an incubation period of about 35 days. The clinic is the initial period (5-6 days) predominate characteristics of an HA, however feverish reaction is

not expressed. Increasingly concerned about the general weakness, loss of appetite, nausea, aching pain in the right upper quadrant and epigastric. With the advent of jaundice (icteric period of 2-3 weeks.) Syndrome general intoxication is not reduced.

Women in the second half of pregnancy often occurs on malignant fulminant type with the rapid development of massive hepatic necrosis and acute hepatic encephalopathy, DIC, arrester.

Diagnosis: history (water transfer mechanism), clinic (CAA signs with easier flow), detection of anti-HEV IgM (anti-HEV IgG appear much later and show previous diseases).

a) RNA virus (HEV RNA) - appears in the blood in 2-3 weeks after infection. Viremia indicative of the fact of infection and lasts about 2 weeks.

b) antibodies to class M (antiHEV IgM) - appear in the blood after 3-4 weeks after infection and disappear after a few months. Is an indicator of the acute phase of the disease.

c) antibodies to class G (antiHEV IgG) - dominate the stage of recovery with an increase in titer to high values for several months. As in the case of hepatitis A, the presence of only antiHEV IgG does not confirm a diagnosis of hepatitis E virus; diagnosis can be made with simultaneous detection antiHEV IgM.

Complications of hepatitis:

1) cholestasis syndrome - occurs when an atypical course of the CAA, is characterized by persistent cholestatic jaundice and itching.

2) massive liver necrosis (fulminant hepatitis, viral hepatitis fulminantnaya form) - is more common in hepatitis B, D and E, is usually the first sign - hepatic encephalopathy, often with the development of deep coma. Characterized by a rapid decrease in the liver, increase in bilirubin, stupor, disorientation, lethargy, ascites, edema. Often develop cerebral edema; in the terminal phase of the disease may be compression of the brain stem, gastrointestinal bleeding, sepsis, respiratory failure, shock and surge arresters.

3) pancreatitis, myocarditis, SARS, aplastic anemia, neuropathy and transverse myelitis.

4) acute liver failure (encephalopathy)

Treatment: SAME WITH HAV.

Discharge criteria in viral hepatitis:

a) The absence of complaints, jaundice, liver decrease to normal size or a clear downward trend

b) the absence of bile pigments in the urine, normal levels of bilirubin in the blood.

Allowed extract with increasing aminotransferase (2-3 fold) increase in hepatic or 1-2 cm.

After discharge all been ill subject to mandatory health examinations.

## 1. Study purpose of practical studies:

### 2.1. The student must have an idea (read):

**$\alpha$  -1**

Prevalence of acute viral hepatitis in Ukraine

- The consequences of acute viral hepatitis
- Differential diagnosis of acute viral hepatitis.

### 2.2. Student have to know:

**$\alpha$  -2**

- Concept of manifest and latent forms, carrier, persistence, recurrence and exacerbation

- Clinical classification of viral hepatitis

- Definition of "hepatitis", "fulminant form of viral hepatitis"

Current views on the etiology and pathogenesis of viral hepatitis major

- Especially the clinical symptoms and course options for acute viral hepatitis

- The frequency and nature of complications, the prognosis of various viral hepatitis

- Principles of clinical diagnosis of major viral hepatitis.

Criteria for severity of viral hepatitis

- Clinical course of viral hepatitis in the case of co- and superinfection

- The nature of biochemical changes in the blood during acute viral hepatitis

- Present a specific diagnosis of acute viral hepatitis

- Diagnostic value of ultrasound examination in acute viral hepatitis
- Principles of treatment of acute viral hepatitis
- Clinical and laboratory manifestations of acute hepatic encephalopathy
- Indications for glucocorticosteroid therapy in acute viral hepatitis
- Rules of hospital discharge of patients with acute viral hepatitis

### **2.3. Student have to be able:**

**$\alpha$  -3**

1. Observe the basic rules of personal prevention of viral hepatitis critical when working at the bedside, in outpatient admission;
2. To take the history of the disease with the evaluation of epidemiological data.
3. inspect the patient and identify the main symptoms, especially pathological manifestations and timing of their disappearance.
4. Formulate a preliminary diagnosis according to existing classifications to justify it according to the epidemiological anamnesis, history of the disease, objective review;
5. Make a plan of examination and treatment depending on the type of pathology and clinical forms, peculiarities and difficulties encountered, premorbid background
6. Assign the necessary laboratory and instrumental examination, give an interpretation of the results obtained to justify the final clinical diagnosis according to the clinical classification;
7. Conduct a differential diagnosis with other related infectious diseases;
8. To make individualized therapy based on etiology, pathogenesis, patient's age, the severity, the disease and the presence of emergency conditions.
9. Give recommendations on oversight regime and diet nutrition during convalescence.

### **2.4. Educational goals (goals of the person):**

To develop the creative abilities of students in the course of clinical trials, analysis of scientific sources, to involve in student scientific circle of the department; suggest topics for essays on the most pressing issues.

- 2. Materials for out-class self-training (before practical classes)**
1. To form an idea of the basics of psychotherapeutic approaches to patients with acute viral hepatitis.
  2. To develop understanding of the impact of hygiene and social factors on human health in acute viral hepatitis.
  3. On the topic material to develop a sense of responsibility for the timeliness and appropriateness of professional action.
  4. Be able to set individual psychological contact with the patient and his relatives.

**2.1. Basic knowledge, skills which are necessary for studying of topic (interdisciplinary integration)**

<b>Discipline</b>	<b>To know:</b>	<b>To be able to:</b>
Microbiology	Etiology of acute viral hepatitis with parenteral mechanism of transmission	Performance of virological investigations and analysis of polymerase chain reaction data
Anatomy, Pathologic Anatomy and Histology	Liver structure, histological changes in liver affected by hepatitis B, C and D viruses.	Identification of histological hepatic changes (microscopic investigation)
Epidemiology	Ways of transmission and infection mechanism in acute viral hepatitis B, C and D.	Anti-epidemic and preventive measures in the effective disease area.
Immunology and Allergology	Liver affection in infection-allergic and cytopathogenic mechanisms. Serological examination procedure.	Analysis of immunologic investigation data
Nervous Diseases	Semiotics of nervous system damage in acute hepatic	Identification of the following clinical symptoms:

	<p>encephalopathy:</p> <ul style="list-style-type: none"> <li>• general cerebral symptom</li> <li>• focal symptom</li> <li>• hypertensive symptom</li> <li>• meningeal symptom</li> </ul>	<ul style="list-style-type: none"> <li>• general cerebral</li> <li>• focal</li> <li>• hypertensive</li> <li>• meningeal</li> </ul>
Introductory Course of Childhood Diseases	Basic stages and methods of patient clinical examination	Analysis of patient clinical examination data, including laboratory test data.
Pharmacology	<p>Basic medicines of the following pharmacological groups:</p> <ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antihistaminic</li> </ul> <p>Age dosage of medicines.</p>	<p>Writing out of a prescription for basic medicines of the following pharmacological groups:</p> <ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antihistaminic</li> </ul>
Resuscitation and Intensive Care	<p>Urgent conditions:</p> <p>- acute hepatic encephalopathy</p>	Urgent medical care for acute hepatic encephalopathy.
Other courses		
Family Medicine	<p>Pathogenesis, epidemiology, dynamics of clinical manifestations and possible complications of viral hepatitis. Principles of prevention and treatment.</p>	<p>Differential diagnostics of other diseases of various geneses versus viral hepatitis. Identification of viral hepatitis with parenteral mechanism of transmission and its possible complications; interpretation of</p>

		laboratory examination data. Timely hospitalization of patient in the infectious inpatient department. Completion of an emergency report. Provision of urgent medical care, if necessary
<b>Intracourse integration</b>		
<b>Infectious Diseases</b>	Special characteristics of infectious diseases. Principles of diagnostics, treatment and prevention of infectious diseases. Pathogenesis, epidemiology, dynamics of clinical manifestations, laboratory diagnostics, possible complications and clinical course peculiarities of viral hepatitis. Principles of prevention and treatment.	Differential diagnostics of viral hepatitis with parenteral mechanism of transmission versus other diseases of various geneses. Identification of viral hepatitis and its possible complications; interpretation of laboratory examination data. Prescribe treatment.

### 3.4. Self-control materials

#### 3.4.1. Questions to be answered

**α -1**

1. Source of infection and route of infection in acute viral hepatitis.
2. Classification of viral hepatitis.
3. Risk groups for acute viral hepatitis.



4. The main clinical forms of acute viral hepatitis.
5. The clinical course of acute viral hepatitis preicteric period.
6. The main clinical features of acute viral hepatitis jaundice period.
7. Variants of the course of acute viral hepatitis D. Clinical manifestations.
8. Complications and consequences of acute viral hepatitis.
9. Criteria for severity of acute viral hepatitis.
10. Ultrasound diagnosis of acute viral hepatitis.
11. The nature of the laboratory changes in acute viral hepatitis.
12. The specific diagnosis of acute viral hepatitis.
13. The main stages of the pathogenesis of acute hepatic encephalopathy.
14. The main clinical manifestations of acute hepatic encephalopathy.
15. The nature of the laboratory changes in acute hepatic encephalopathy.
16. The principles of treatment of acute viral hepatitis.
17. The principles of dietary nutrition in acute viral hepatitis.
18. The principles of treatment of acute hepatic encephalopathy.
19. Non-specific and specific prophylaxis of acute viral hepatitis.
20. Rules of hospital discharge of patients with viral hepatitis.

#### **3.4.2. Self-control tests**

**$\alpha$  -2**

#### **3.4.3. Situational tasks of the second level learning**

**$\alpha$  -3**

##### **Task 1**

Patient '17 came to the clinic on the 6th day of illness. Acutely sick with fever up to 39.0°C, weakness, on the 2nd day of illness decreased appetite, temperature returned within 4 days, and then decreased to normal after 5 days of illness - darkened urine  
 Ob-no: T 36.8°C, skin and mucous membranes subicteric without rash. Abdominal palpation soft and painless, enlarged liver and spleen not palpable. Heart rate - 64 beats / min. Blood pressure - 110/70 mm

1. Preliminary diagnosis.
2. Plan examination.
3. Treatment

##### **Task 2**

The patient was 27 years old student living in a dormitory. Entered the hospital on the 5th day of illness with complaints yellowing of sclera and skin, weakness, lack of appetite. The disease began acutely with a rise in temperature to 38.0, catarrhal syndrome was not. Drugs tried, dorm were cases of hepatitis A.

Ob-no: T - 36,3oS, a small weakness. Mild jaundice of the skin and sclera, no rash. The abdomen was soft and painless in all departments, the liver is enlarged, painless, spleen palpable at the costal arch. Pulse - 64 beats / min., Blood pressure - 110/70 mm In the blood, marked leukopenia, relative lymphocytosis, SHZE - 5 mm / h.

1. Preliminary diagnosis.
2. Plan examination.
3. Treatment

### **Task 3**

Patient N., 23 years old, admitted to hospital on the 8th day of illness with complaints of worsening overall health, the severity of abdominal pain, especially after eating, appetite loss. Disease acute: fever, temperature 38.50, headache, pain when turning eyeballs. On the 3rd day of illness the temperature returned to normal, but health worsened: decreased appetite, became acutely feel the cooking smells, felt immediately from tobacco smoke. 2 days ago noticed dark urine, feeling much improved. 3 months. back - removal of tooth. 7-year-old son with similar complaints of days hospitalized in pediatric infectious disease clinic. Vacationing with your family (she, her husband and 2 children 7 and 11 years) in camping in Crimea, 3 weeks ago.

1. Preliminary diagnosis.
2. Plan examination.
3. Treatment

## **4. Materials for the class of independent work**

### **4.1. List of study practical tasks to be performed in the practice:**

1. Learn the basic rules work at the bedside .
2. Take the history of the disease with the evaluation of epidemiological data.
3. To provide curation of patient and identify symptoms and syndromes of cholera, dehydration shock, boulders based on their clinical stages substantiate the clinical diagnosis for timely referral of the patient to the hospital .
4. Making medical records of suspected cholera, dehydration shock.
5. Based on clinical examination time to recognize possible complications cholera.
6. Making medical documentation at establishing a preliminary diagnosis cholera.
7. Make a plan and additional laboratory examination of the patient.
8. Interpret the results of laboratory testing.
9. Right, depending on the material and the term survey to assess the results of specific diagnostic methods. To make a treatment plan based on epidemiological data , stage of disease , presence of complications, severity of condition, allergist anamnesis, concomitant pathology, able to provide immediate assistance.
10. Make a plan and emergency for the prevention of the source of infection .
11. Give recommendations on treatment , diet, inspection , supervision in the recovery period.
12. To carry out differential diagnostics of a dehydrationous shock, an enterorrhagia.
13. To make the plan of laboratory examination.
14. To interpret results of specific patient examination with an enterorrhagia, and dehydrationous shock
15. To define medical tactics in the case urgent conditions origin.
16. To issue the medical documentation.

#### **4.2 Professional algorithm for diagnostics skills and ability formation**

№	Task	Execution sequence	Annotation, notices for self-checking

<p><b>1</b></p>	<p>To seize a technique of cholera patient clinical examination, a patient with a dehydration shock</p>	<p><b>I</b> To find out the patient complaints.</p> <p><b>II</b> To find out an anamnesis:</p> <p><b>1.</b> Medical history</p> <p><b>2.</b> Patient history.</p> <p><b>3.</b> Epidanamnesis.</p>	<p>To separate complaints that characterised cholera, dehydration syndrome.</p> <p>To pay attention to:</p> <p>Origin succession, symptoms dynamic peculiar for a dehydrationous shock, an enterorrhagia.</p> <p>To find out commemorative diseases.</p> <p>To find out the data concerning fecal-oral transfer route realisation, to pay attention for patient's stay in the high risk cholera infection regions.</p>
<p><b>2</b></p>	<p>To carry out a patient curation</p>	<p><b>II</b> To carry out an objective examination.</p>	<p>Remember: presence, evidence, symptoms dynamic, provided with term and severity</p>

		<p><b>1. Common examination:</b></p> <ul style="list-style-type: none"> <li>- patient general condition;</li> <li>- skin, fauces mucous tunic</li> </ul> <p><b>2. Digestive system:</b></p> <ul style="list-style-type: none"> <li>- tongue examination;</li> <li>- abdomen percussion;</li> <li>- abdomen palpation;</li> <li>- excrements characteristic.</li> </ul> <p><b>3. Cardiovascular system:</b></p>	<p>disease course, depend on patient age, accompanying pathology.</p> <p>To pay attention for:</p> <ul style="list-style-type: none"> <li>- slackness, adynamy, the patient block;</li> <li>- a body temperature;</li> <li>- the skincyanosis, acute lowered turgor.</li> </ul> <p>To pay attention for:</p> <ul style="list-style-type: none"> <li>- tongue is furred with white incrustation;</li> <li>- dry tongue.</li> <li>- Peritonitis symptoms (presence testifies to punching of a small intestine).</li> <li>- quantity, character, propensity to constipation (occurrence melena testifies to an enterorrhagia).</li> </ul> <p>To pay attention for:</p>
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		<ul style="list-style-type: none"> <li>- pulse;</li> <li>- blood pressure;</li> <li>- heart auscultation</li> </ul>	<ul style="list-style-type: none"> <li>- a tachycardia;</li> <li>- moderately lowered blood pressure (considerable decrease testifies to complications);</li> <li>- moderate dullness of heart sounds</li> </ul>
3	To set laboratory and additional researches, to interpret the results	<ol style="list-style-type: none"> <li>1. Complete blood count</li> <li>2. Clinical urine analysis</li> </ol>	<p>To pay attention of typical changes:</p> <ul style="list-style-type: none"> <li>- leukopenia, or leukocytosis, lymphocytosis, aneosinophilia, thrombocytopenia (leukocytosis, anemia, ESR, appear in case of development enterorrhagia).</li> </ul> <p>The absence of significant changes in typical course</p>

### **4.3. Materials methods of the final phase of occupation**

#### **4.3.1. Objectives of the 3rd level**

***α* -3**

#### **Task 1**

B. 19 years old, a kindergarten teacher, complains of general weakness, loss of appetite, nausea, epigastric discomfort, dark urine, feces ahoic, jaundice. Sick 7 days ago. The garden had contact with children who suffered from jaundice. Objectively: skin and mucous membranes ikterict, the liver is increased by 2-3 cm, palpable spleen.

1. To define the diagnosis.
2. To make the plan of patient examination.
3. To make the treatment plan.

### **The right answers**

#### **Task 1**

1. Hepatitis A, acute icteric form

2. - general blood analysis with thrombocytes; RW; hepatic tests: general and conjugated bilirubin, lAT, AlAT in dilution (1 : 10), thymol turbidity test, alkaline phosphatase, GGT; cholesterin;-proteinogram; blood sugar; amylase; coagulogram; rest nitrogen, creatinine; markers of viral hepatitis: B, C, D et al. PCR; urine analysis for bile pigments; ultrasound examination of hepatobiliary system.

3. 1) under light and moderate forms - polupostelny regime under heavy - bed; adherence obschegigienicheskikh rules, including oral hygiene and skin; itching of the skin - wiping it with a solution of edible vinegar (1: 2), a 1% solution of menthol alcohol, hot showers at night.

2) control of the daily fluid balance, regular bowel movements - constipation laxatives herbal magnesium sulfate.

3) Therapeutic diets №5, food in the form of heat, food fractional without extractives.

4) when the GA benign course antivirals are not shown.

5) in the absence of pigment crisis within a week from the beginning of the crisis period - chelators (Polyphepanum, bilignin, coal granular sorbents type SKN-P, KAU, SUGS et al.).

6) enzyme preparations to strengthen the digestive function of the stomach and pancreas (pancreatin, Creon, likreaza, mezim fort pantsitrat, festal, enzistal, panzinorm, yunienzaym, zimopleks, Pankreoflat, abomin et al.)

7) infusion-detoxification therapy (5% glucose solution, gemodez).

8) with a deterioration of the patient on the background ongoing pathogenetic therapy - oral and parenteral corticosteroids, and in their inefficiency within 2-3 days - extracorporeal detoxification (hemisorbtion, plasmapheresis with partial plazmoobmenom, plasmatorption, ultrafiltration).

9) with edematous ascitic syndrome: concentrated (10-20%) solutions of albumin, plasma; restriction of sodium chloride and 5 g / day; kalisodergaszczycze solutions; aldosterone antagonists (veroshpiron) triampur, with no effect - furosemide 40 mg / day 2-4 times a week

10) at 1-3 months hepatoprotectors .: derivatives silymarin (legal, Kars leprotek, silegon, Silimar, siromin), preparations of plant extracts (gepaliv, gepatofalk, gepabene) essentielle, Riboxinum, potassium orotate.

11) immunotherapy: drugs thymus (timalin, timogen, taktivin, leukinferon), interleukin-2 (roncoleukin).

12) phenobarbital (if prolonged postgepatitnoy hyperbilirubinemia)

## **5. Materials of after-work**

**$\alpha$  -4**

Proposed topics for essays on the most pressing issues, such as:

Peculiarities of hepatitis E.

Modern methods of specific diagnosis of hepatitis A.

Problems etiotropic treatment of typhoid fever today.

Specific prevention of hepatitis A.

### **The Viral hepatitis with parenteral mechanism of transmission**

**Urgency of the issue:** Diagnostics and treatment of viral hepatitis is one of the most difficult issues of current medicine. The urgency of this issue is related to wide



occurrence of the disease in various forms, similarity of clinical and laboratory data, difficulties in treatment, and large number of complications and adverse consequences. Viral hepatitis is the most frequent cause of infectious jaundice. Viral B hepatitis is still a significant problem for healthcare system all over the world. According to estimates of the WHO experts, about 400 million people have been infected by the agent of this disease. Annually, 1.5 to 2 million patients die throughout the world due to hepatic diseases induced by hepatitis B virus, including 500,000 to 600,000 persons dying due to hepatocellular carcinoma (5<sup>th</sup> position among the causes of tumor-induced lethality). Consequences of HBV infections are ranked tenth among all other death causes in the world. In Ukraine, about 1 million persons are carriers of this virus.

At the same time, the growth and proliferation of infections caused by HCV and also the extremely frequent occurrence of its negative consequences determine the urgency of studying pathogenesis, clinical forms, diagnostics and treatment of this disease. Not less than one third of persons infected by HCV also suffer from chronic viral hepatitis (CVH), liver cirrhosis (LC) and hepatocellular carcinoma (HC). It is HCV that is considered to be the most frequent factor provoking CVH and may result, depending on its particular sub-type, in chronicity of the disease in 60% to 90% of cases. According to different estimates, 400 to 700 million people on the planet are infected by this agent, including not less than 300 million people having signs of chronic liver lesion, and more than 10 million of them die annually. If we fail to take urgent measures, then in 10 to 20 years the rate of mortality due to HCV infection and its consequences would increase three times and would be significantly higher if accompanied by HIV infection. During this period, according to WHO forecasts, chronic hepatitis C would become the main problem of the national healthcare systems all over the world.

Not only infection disease doctors examine and provide treatment to patients with viral hepatitis, but also district doctors, family physicians and emergency doctors. Taking into account the highly wide occurrence of viral hepatitis and possibility of complicated clinical course or lethal outcome of the disease, it is

important that every physician of any specialization can diagnose, provide treatment for and prevent progression of the disease.

**Viral hepatitis B.** Etiology: Hepatitis B virus (HBV) - Marketing hepadnavirus DNA. Viral envelope protein contains the surface antigen (HBsAg) - «Australian" AH contains a nucleocapsid core (core) antigen (HBcAg), infectious (HBeAg), HBxAg, DNA and enzymes (polymerase, and protein kinase). Each of the HBV antigen elicits a humoral immune response, manifested generation of appropriate antibodies (anti-HBs, anti-HBc, anti-HBe).

Epidemiology: source - patients as symptomatic and asymptomatic forms of acute and chronic HB. Mechanism of transmission: parenteral; pathways: natural (from mother to child - vertical and perinatal, sexual contact with an infected person - sexual, with other contacts with an infected person - horizontal) and artificial (in violation of the integrity of skin and mucous membranes).

Pathogenesis: The virus is not peculiar to the direct damaging effect on hepatocytes, their cytolysis carried immunomediated by reaction on the part of cellular immunity through cytotoxic T- lymphocytes.

Clinic: a) The incubation period of 42 to 180 days, with an average of 60-120 days.

b) initial (preicteric) period - 7-14 days; disease begins with symptoms of mixed options before jaundice period without a significant increase in body temperature. Symptoms of intoxication and dyspeptic manifestations are moderate, a third of patients arthralgic version of the initial period (increased pain in the large joints at night and in the morning). The initial period of illness lasts for 7-14 days or more, but infections associated with blood transfusions, it may be shorter.

c) the icteric period - 3-4 weeks, more severe and prolonged pain, sometimes sharp pain in the right upper quadrant. Continued weakness, loss of appetite comes to anorexia. Frequent nausea and vomiting even. Often itchy skin. The liver is always enlarged, palpation is smooth, with a few compacted consistency, there is enlargement of the spleen. In peripheral blood leukopenia with lymphomas and monocytosis, sometimes - with the plasma reaction. ESR is reduced to 2-4 mm / h, during the convalescence period can be accelerated to 18-24 mm / h, followed by

normalization in the absence of complications. Hyperbilirubinemia more pronounced and persistent than hepatitis A, especially for 2-3 weeks jaundice period; there is an increase of transaminases in blood serum in reducing sublimate test and prothrombin index. When serological tests of blood found HBsAg, anti-HBc IgM.

In severe - signs of growing liver failure and progression of necrotic processes in the liver - increased general muscle weakness, dizziness, lethargy, anorexia, nausea, frequent vomiting, the appearance of unmotivated excitation, memory impairment; progressive increase in the icteric coloration of the skin; reducing the size of the liver, increased morbidity its edge; occurrence of hemorrhagic syndrome (petechial rash on the skin, nosebleeds, bleeding at the injection site, "tarry" stools, vomiting with blood); edematous appearance of ascitic syndrome (swelling of the feet and the lower third of shins, ascites); the occurrence of fever, tachycardia, leukocytosis; increase in total serum bilirubin at increase its indirect tailcoat-tion; reduction of cholesterol below 2.6 mmol / l.

g) the period of convalescence - normalization of aminotransferase activity by 30-35 th day of the disease in mild, with moderate - to 40-50 th, in severe - 60-65 th day.

Diagnosis: clinical history (blood transfusions, surgery, etc.), clinical (gradual onset, long preicteric period, allergic skin rash, lack of improvement or worsening of health with the appearance of jaundice, prolonged jaundice during the slow disappearance of symptoms in convalescence period).

a) Virus DNA (HBV DNA) - is detected in the serum / plasma by molecular hybridization, sometimes with the preceding stage polymerase chain reaction. Detection of HBV DNA means virusemicheskuyu stage of the process and shows a high replicative activity of the virus. In acute hepatitis B content of HBV DNA in the blood increases rapidly during the incubation period and becomes maximum at the beginning of the crisis period. The circulation of virus DNA over 5-6 months is a poor prognostic sign and is often indicative of chronic hepatitis B.

b) surface antigen (HBsAg, Hepatitis B from the virus surface antigen) - the earliest marker of HBV, which appears in the blood is still in the incubation period. HBsAg circulates in acute course of the disease up to 5-6 months. Detection of HBsAg in the

blood of more than 6 months may indicate the beginning of the chronic process. A positive result must be checked by repeating the test in two parallel samples. If at least one of the two samples of the serum / plasma at this again gives a positive result, the sample is considered positive and initially tested in a confirmatory test system based on the neutralization reaction. It must be borne in mind, as many immunoassay systems produced in Russia, having low specificity, capable of producing a large number of false positives.

A negative test result for HBsAg usually indicates the absence of hepatitis B in the subject, but the diagnosis can not be excluded completely, because infection may be latent or sensitivity of the test system is inadequate.

c) antibodies to the surface antigen (antiHBs) - an indicator of immunity to hepatitis B virus or the immune response to vaccination against hepatitis B. In the latter case, the titer antiHBs gradually increases, whereas antibodies to the core antigen of the virus are not formed. The level antiHBs 10 mIU / mL or higher indicates an adequate response to vaccination.

d) core antigen (HBcAg, from Hepatitis B virus core antigen) - a strong immunogen elicited antibodies (antiHBc), which appear in the blood within 1.5-2 months from the beginning of the crisis period and may circulate for years, often as the only marker of myocardial hepatitis B (especially in the step serological "window" between the disappearance of HBsAg and the appearance of antibodies thereto).

e) antibodies to the core antigen class IgM (antiHBc IgM) - their appearance in the blood is a confirmation of acute hepatitis B. Because the diagnosis usually made on the basis of the clinical picture, elevated alanine aminotransferase (ALT) serum and detection of HBsAg in blood diagnostic value especially antiHBc IgM large in the following cases:1) when specific diagnosis of hepatitis B for some reasons made later; with HBsAg in blood can already be absent;2) in acute hepatitis in patients with chronic hepatitis B. If a hepatitis associated with superinfection by other hepatotropic viruses, the characteristic is the absence or low titer antiHBc IgM.

f) antibodies to the core antigen of class IgG (antiHBc IgG) - are more important marker of past infection compared with antiHBs, since: antiHBs may occur as a result

of vaccination; antiHBc may be present in phase "seronegative window" when HBsAg is no longer present, antiHBs not yet appeared; antiHBs in acute hepatitis B do not develop in 15% of convalescents; within 6 years after acute hepatitis B antiHBs may disappear in 20% of recovered; in endemic areas of hepatitis B in 20% of the population can be detected only in the absence antiHBc other markers of infection.

g) "infectivity antigen" (HBeAg) - appears in the blood of patients with acute and chronic hepatitis is usually in the phase of viremia. The presence of HBeAg in the blood is a characteristic expression of his mature virus so-called wild-type (normal, the most common option). Finding him in acute hepatitis B allows to predict disease outcome, since HBeAg disappears from the circulation within 1.5-2 months from the beginning of her illness with a favorable course. Test for the presence of HBeAg is meaningful only HBsAg-positive sera, since its discovery in HBsAg-negative samples are rare and for the most part is a false positive result

h) antibodies' antigen infectivity »(antiHBe) - asymptomatic" carriers of »HBsAg and absence of viremia antiHBe can be considered as an additional indicator of the inactive wild-type virus infection.

Treatment:

1. Antiviral - alpha interferon (recombinant: Intron A, Roferon A, and native realdiron: vellferon, che lovechesky leukocyte interferon) with the threat of chronic or progradiently during synthetic nucleosides - famciclovir (Famvir), lamivudine (Epivir), zidovudine (Retrovir, AZT, timozid), protease inhibitors - saquinavir (Invirase), indinavir (Crixivan), interferon inducers - neovir (tsikloferon) amiksin, immunomodulators - leukinferon, interleukin-1 (Betaleukin), interleukin-2 (roncoleukin). Shown in severe hepatitis B (in the presence of markers of active viral replication) with the threat of acute liver failure, especially when the signs of hepatic encephalopathy, as well as the threat of chronicity.

2. When complications associated with encephalopathy: arresting agitation sodium oxybutyrate, seduk-sen; gastric lavage (nasogastric tube) and high or siphon enema to reduce autointoksi-cation; oral administration of poorly absorbed antibiotics

(kanamycin 0.5 g 4 times a day), enterosorbents; fractional enteral nutrition combined with parenteral; Glucocorticosteroids (at least 180-240 mg prednisolone) parenterally; in-fusional therapy; with hemorrhagic manifestations - protease inhibitors and fibrinolysis (epsilon-aminocaproic acid, contrycal, gordoks).

**Viral hepatitis C.** Disease similar on epidemiological grounds with HBV, but occurs more easily and at different icteric relatively rapid regression of the disease. More common anicteric, subclinical and inapparent forms TOS that are transferred without hospital treatment, but in 80-90% of cases become chronic hepatitis, and in 20-30% of patients - in cirrhosis.

Etiology: hepatitis C virus (HCV, HCV) family of flaviviruses RNA Marketing.

Epidemiology: source - patients with acute and chronic forms of the infection; mechanism of infection - parenteral (hepatitis drug addicts), less infection in the home, during sexual intercourse and childbirth from an infected mother.

Pathogenesis: the main difference between the biological properties of HBV HCV - the dominant role of the biological properties of the immune response, the virus has a direct cytopathic effect. The high variability of HCV allows to persist in the human body.

Clinic: The average incubation period - 40-50 days. Acute heavy usually remains unrecognized because the pathological process generally proceeds latent (subclinical, inapparent form) and can be diagnosed only by increase of ALT, positive test for RNA HCV, somewhat less anti-HCV IgM, IgG. In the initial period of anicteric and icteric forms characteristic asthenovegetative and dyspeptic syndromes, weakness, lethargy, fatigue, poor appetite, and sometimes a feeling of heaviness in the right upper quadrant. In icteric period signs of general intoxication insignificant manifestations of jaundice minimal (subikterichnost sclera and mucous membranes of the sky, light staining of the skin, and transient holuriya acholia) may be a slight increase in liver. Characteristically for easy and absence of acute liver failure.

Diagnosis:

a) RNA virus (HCV RNA) - detected by molecular hybridization in serum / plasma, white blood cells and liver puncture biopsy specimens. Detection of HCV RNA

confirms active viral infection. On the other hand, the quantification of HCV RNA is important for assessing the effectiveness of antiviral therapy. Identification of HCV RNA is the basis of the diagnosis of hepatitis C cases seronegative example, at an early stage of infection, when the virus antibodies have not yet developed, and in immunosuppressed patients (such as after organ transplantation), in which antibody response is suppressed.

b) antibodies to the virus (antiHCV) class IgG - AntiHCV indicate contact of the patient with the virus and are found both in acute and chronic hepatitis. In most cases, their presence is established by using enzyme immunoassay systems of the third generation, is correlated with the presence in the blood of HCV RNA. In the service of the blood of all the countries testing donated blood for antiHCV is mandatory. Identifying antiHCV in a blood sample is the undisputed basis for its rejection.

Treatment: The treatment principles - see. Hepatitis B, but should be administered antiviral therapy in all cases of acute HS, given the extremely high probability of chronicity.

**Viral hepatitis D** - by RNA-containing viruses that are able to replicate only in the presence of HBV incorporating into its outer shell.

Epidemiology: the sources and the transmission mechanism - as with HBV.

Pathogenesis: the main feature is the leading role of HDV compared with HBV, while active replication HDV often leads to the suppression of reproduction HBV.

GD has a direct cytopathic effect on hepatocytes.

Clinic: incubation period of 20-40 days; unlike HBV hepatitis delta different:

- 1) higher and more prolonged febrile reaction
- 2) the more frequent occurrence of polymorphic rash, joint pain
- 2) increase in spleen
- 4) two-wave course of the disease
- 5) more frequent forms of fulminant

In the blood reveal markers of acute phase: anti-HBc IgM and anti-HDV IgM. For the mixed hepatitis B + D is mainly characterized by cyclic moderate forms, culminating in recovery. When HDV / HBV-superinfection clinically symptomatic acute hepatitis

observed much less frequently than with coinfection, but it is often observed severe and fulminant forms with pronounced symptoms of intoxication, hemorrhagic, and often edematous-ascitic syndrome, pain in the right upper quadrant, with repeated waves of exacerbation, sometimes exceeding the severity first. In some cases there is a rise in transaminases without clinical manifestations.

Diagnosis: detection in blood anti-HDV IgM, HDV RNA along with HBsAg. In this case the absence of anti-HBc IgM, HBeAg (and conversely, the presence of anti-HBc IgG, anti-HBe) indicates HDV / HBV-superinfection. On the contrary, the presence of anti-HBc IgM and HBeAg characteristic of HDV / HBV-coinfected. HDAg serum rarely found, anti-HDV IgG appear later.

## **2. Learning objectives**

### **2.1. Student should have an idea of (gain an insight to):** **a-1**

- incidence of parenteral viral hepatitis in Ukraine;
- consequences of acute viral hepatitis B, C and D;
- differential diagnostics of parenterally transmitted acute viral hepatitis.

### **2.2. Student should know:** **a-2**

- overt and latent forms, carrier state, persistence, recurrence and acute condition;
- clinical classification of viral hepatitis;
- definitions of the following notions: ‘viral hepatitis’, ‘fulminant viral hepatitis’, ‘coinfection of hepatitis virus’, ‘viral hepatitis superinfection’, ‘acute viral hepatitis’, ‘lingering viral hepatitis’, and ‘chronic viral hepatitis’;
- current concept of etiology and pathogenesis of the most important types of viral hepatitis;
- special clinical symptoms and variants of clinical course of parenterally transmitted acute viral hepatitis;
- incidence and nature of complications and prognosis for various types of viral hepatitis;
- principles of clinical diagnostics of the most important types of viral hepatitis;



- basic criteria of the viral hepatitis severity grades;
- special characteristics of viral hepatitis clinical course in case of coinfection and superinfection;
- pattern of biochemical blood changes in acute viral hepatitis;
- modern specific diagnostics of acute viral hepatitis;
- diagnostic value of ultrasound examination in acute viral hepatitis;
- treatment principles for acute viral hepatitis;
- clinical and laboratory manifestations of acute hepatic encephalopathy;
- indications for glucocorticosteroid therapy in acute viral hepatitis;
- rules for discharging patients with acute viral hepatitis from hospital.

**2.3. Student should be able to:**

***a-3***

1. Comply with the basic personal prevention rules for the most important types of viral hepatitis, when attending patients in the hospital or performing outpatient examination;
2. Prepare case history and provide analysis of the available epidemiological data.
3. Examine a patient and identify main symptoms, peculiarities of pathologic manifestations and their possible duration.
4. Formulate a provisional diagnosis based on the existing classifications and substantiate it with reference to the available data of epidemiological anamnesis, case history and objective examination;
5. Make an examination and treatment plan depending on particular type of pathology, clinical form, peculiarities of clinical course and arising complications, and premorbid background;
6. Determine necessary laboratory and instrumental examination and give interpretation of the received findings for the purpose of substantiating final clinical diagnosis according to the existing clinical classification;
7. Perform differential diagnostics as compared to other similar infectious diseases;

8. Prescribe an individual therapy course taking into account the etiology, pathogenesis, patient's age, severity grade and duration of the disease, and acute conditions, if any.

9. Give recommendations as to care, nutrition regimen and diet during the period of convalescence.

### **3. Reference materials for pre-classroom independent activities:**

#### **3.1. Basic knowledge and skills required to master the subject.**

##### **Interdisciplinary integration**

Discipline	To know:	To be able to:
Microbiology	Etiology of acute viral hepatitis with parenteral mechanism of transmission	Performance of virological investigations and analysis of polymerase chain reaction data
Anatomy, Pathologic Anatomy and Histology	Liver structure, histological changes in liver affected by hepatitis B, C and D viruses.	Identification of histological hepatic changes (microscopic investigation)
Epidemiology	Ways of transmission and infection mechanism in acute viral hepatitis B, C and D.	Anti-epidemic and preventive measures in the effective disease area.
Immunology and Allergology	Liver affection in infection-allergic and cytopathogenic mechanisms. Serological examination procedure.	Analysis of immunologic investigation data
Nervous Diseases	Semiotics of nervous system damage in acute hepatic encephalopathy: <ul style="list-style-type: none"> <li>• general cerebral symptom</li> <li>• focal symptom</li> <li>• hypertensive symptom</li> <li>• meningeal symptom</li> </ul>	Identification of the following clinical symptoms: <ul style="list-style-type: none"> <li>• general cerebral</li> <li>• focal</li> <li>• hypertensive</li> <li>• meningeal</li> </ul>
Introductory Course of Childhood	Basic stages and methods of patient clinical examination	Analysis of patient clinical examination data, including laboratory test data.

Diseases		
Pharmacology	<p>Basic medicines of the following pharmacological groups:</p> <ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antihistaminic</li> </ul> <p>Age dosage of medicines.</p>	<p>Writing out of a prescription for basic medicines of the following pharmacological groups:</p> <ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antihistaminic</li> </ul>
Resuscitation and Intensive Care	<p>Urgent conditions:</p> <p>- acute hepatic encephalopathy</p>	<p>Urgent medical care for acute hepatic encephalopathy.</p>
Other courses		
Family Medicine	<p>Pathogenesis, epidemiology, dynamics of clinical manifestations and possible complications of viral hepatitis. Principles of prevention and treatment.</p>	<p>Differential diagnostics of other diseases of various genes versus viral hepatitis. Identification of viral hepatitis with parenteral mechanism of transmission and its possible complications; interpretation of laboratory examination data. Timely hospitalization of patient in the infectious inpatient department. Completion of an emergency report. Provision of urgent medical care, if necessary</p>
Intracourse integration		
Infectious Diseases	<p>Special characteristics of infectious diseases. Principles of diagnostics, treatment and prevention of infectious diseases. Pathogenesis, epidemiology, dynamics of clinical manifestations, laboratory diagnostics,</p>	<p>Differential diagnostics of viral hepatitis with parenteral mechanism of transmission versus other diseases of various genes. Identification of viral hepatitis and its possible complications; interpretation of laboratory examination data.</p>

	possible complications and clinical course peculiarities of viral hepatitis. Principles of prevention and treatment.	Prescribe treatment.
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3.2. Logic block diagram:

Acute viral hepatitis with parenteral

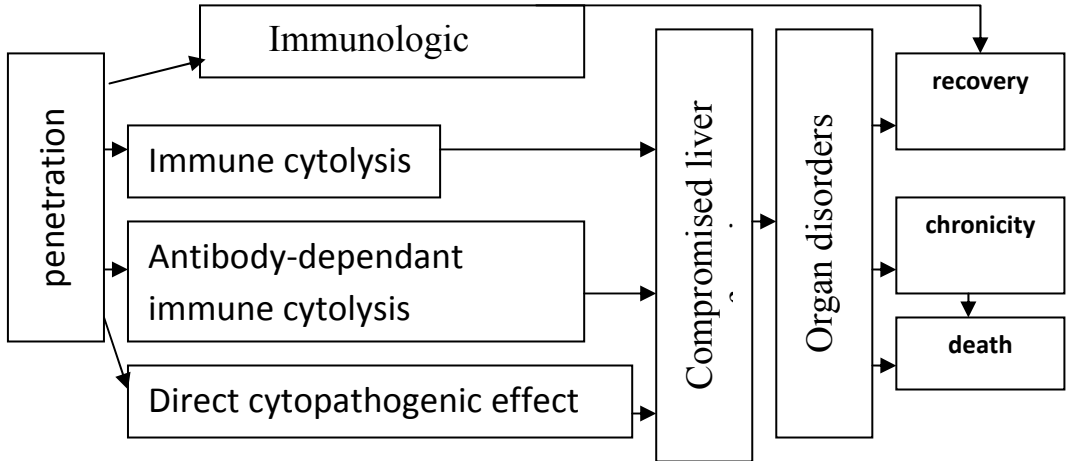
Ethiology

HBV, HCV, HDV

Epidemiology

Source of infection: sick persons and carriers.  
Ways of transmission: parenterally, sexually, vertically,

Pathogenesis



Clinical presentation

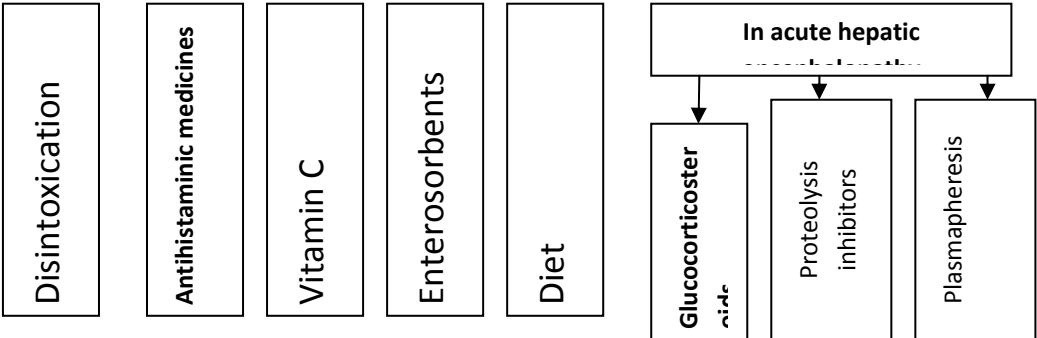
- Aasthenovegetative syndrome
- Dyspeptic syndrome
- Arthralgic syndrome
- Allergic syndrome
- Jaundice
- Hepatolienal syndrome
- Hemorrhagic syndrome

Specific diagnostics

Polymerase chain reaction

Enzyme immunoassay

Treatment



Prevention

Non-specific

Vaccination

### **3.4. Materials for self-control.**

#### **3.4.1 Questions for self-control**

$\alpha=2$

1. Source of infection and infection ways in acute viral hepatitis B, C and D.
2. Viral hepatitis classification.
3. Risk groups in acute viral hepatitis B, C and D.
4. Main clinical forms of acute viral hepatitis.
5. Variants of pre-jaundice period course in acute viral hepatitis B, C and D.
6. Basic clinical manifestations during jaundice period in acute viral hepatitis B, C and D.
7. Variants of acute viral hepatitis D. Clinical manifestations.
8. Complications and consequences of acute viral hepatitis B, C and D.
9. Viral hepatitis severity criteria.
10. Ultrasound diagnostics in acute viral hepatitis B, C and D.
11. Pattern of laboratory changes in acute viral hepatitis B, C and D.
12. Specific diagnostics of acute viral hepatitis B, C and D.
13. Basics stages of pathogenesis in acute hepatic encephalopathy.
14. Main clinical manifestations in acute hepatic encephalopathy.
15. Pattern of laboratory changes in acute hepatic encephalopathy.
16. Treatment principles for acute viral hepatitis B, C and D.
17. Dietary principles in acute viral hepatitis.
18. Treatment principles for acute hepatic encephalopathy.
19. Non-specific and specific preventive measures in acute viral hepatitis B, C and D.
20. Rules for discharging patients with viral hepatitis from hospital.

#### **3.4.3. Problems for self-control:**

$\alpha=2$

##### Problem 1

Patient Z. of 42 years was hospitalized with the infectious disease ward on the 23<sup>rd</sup> day of the disease with complaints of general weakness, absence of appetite, sickness

and periodical vomituration. The disease progressed gradually and began with general weakness, sickness, deterioration of appetite and pain in joints. Several days later: vomituration especially in response to smell of cooking food, anorexia, heaviness in the epigastriac area; on the 16<sup>th</sup> day of the disease, the patient observed dark urine, and during the following days – typical signs of jaundice. The patient was operated 3 months ago for colonic intussusception with hemotransfusion in the postoperative period.

Objective data: grave condition. Answered to questions apathetically. Vomiting during the examination. Significant signs of jaundice, scratches on the skin. Meteorism. Liver is 4cm below the costal margin. The lower pole of spleen is palpated. Faeces are light.

1. Provisional diagnosis.
2. Examination plan.
3. Treatment.

#### **3.4.4. Situational tasks of the second level learning**

**α -3**

In resuscitator-old, who 2 years ago was banned due to the appearance donation in his blood HBsAg, appeared rapidly growing weakness, arthralgia, nausea, anorexia, fever up to 38, 50 C, and after 4 days - bright jaundice, vomiting, insomnia, dizziness, epistaxis, slight tachycardia, a significant enlargement of the spleen, liver tenderness with increased density of the edge. When viewed on a 10 - day of illness, excited, solid, bright jaundice, painful, paste the liver, palpable at the costal arch, tachycardia, hypotension. Appeared ascites. In the Blood - moderate neutrophilic leukocytosis, erythrocyte sedimentation rate 20 mm / h, slightly dominates direct bilirubin, ALT - 4650 U / L, AST - 5240 U / L, thymol test - 22 units, creatinine - 90 mmol / l., Urea 1.6 mmol / l, albumin 18 g / L, globulin - 33 g / l, γ - globulin - 34%, prothrombin index of 32%.

11. Provisional diagnosis.
2. Examination plan.
3. Treatment.

**The right answers**

1. Hepatitis B, acute icteric fulminant form, severe course complicated by acute hepatic encephalopathy

2. - general blood analysis with thrombocytes; RW; hepatic tests: general and conjugated bilirubin, IAT, AlAT in dilution (1 : 10), thymol turbidity test, alkaline phosphatase, GGT; cholesterol;-proteinogram; blood sugar; amylase; coagulogram; rest nitrogen, creatinine; markers of viral hepatitis: B, C, D et al. PCR; urine analysis for bile pigments; ultrasound examination of hepatobiliary system.

3. Antiviral - alpha interferon (recombinant: Intron A, Roferon A, and native realdiron: vellferon, che lovechesky leukocyte interferon) with the threat of chronic or progradiently during synthetic nucleosides - famciclovir (Famvir), lamivudine (Epivir), zidovudine (Retrovir, AZT, timozid), protease inhibitors - saquinavir (Invirase), indinavir (Crixivan), interferon inducers - neovir (tsikloferon) amiksin, immunomodulators - leukinferon, interleukin-1 (Betaleukin), interleukin-2 (roncoleukin). Shown in severe hepatitis B (in the presence of markers of active viral replication) with the threat of acute liver failure, especially when the signs of hepatic encephalopathy, as well as the threat of chronicity.

2. When complications associated with encephalopathy: arresting agitation sodium oxybutyrate, seduk-sen; gastric lavage (nasogastric tube) and high or siphon enema to reduce autointoksi-cation; oral administration of poorly absorbed antibiotics (kanamycin 0.5 g 4 times a day), enterosorbents; fractional enteral nutrition combined with parenteral; Glucocorticosteroids (at least 180-240 mg prednisolone) parenterally; in-fusional therapy; with hemorrhagic manifestations - protease inhibitors and fibrinolysis (epsilon-aminocaproic acid, contrycal, gordoks).

#### **4. Reference materials for classroom independent activities.**

##### **4.1. List of practical tasks to be performed during a practical session:**

- To master a technique of examining patients with parenterally transmitted viral hepatitis.
- To monitor patients with viral hepatitis B, C and D.



- To perform differential diagnostics of with viral hepatitis B, C and D.
- To make a laboratory investigation plan.
- To interpret examination data of a patient with viral hepatitis.
- To identify complications of viral hepatitis.
- To make a treatment plan for a patient with viral hepatitis and to determine medical tactics for possible urgent conditions.

#### **4.2. Professional algorithm for development of practical skills in diagnostics of parenterally transmitted viral hepatitis.**

№	Task	Order of performance	Remarks and notifications as to self-control
1.	To master a technique of examining patients with parenterally transmitted viral hepatitis	1. Identification of complaints  2. Case history  3. Patient's life history 4. Epidemiological anamnesis	Screen out those complaints that are typical for: - general intoxication; - organ injuries; - additional injuries.  Order and time of occurrence of: - asthenic syndrome; - dyspeptic syndrome; - jaundice; - arthralgia, rash.  Chronic diseases of gastrointestinal tract.  To find out: - possible ways of infection;

			- vaccination data.
2.	To monitor a patient	<p>1. General examination</p> <p>Skin, mucous membranes</p> <p>Nervous system</p> <p>2. Palpation</p> <p>Abdomen</p> <p>3. Percussion</p> <p>Heart</p> <p>4. Auscultation</p> <p>Heart</p> <p>Lungs</p>	<p>- To determine the overall patient's condition.</p> <p>- any signs of jaundice, rash and its pattern.</p> <p>- meningeal signs;</p> <p>- focal symptomatology;</p> <p>- pathological reflexes.</p> <p>- size and characteristics of liver, spleen and pancreas;</p> <p>- palpatory tenderness, Voskresensky's symptom.</p> <p>- percussion heart borders, pulse, arterial blood pressure</p> <p>- rhythm and intensity of heart sounds;</p> <p>- breathing pattern: vesicular, rough or bronchial; diminished or intensive.</p>
3.	To perform laboratory investigation	<p>1. General blood analysis</p> <p>2. General urine</p>	<p>- level of leucocytes, stab neutrophils and lymphocytes, ESR;</p> <p>- presence of leucocytes, erythrocytes, protein, bile pigments;</p> <p>- level;</p> <p>- presence of stercobilin.</p>

	analysis	
	3. Coprocytogram	Size and acoustic characteristics of liver and spleen; gall bladder and pancreas status.
	4. Urine diastase	
	5. Electrocardiogram	
	6. Ultrasound examination of abdominal organs	Detection of DNA and RNA agents in blood.
	7. Polymerase chain reaction	Antigens and antibodies to antigens of viral hepatitis agents.
	8. Enzyme immunoassay	

### 5. Materials for methodical supplying of lesson( $\alpha=3$ )

#### General classification of parenteral viral hepatitis.

Classification	Characteristics		Etiology		
			B	C	D
By duration:	acute		+	+	+
	prolonged		+	+	+
	chronic		+	+	+
By clinical	asymptomatic	carriage	+	+	+

manifestation:		subclinical	+	+	+
	with clinical manifestation :	non-jaundice	+	+	+
		jaundice	+	+	+
By course:	cyclic		+	+	+
	acyclic	with exacerbation	+	+	+
		with relapses	+	+	+
By severity:	mild		+	+	+
	moderate		+	+	+
	severe		+	+	+
	fulminant	early	+	+	+
		late	+	+	+

## 6. Materials of after-work

**α -4**

Proposed topics for essays on the most pressing issues, such as:

- " Peculiarities of fulminant hepatitis.
- → Modern methods of specific diagnosis of hepatitis C.
- → Ways of specific prevention hepatitis B.

## **The HIV-infection. AIDS-associated infections and invasions**

### **Actuality of theme**

Hardly in the world there is now a pathology that would attract as much attention as HIV infection. Particularly acute problem gives not only that suffer mostly young people are the most active life -trudovuyu, social, sexual, and that while each contracting HIV doomed. The prevalence of HIV - infection in Ukraine - 283.6 per 100 thousand. Population with more than 24 thousand. HIV - infected persons has reached the final stage of the disease - AIDS. Has a prevalence rate of 52.9 per 100 thousand People. Despite the most active work of scientists of all countries, yet manages to achieve only that the use of antiretroviral drugs prolongs the life of the patient.

### **Study purpose of practical studies:**

#### Etiology.

HIV infection is caused by the human immunodeficiency virus (HIV). HIV belongs to the family of retroviruses (Retroviridae).

The mature HIV virions - a spherical particle diameter of about 100 nm, consisting of a core and a shell. Each RNA molecule contains nine genes (three structural and six regulatory genes). The structural genes include gag, env, pol.

Gag gene encodes the formation of internal proteins (p 17/18, p 24/26, p 55/56). HIV1 and HIV2 differ in molecular weight internal proteins. Thus, HIV-1 contains p 24, HIV-2 - p 26. In the early stages of the disease antibodies appear specifically to p24 and p 26.

HIV env gene encodes the viral envelope protein (gp120 / 105, gp41 / 36). Thus, HIV-1 contains the gp120, and HIV 2 - gr105 that as studs above the surface of cells, gp41 of HIV-1 and HIV-2 gr36 like rod immersed in the membrane. Due to this virus glycoprotein complexes and is capable of attaching to penetrate a cell having CD4-receptors. The pol gene encodes three enzymes: protease, reverse transcriptase, an

endonuclease. Reverse transcriptase, using viral RNA as a template, carries out the synthesis of viral DNA. Endonuclease produces insertion of viral DNA into the host cell genome. Retroviruses are harmful to cells, as incorporated into the chromosome of the cell, acquiring the status of the cellular genome.

Besides the structural genes are regulatory: tat, rev, nef, vpr, vit, vpu. From laboratory animals susceptible to HIV only chimpanzees.

The virus is unstable in the environment. When boiling kill the virus in 1-5 minutes for pasteurization - 30 minutes. 96 ° alcohol kills the virus within 1 minute. Rapidly killed by the action of bleach, 3% hydrogen peroxide solution (3-5 minutes). Resistant to ultraviolet radiation, ionizing radiation, and frozen at -70 ° C. There is evidence of the possibility of agent stored in the external environment for a few days in the dried state, especially in blood and semen.

#### Epidemiology.

HIV-1 is found everywhere, HIV-2 infection is spread primarily in West Africa.

Source of HIV infection - the person: the patient or virus carrier. Human immunodeficiency virus is found in all body fluids: blood, lymph, vaginal secretions, saliva, tears, sweat gland secretions, breast milk, semen, menstrual secretions, cerebrospinal fluid, urine, and bronchial fluid. To infect important concentration of the virus. Sufficient to infect share infekta are blood, semen, vaginal secretions. An important factor influencing the contagious stage of the disease is. Infected blood contains a high dose of the virus in the early stage and the stage of AIDS.

#### Pathogenesis.

HIV can penetrate only in those cells which have receptors thereto. Receptor complex is an antigen CD4. This receptor, the CD4 antigen on the membranes attaching helper, macrophages, monocytes, glial cells and other cells. Therefore, the virus can infect macrophages, oligodendroglial cells and astrocytes of the brain, thymus, bone marrow, endothelial cells of blood vessels, lymph nodes, macrophages, alveolar (lung), Langerhans cells (skin), cervical cells, chromaffin cells and other cells of the

intestine. When HIV-1 is close to the cells having the receptor CD4, the coat protein gp-120 binds to CD4. As a result of exposing the viral transmembrane protein gp-41, one end of which is embedded in the cell membrane of the affected cells, resulting in cell membrane fusion and virus.

Due to the depletion of the population of helper number of drops. AIDS index CD4 / CD8 is reduced to 0.5 or less. Reducing the number and functional activity of the immune system T is a risk factor for cancer and opportunistic infections.

### Clinic

The incubation period lasts from 2-4 weeks to 2-3 months, and according to some longer.

Category A. This category includes asymptomatic HIV infection (the period of the primary and secondary latency), the acute phase of HIV infection and persistent generalized lymphadenopathy.

Clinical manifestations of acute HIV infection are often nonspecific and are polymorphic. allocate:

1. The syndrome of upper respiratory tract and lungs.
2. The syndrome lesions of the gastrointestinal tract.
3. The syndrome of the nervous system.
4. lymphadenopathy syndrome.
5. Syndrome thrombocytopenia.

The content of T-helper cells of patients in this category is equal to or more than 500 cells per l, helper-suppressor index is lowered due to the increase of CD8 lymphocytes. The majority of patients in the blood for HIV antibodies appear, however, 10% of patients antibodies appear later, after 3-6 months, and at 1% - at a later date.

Categories according to AIDS-related complex. The most important syndromes of this stage are: localized skin lesions and mucous membranes of viral, bacterial, fungal

origin. Lack of generalization of the process - the main difference between this stage of AIDS;

CATEGORY C or directly characterized by severe immunodeficiency of AIDS when the number of T-helper cells in the blood serum of less than 200 cells per l, in the terminal phase - about 50 cells per ml. Regardless of the number of CD4 lymphocytes, the presence of the AIDS clinic also gives you the opportunity to diagnose AIDS.

For AIDS characteristic generalization process caused by fungi, protozoa, viruses, bacteria. Accession of opportunistic infections caused by opportunistic pathogens, infection which in humans with a normally functioning immune system is not capable of causing disease or infectious process is easy.

### **Pneumocystis pneumonia**

Pathogen - Yeast-like fungus *Pneumocystis jiroveci* (formerly *carini*).

Source of infection - the sick person or vehicle.

The route of transmission - airborne.

At the beginning of the AIDS PCP little noticeable prodrome stretched somewhere to 3 weeks. Fever may be high, but there is shortness of breath (the number of 30 or more breaths per min.), Cyanosis. Troubling severity with scarce local data (auscultation - dry rales on chest radiograph - increased lung markings). Then appears unproductive cough with expectoration of so-called "milk" phlegm (frothy, dense). Radiological examination held for 3-4 weeks of illness, you can see the fine meshed pattern, gain root infiltration, a symptom of "frosted glass" oblakovidnye shadow areas balloon emphysema, ie we see the light, as if through a veil. Patients die from severe respiratory failure.

The diagnosis is confirmed by detection of *Pneumocystis* in bronchial secretions obtained by bronchoscopy.



Important is the prevention of AIDS patients PCP. By reducing the content of CD4-lymphocyte less than 200 cells in 1 mm appointed trimethoprim-sulfamethoxazole (480 mg) two tablets daily.

In the case of *Pneumocystis carinii* pneumonia in AIDS patients is considered the gold standard of treatment assignment trimethoprim-sulfamethoxazole (Biseptol, Bactrim, Septra). In severe or moderate during the drug is administered intravenously (5-6 vials three times per day). After stabilization of the patient's condition is applied at a dose of 1820 mg (four tablets of 480 mg) orally. Duration of treatment 21 days. In mild cases, treatment may be administered orally once.

**Candidiasis** - a disease caused by the fungus *Candida albicans* and *Candida tropicalis*.

The most reliable method of diagnosis is the detection of fungi in the material with the mucous membranes of the blood and other body fluids and isolation of pure cultures.

For the treatment of esophageal candidiasis in AIDS patients using 400 mg of fluconazole and after the disappearance of pain 1 200 mg once a day orally or intravenously during 14-21 days, or ketoconazole 200 mg 2 times a day orally for 21 days.

For treatment of vaginal candidiasis, fluconazole is used 100 mg orally once or clotrimazole 500 mg once vaginally.

For the treatment of systemic candidiasis administered fluconazole 600 mg, at normal temperature - 400 mg 1 time per day intravenously for 2-3 weeks, or 0.6-0.8 mg amphotericin B / kg 1 time a day for 2-3 weeks intravenously

**Cryptococcosis** - a disease caused by the fungus *Cryptococcus neoformans*.

Cryptococcus can be found in soil, in various foods, for vegetables. Most infections of the environment occurs with droppings of pigeons, which Cryptococcus breed in large numbers. Human infection occurs mainly by inhalation of dust particles containing Cryptococcus.

Important is the prevention of cryptococcosis. With a decrease of CD4 lymphocytes <50 / ml fluconazole assigned 100-200 mg per day.

With the development of cryptococcosis in patients with AIDS is assigned, despite the high toxicity of amphotericin B 1.0 mg / kg one time a day in combination with intravenous 5-flucytosine 25 mg / kg four times a day intravenously 14 days; Further fluconazole 400 mg 1 time per day orally for at least 10 weeks, followed by 200 mg fluconazole once in one day orally long.

**Cryptosporidiosis** - protozoal infection caused by the intracellular parasite Cryptosporidium. Source of infection - animals: patients and carriers. Transfer mechanism - fecal-oral.

Once in the human body, the oocyst quietly pass through the stomach and into the small intestine of each oocyst leaves 4 sporozoites that invade epithelial cells and multiply rapidly. Kriptosporodii located inside intestinal epithelial cells at the interface between the fibers and the cytoplasm. Villus atrophy, the cell is not receiving power, violated all kinds of exchange.

In immunocompetent persons diarrhea lasts 3-5 days. Patients complain of fever, weakness, nausea, abdominal pain. The chair can be 5-15 times per day. The stools have a very unpleasant smell. Later in 2-3 weeks can be released oocysts.

AIDS clinic characterized by prolonged debilitating fever, diarrhea that can last for months or even years, when the depletion dotigaet critical degree, rapid weight loss. Chance and bronchopulmonary cryptosporidiosis, when he smote the epithelium of the upper respiratory tract. Dyspnea, cyanosis.

Laboratory: Microscopy of the test material (sputum, duodenal contents, feces). When stained by Ziehl-Neelsen kriptosporidii visible.

For the treatment of cryptosporidiosis in AIDS patients administered paramomitsin 1.0 grams 3 times per day in combination with azithromycin, 600 mg 1 time per day orally for 4 weeks; Further paramomitsin 1.0 g 2 times a day orally for 8 weeks.

**Toxoplasmosis** - protozoan diseases. Pathogen - *Toxoplasma gondii*. The source of infection - the animals, especially cats, are excreted in the feces of *Toxoplasma* oocysts.

In order to prevent toxoplasmosis in AIDS patients with a decrease in CD4 cell counts less than 100 cells / mm trimethoprim - sulfamethoxazole (TMP - SMZ) 2 tablets every day.

With the development of cerebral toxoplasmosis in AIDS patients administered 200 mg pyrimethamine once the first day, then pyrimethamine 25 mg three times daily or 50 mg twice daily in combination with leucovorin 15 mg once per day and 1.0 g sulfadiazine into every 6 hours 6- 8 weeks.

Of primary importance of viral infections are herpes and, above all, **cytomegalovirus** (CMV), which is diagnosed in 20-40% of HIV-infected people and the cause of death in every five of them.

Pathogen CMV - herpes virus type 5 is not sensitive to interferon.

In order to prevent dissemination of CMV infection in AIDS patients with a decrease of less than 100 CD4- lymphocytes in 1 l of the need for primary prophylaxis with ganciclovir.

If there clinic CMV infection in AIDS patients using ganciclovir 5 mg / kg i.v. 2 times a day for 14-21 days, or 90 mg foscarnet / kg i.v. 2 times a day for 14 days.

Among bacterial infections of the greatest urgency gain mycobacterioses tuberculosis.

Laboratory diagnosis: For diagnosis of HIV infection requires laboratory confirmation: detection of antibodies to HIV antigens, genetic material of the virus,

and the virus itself. However, HIV testing is carried out on-infetsiyu Informed consent.

Already in the acute phase, many patients appear antibodies to p24, gp 120 and gp 41. Number of antibodies decreases during AIDS. For detection of antibodies using enzyme immunoassay (EIA).

Determination of viral antigens. Most determine p24 protein by ELISA. The method is very simple, used in blood transfusion. The result is obtained in a few (3-5) minutes. However, p24 protein can be detected only to its binding with antibodies to it, which, unfortunately, occur early in the disease.

In recent years, the greatest recognition of the PCR (polymerase-chain reaction), has a very high degree of sensitivity. There are test kits that detect 20 HIV RNA in 1 ml of serum. There are 2 variants of PCR:

- detection of HIV RNA included in the virions (this method is used for the quantitative determination of HIV in the blood and control treatment);
- detection of HIV proviral DNA integrated into the genome of peripheral blood mononuclear cells (used to diagnose HIV infection).

Recommend set of PCR in combination with ELISA (ELISA first defining the antibody is then PCR). These reactions are not interchangeable.

Reliable sign of HIV infection is the isolation, cultivation and identification of the virus in cell cultures. However, this method is time-consuming, requires highly skilled performers and special equipment.

### **Treatment**

Currently, there is no possibility of complete elimination of HIV from the human body. The goal of therapy in prolonging the life of the patient and longer maintaining the quality of life of those infected. For therapy using anti-retroviral drugs (ARV). The essence of ART is that the reverse transcriptase builds DNA code.

The main indicator for the decision to initiate ART is the number of CD4-lymphocytes. In Ukraine recommended to administer antiretroviral therapy in patients

with a history of any AIDS defining illness and / or reducing the number of CD4-lymphocyte less than 350 cells / mm. Regardless of the number of CD4-lymphocyte ART should be initiated in the following patient groups: pregnant women; patients with HIV-associated nephropathy; Patients co-infected with HBV / HIV when indicated for treatment of HBV infection. ART can consider individual patients with the number of CD4-lymphocytes, more than 350 cells / mm: high HIV viral load (> 100,000 copies / ml); rapid reduction of CD4-lymphocytes (120 cells / year), which was confirmed in two studies at intervals of 14-28 days; Patients older than 50 years; risk factors not associated with HIV (ischemic heart disease, cancer). ART should be started well in all patients with active tuberkulzom, regardless of the number of CD4-lymphocytes, after reaching a satisfactory tolerability of effective anti-TB therapy.

Antiretroviral drugs are divided into several four groups: nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PI), integrase inhibitors (AIs).

By NRTIs include zidovudine (AZT), lamivudine (ZTS), stavudine (d4T), didanosine (ddI), abacavir (ABC), emtritsitabin (FTC), tenofovir (TDF), the combination of zidovudine + lamivudine (AZT + ZTS), the combination of zidovudine + lamivudine + abacavir (AZT + CCTV + ABC).

NNRTI include efavirents (EFV) and nevirapine (NVP), etravirine (ETR).

To protease inhibitors include lopinavir + ritonavir (LPV / rtv), nelfinavir (NFV), pitonavir (RTV), saquinavir (SQV), atazanavir (ATV), fosamprenavir (FPV), darunavir (DRV).

By the integrase inhibitor raltegravir applies (RAL).

Since 1996, the combination therapy of HIV infection in three preparations. HAART - highly active antiretroviral therapy. Appointed 2 drug NRTI and NNRTI one drug or drug SP 1. First-line regimens recommended in Ukraine, based on the recommendations of the WHO Expert. These are:

1. Schemes based on 1 NNRTI + 2 NRTIs:

efavirents + TDF + emtritsitabin (or lamivudine);

2. Schemes based on 1 PI / rtv + 2 NRTIs:

Lopinavir / ritonavir (LPV / rtv) + tenofovir + emtritsitabin (or lamivudine)

Alternative regimens:

**2.1. The student must have an idea (read):**  **$\alpha$  -1**

For its short history the world was enveloped in the HIV/AIDS epidemic which turned into a pandemic result in death over 3 million people of mainly young age annually.

For Ukraine, as well as for most other countries of the world, a problem of HIV/AIDS is not only medical but also socioeconomic.

On the rates of HIV-infection spread, Ukraine has a significant place in Europe. HIV-infection growth in Ukraine is accompanied by tuberculosis drug addiction and other epidemics.

**2.2. Student have to know:**  **$\alpha$  -2**

- etiology of HIV, pathogenicity factors of the pathogen ;
- epidemiology of HIV;
- pathogenesis of HIV;
- classification and clinical forms of HIV;
- clinical manifestations of HIV;
- complications of HIV;
- clinical features and laboratory examination of patients with HIV ;
- research methods used for the specific diagnosis of HIV;
- rules for fencing material for laboratory studies of patients with HIV ;
- clinical and laboratory diagnosis HIV;
- principles of treatment of HIV;
- • prognosis of HIV;
- principles of prevention of HIV;

- rules for convalescents discharge from hospital ;
- rules of the observation convalescents ;
- principles of HIV hospital.

**2.3. Student have to be able:**

**α -3**

1. HIV-infection definition.
2. Etiology of the disease: the agent, its basic properties, classification.
3. Epidemiology: source of infection; mechanism, ways of transmission. Risk groups. Diaplacental infection. Immunity. Features of epidemiological process clinical behavior.
4. Pathogenesis: virus' penetration; stages of interaction with immunocytes; mechanism of immunological disorders forming; activating the opportunistic infections agents. Pathologic anatomy. Pathomorphology.
5. Clinical picture: classification; stages of HIV-infection clinical course; incubative period; generalized lymphadenopathy syndrome; AIDS-associated complex; HIV terminal stage (AIDS). Criteria of diagnosing and diagnosis' statement. Characteristics of basic opportunistic infections. Differential diagnostics. Pathologic anatomy. Pathomorphology.
6. Diagnostics: value of epidemiologic, clinical and laboratory information complex for early HIV-infection diagnostics; specific methods of HIV-diagnostics: serologic (IFA, immunobloting and other), virologic, examination of immune system state (phagocytosis, cell-mediated and humoral immunity).
7. Treatment: specific therapy: treatment regimen and courses of treatment; anti-virus drugs, groups
8. Prophylaxis: personal, urgent

**2.4. Educational goals (goals of the person):**

- Develop deontological conception in the study subjects.

- To be able to observe the rules of conduct in the bedside, the principles of medical ethics.
- Master the ability to establish psychological contact with the patient and his relatives.
- Develop knowledge of the impact of socio-hygienic factors on the prevalence of HIV.
- The subject materials to develop a sense of responsibility for the timeliness and accuracy of professional activities.

**3. Materials for out-class self-training (before practical classes)**  
**3.1 Materials for out-class self-training (before practical classes)**

1. To form an idea of the basics of psychotherapeutic approaches to patients with HIV.
2. To develop understanding of the impact of hygiene and social factors on human health in HIV.
3. On the topic material to develop a sense of responsibility for the timeliness and appropriateness of professional action.
4. Be able to set individual psychological contact with the patient and his relatives.

**3.2 Basic knowledge, skills which are necessary for studying of topic (interdisciplinary integration)**

<b>Discipline</b>	<b>To know:</b>	<b>To be able to:</b>
Microbiology	Etiology of HIV	Performance of virological investigations and analysis of polymerase chain reaction data
Anatomy,	Immune system structure,	Identification of histological



Pathologic Anatomy and Histology	histological changes in immune system, lymphocytes	immune changes (microscopic investigation)
Epidemiology	Ways of transmission and infection mechanism in HIV	Anti-epidemic and preventive measures, emergency prevention
Immunology and Allergology	Immune affection in infection-allergic and cytopathogenic mechanisms. Serological examination procedure.	Analysis of immunologic investigation data
Nervous Diseases	Semiotics of nervous system damage in acute encephalopathy (dementia): <ul style="list-style-type: none"> <li>• general cerebral symptom</li> <li>• focal symptom</li> <li>• hypertensive symptom</li> <li>• meningeal symptom</li> </ul>	Identification of the following clinical symptoms: <ul style="list-style-type: none"> <li>• general cerebral</li> <li>• focal</li> <li>• hypertensive</li> <li>• meningeal</li> </ul>
Introductory Course of Childhood Diseases	Basic stages and methods of patient clinical examination	Analysis of patient clinical examination data, including laboratory test data.
Pharmacology	Basic medicines of the following pharmacological groups:	Writing out of a prescription for basic medicines of the following pharmacological

	<ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antibacterial</li> </ul> <p>Age dosage of medicines.</p>	<p>groups:</p> <ul style="list-style-type: none"> <li>• antiviral</li> <li>• corticosteroid</li> <li>• antibacterial</li> </ul>
Resuscitation and Intensive Care	Urgent conditions: - AIDS	Urgent medical care for AIDS
<b>Other courses</b>		
Family Medicine	Pathogenesis, epidemiology, dynamics of clinical manifestations and possible complications of HIV. Principles of prevention and treatment.	<p>Differential diagnostics of other diseases of various geneses versus HIV.</p> <p>Identification of HIV with contact mechanism of transmission and its possible complications; interpretation of laboratory examination data.</p> <p>Timely hospitalization of patient in the infectious inpatient department.</p> <p>Completion of an emergency report. Provision of urgent medical care, if necessary</p>
<b>Intracourse integration</b>		
<b>Infectious Diseases</b>	Special characteristics of infectious diseases. Principles of diagnostics, treatment and	Differential diagnostics of HIV with parenteral mechanism of transmission versus other

	prevention of infectious diseases. Pathogenesis, epidemiology, dynamics of clinical manifestations, laboratory diagnostics, possible complications and clinical course peculiarities of HIV. Principles of prevention and treatment.	diseases of various geneses. Identification of HIV and its possible complications; interpretation of laboratory examination data. Prescribe treatment.
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### 3.3. Self-control materials

#### 3.3.1. Questions to be answered

**α -1**

1. What group of infectious diseases at the source of infection is HIV ?
2. The mechanism of infection, ways and factors of transmission of HIV.
3. The etiology of HIV, pathogenicity factors of the pathogen.
4. The stages of the pathogenesis of HIV.
5. Pathological changes in organs and tissues with HIV.
6. Causes of death in HIV.
7. The prognosis for c HIV.
8. Algorithm examination of patients with suspected HIV
9. Methods of specific diagnostics of HIV.
10. The main stages of the treatment of HIV.
11. Principles therapy for HIV.
12. Terms of convalescents discharge from hospital .
13. Main areas of prevention of HIV.

### 3.3.3. Situational tasks of the second level learning

α -3

#### Task 1

In HIV-infected showed an increase of up to 2 cm and submandibular glands zadnesheyh, the skin over them is not changed, the lymph nodes are not soldered. From lymph node biopsy specimens isolated mycobacteria Gordonae. In immunological -CD4 lymphocytes - 300 cells / mm, CD4 + / CD8 + equals 1.2.

1. Preliminary diagnosis
2. Pattern of examination.
3. Plan treatment.

#### Task 2

Patient A. 30 years, during 2 months fever 38,0 -38,9 ° C, weakness, abdominal pain, diarrhea, weight loss. Anemia, leukopenia, thrombocytopenia. PCR - HIV RNA. Isolated from the blood of Mycobacterium avium. In immunological: CD4 lymphocytes - 90 cells / mm. Index of CD4 + / CD8 + is 1.0.

1. Preliminary diagnosis
2. Pattern of examination.
3. Plan treatment.

#### Task 3

HIV-infected complains pronounced sweating, increased temperature- 38,5 °, cough, chest pain. Radiography - focal pneumonia. Isolated from sputum Mycobacterium Kansasii. In immunological CD4limfotsity - 400 cells / uL, CD8limfotsitov - 300 cells / mm.

1. Preliminary diagnosis

2. Pattern of examination.
3. Plan treatment.

#### **4. Materials for the class of independent work**

##### **4.1. List of study practical tasks to be performed in the practice:**

1. Learn the basic rules work at the bedside .
2. Take the history of the disease with the evaluation of epidemiological data.
3. To provide curation of patient and identify symptoms and syndromes of HIV boulders based on their clinical stages substantiate the clinical diagnosis for timely referral of the patient to the hospital .
4. Making medical records of suspected HIV.
5. Based on clinical examination time to recognize possible complications HIV.
6. Making medical documentation at establishing a preliminary diagnosis HIV.
7. Make a plan and additional laboratory examination of the patient.
8. Interpret the results of laboratory testing.
9. Right, depending on the material and the term survey to assess the results of specific diagnostic methods. To make a treatment plan based on epidemiological data, stage of disease , presence of complications, severity of condition, allergist anamnesis, concomitant pathology, able to provide immediate assistance.
10. Make a plan and emergency for the prevention of the source of infection .
11. Give recommendations on treatment , diet, inspection , supervision in the recovery period.
12. To define medical tactics in the case urgent conditions origin.
13. To issue the medical documentation.

#### **4. Materials of after-work**

**α -4**

Proposed topics for essays on the most pressing issues, such as:

"Prospects for early diagnosis of HIV»

"Clinical and epidemiological characteristics of HIV"

"Differential diagnosis of HIV"

«Prevention of HIV"

"Opportunistic infection"

## The Herpesvirus infections

### Actuality of theme:

Herpetic infection is characterized with different localization of process and various clinical forms and caused by viruses from Herpesviridae family, that contain DNA and characterized by long persistence in human population, taking 3rd place after cardiovascular and oncological pathology. Diseases caused by herpes viruses 1<sup>st</sup> and 2<sup>nd</sup> type taking 2<sup>nd</sup> place (15,8%) after influenza (35,8%) as cause of death after viral infections by data of WHO. In 70% of cases infection is results from asymptomatic carriers.

Etiologic role in pathology of human take part 8 types of herpesviridae, that by biological properties and genetic likeness divided on 3 subfamilies:

$\alpha$ - herpesviridae: Poikilovirus (Herpes zoster - VZV or HHV-3) and genus of Simplex virus (virus simple herpes 1 type HSV-1 and 2 type - HSV-2).  $\alpha$ -herpes viruses have least long cycle of replication and cause a strong cytopathogenic effect in cellular cultures and are affined to the epithelium and cells of a central nervous system.

$\beta$  - herpesviridae is included genus of Cytomegalovirus (cytomegalovirus or CMV, or HHV-4). This group of viruses circulates in the organism of human and are affined to lymphatic tissue. Herpes viruses 6 and 7 types last years were obtained from a human organism, their clinical value now is unclear. It is known that HHV-6 causes the syndrome of sudden exanthema in children, and HHV-7 has a pathogenetic value in development of syndrome of chronic fatigue.

$\gamma$  - herpesviridae, genus Rhadinivirus. Genus Lymphocryptovirus is also belongs to  $\gamma$ - viruses, like a 4 type virus of human or Epstein-Barr virus (HHV-5, EBV).

### 1. Tasks of the lesson (with pointing of level of mastering which is planned):

2.1. A student must know:

a-2

- etiology of herpetic infection, pathogenic factors of causative agents;
- epidemiology of herpetic infection;
- pathogenesis of herpetic infection;
- clinical symptoms of herpetic infection depending on type of agent and gravity;
- clinical and epidemical features of herpetic infection;
- pathogenesis, incubation period and clinical symptoms of complications of herpetic infection;
- laboratory diagnostics of herpetic infection;
- principles of treatment of herpetic infection;
- principles of prophylaxis of herpetic infection;
- tactic of conduct of patients in the case of a urgent states;
- prognosis of disease depending on gravity and form of herpetic infection;
- rules of discharge of convalescents from a hospital;
- rules of a outpatient system for convalescents.

**2. Student must be able:**

**a-3**

- to adhere to the basic rules of work bedside of patient with a herpetic infection, chicken pox and herpes zoster;
- to collect anamnesis of illness with the estimation of epidemiology information;
- to examine a patient and find out basic symptoms and syndromes of herpetic infection, chicken pox and herpes zoster, to ground a clinical diagnosis for timely direction of patient in a hospital;
- to conduct differential diagnostics of herpetic infection, chicken pox and herpes zoster;
- to make a plan of laboratory and instrumental diagnostics;
- to interpret results of laboratory and instrumental diagnostics.
- to work out an individual plan of treatment taking into account epidemiological information, clinical form, stage of illness, presence of complications, condition of a



patient, allergic anamnesis, accompanied pathology; to give the first aid on the outpatient level;

- to work out a plan of anti epidemic and prophylactic measures in the pesthole of infection;

- to give recommendations about bed regimen, diet, diagnostic measures and supervision in the period of convalescence.

## 2.1 Educate tasks (for development of personality):

2.1. Basic knowledge and skills for learning of the theme (interobjective integration):

Discipline	To know	To be able
Previous disciplines		
Microbiology	Properties of Herpes simplex I and the II types;	To estimate the results of specific methods of diagnostics
Physiology	Parameters of physiological norm of organs and systems of human; indexes of normal values of laboratory tests (general analysis of blood, urine, CSF, biochemical blood test, parameters of acid-alkaline balance, electrolytes, etc.).	To estimate information of laboratory tests
Pathophysiology	Mechanism of disturbance of functions of different organs and systems in	To assay pathological changes by results of laboratory tests at disturbances of functions of
Immunology and allergology	Basic concepts of the object, role of the immune system in infectious process, influencing for the term of elimination of	To estimate data of immunological tests.

Epidemiology	Epidemic process (source, mechanism and ways of transmission) of herpetic infection.	To collect epidemiological anamnesis, conduct anti epidemic and prophylactic measures.
Dermatology	Pathogenesis, clinical characteristic of exanthema and enanthema	To recognize rash in patient with herpetic infection.
Neurology	Pathogenesis and clinical signs of meningitis,	To conduct a clinical examination of patient with
Ophthalmology	Pathogenesis and clinical signs of keratitis, conjunctivitis, iridocyclitis,	To conduct a clinical examination of patient with affection of eyes.
Propaedeutics of therapy	Methods and basic stages of clinical examination of patient.	To collect anamnesis, conduct the clinical examination of patient, show pathological
Clinical pharmacology	Pharmacokinetics and pharmacodynamics, side effects of antiviral and supportive drugs.	To prescribe treatment depending on age, individual features of patient, to choose the optimum mode of reception
Reanimation and	Urgent stages: brain edema.	To diagnose in good time and
Next disciplines		
Family medicine	Pathogenesis, epidemiology, dynamics of clinical symptoms, possible complications of herpetic infection. Features of clinical course of herpetic	To conduct differential diagnostics of illnesses of different genesis with a herpetic infection. To recognize a herpetic infection, its complications; to estimate
Interdisciplinary integration		

Infectious diseases	Features of infectious diseases. Principles of diagnostics, treatment and prophylaxis of infectious diseases. Pathogenesis, epidemiology, dynamics of	To conduct differential diagnostics of herpetic infection with other infectious diseases. To recognize herpetic infection, its complications; to estimate information of laboratory
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### 3.2. Content of topic of lesson\*

## Herpetic infection

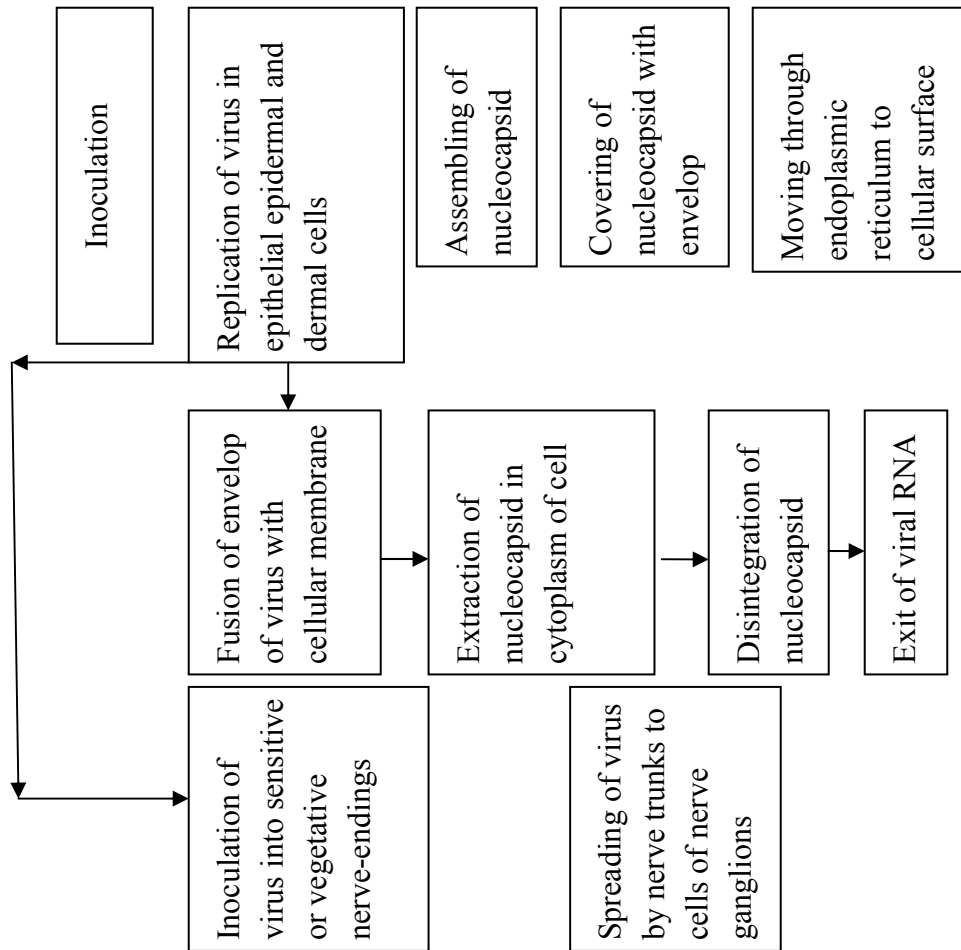
Etiology

**Herpes simplex (HSV-1, HSV-2).**

Epidemiology

Host – infected human. Routs of transmission – air-droplet, contact, sexual, transplacental, intranatal.  
High sensitivity.

pathogenesis



Clinical classification

Localized form

Skin affection

Herpetic dermatitis

Herpeticform eczema  
Kaposi

Ulcerous necrotic  
dermatitis

	Ulcerous necrotic dermatitis
Affection of mucosal layer of mouth	Gingivostomatitis (aphthous, ulcerous, necrotic)
Affection of mucosal layer of respiratory tract	nasopharyngitis
	laryngitis
Affection of eyes	keratitis
	conjunctivitis
	iritidocyclitis
	chorioretinitis
	uveitis
	Neuritis of ophthalmic nerve
Affection of genital organs	urethritis
	balanoposthitis
	vulvovaginitis
	cervicitis
disseminated (generalized) form	
Affection of nervous system	encephalitis
	meningoencephalitis
	neuritis
Affection of other internal organs or systems	hepatitis
	pneumonia
	esophagitis
	enterocolitis

Herpetic affection of skin

Rash on lips, around mouth, sides of nose, less frequent on eyelids, hands, trunk. hyperemia → papule → vesicle → erosion → crust

Plentiful elements → t=37,5-38,5°C, chill, headache

Clinic

Acquired herpetic infection: More early infection – more severe course; Late infection gives milder forms/ asymptomatic; Primary herpetic infection has more severe course, toxicosis, spreading of affection and longer course

Inborn herpetic infection – transplacental route Early term of gravidity – death c of fetus/defects of development (mycrocephalia, microphthalm, choreoretinitis); Late stage / delivery – death of infant/development of generalized herpetic infection (often with fatal outcome)

Without clinical focuses of affection

Latent form

Skin and internal organs

Mixed form

nephritis

Herpetic affection of nervous system

Herpetic encephalitis:  
↑t°C, chill, fever,  
nausea, vomiting,  
headache, focal  
affection (paralysis,  
coma, plegia,  
pathologic reflexes,  
hyperkinesis);  
disturbances of  
consciousness

Retinitis:  
inflammatory  
granulomas on  
retina

Herpetic affection of eyes

keratitis/keratoconjuncti-  
vitis: pain in eye,  
deterioration of vision,  
edema of conjunctives,  
affection of cornea

Relapses: deep  
keratoiritis,  
parenchymatous uveitis,  
keratitis, deep ulcer  
with hypopyon

Herpetic affection of genital organs

↑t°C, weakness,  
myalgias, hyperemia  
and edema of tissues,  
local pain, itching,  
painful urination,  
inguinal lymphadenitis

Rash on penis, urethritis,  
prostatitis: in women on  
outer genital organs,  
cervicitis, urethritis,  
salpingitis,  
endometritis  
papules/vesicles → erosions  
→ crust

Herpetic affection of mouth cavity

Herpetic stomatitis: ↑t°C,  
weakness, headache, fever,  
discomfort and pain in  
places of affection,  
hypersalivation, neck  
lymphadenitis

Rash on mucosal layer of  
cheeks, pharynx, tongue;  
little vesicles, erosions,  
aphthosis

Herpetic pharyngitis:  
↑t°C, weakness,  
headache, sore throat,  
neck lymphadenitis

Rash on mucosal layer of  
pharynx, tonsils:  
vesicles → erosions

Herpetic infection in HIV-positive patients

Affection of skin (deep ulcers) and mucosal layers (mouth cavity → gullet → trachea → bronchi → lungs), than → generalized form (retinitis, choreoretinitis, encephalitis, meningitis)

Generalized herpetic infection

Hectic fever, chill, headache, weakness syndrome, cramps, affection of skin, mucosal layers and other organs (liver, lungs, brain and meningeal membrane)

Herpetic affection of internal organs

Herpetic pneumonia (nodal character of inflammation)

Herpetic esophagitis: dysphagia, retrasternal pain

Herpetic hepatitis: ↑t°C, weakness, headache, muscular pain, jaundice, dark urine, enlargement of liver and spleen, increasing of serum ALAT



complications	Herpetic encephalitis: disturbances of psychological status and Oculomotor paralysis	Herpetic pneumonia: respiratory insufficiency	Generalizes herpetic infection: hemorrhagic syndrome, acute suprarenal insufficiency,
Differential diagnosis	Chickenpox	Herpetiform dermatitis	Allergic dermatitis Herpes zoster; pancreatitis, glomerulonephritis, monoarticular arthritis

Ulcers of genital organs different etiology	Gingivostomatitis different etiology	piodermia	Secondary syphilis
Viral meningitis and encephalitis	Viral heratitis A, B, C, D	keratoconjunctivitis of different etiology	

Laboratory diagnostics	Detection of virus: genome and antigens in content of vesicles, smears from mucosal layers, cerebrospinal fluid, etc.	PCR and molecular hybridization
		Reaction of immune fluorescence method, electronic microscopy
		Cytological and cytochemical: detection of intranuclear insertions in multinuclear cells
	Detection antibodies to HSV (cross-reactions are possible)	IEA: detection of IgM (acute process) and IgG
		Immune blot (Western Blot): confirmation of IEA and estimation of correlation between different classes of anti-HSV Ig

treatment	Specific therapy	Acyclovir, valacyclovir, phamcyclovir, gancyclovir
		Specific immunoglobulin
		Interferon: lapheron, vipheron, ropheron, intron - A
		Inductors of interferon: amyxin, neovir, inozit, cyclopheron
	Supportive treatment	dezintoxication
		antibiotocs

Prophylactics	Primary herpetic infection – hygienic measures	Acyclovir, amyxin
	Relapsing herpetic infection	Inactivated herpetic vaccine

**VZV-infection**

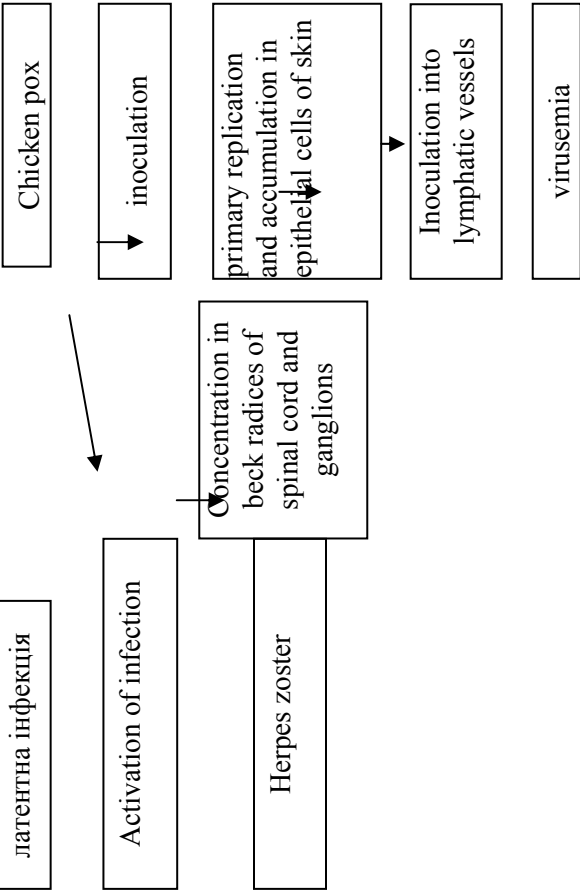
etiology

Varicella (herpes) zoster (VZV)

Epidemiology

Host of infection- person with chicken pox or herpes zoster. Routs of transmission: droplet, contact, perinatal. Susceptibility is high

Pathogenesis



Clinical classification

CHICKEN POX

HERPES ZOSTER

Clinic

- Gangliodermal form
- Ophthalmic form
- Hant syndrome
- Gangrenous (necrotic) form
- Meningoencephalitic form

CHICKEN POX

Incubation period	Prodrome period	Period of rash	Convalescence period
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Moderate weakness, loss of appetite, subfebril temperature

rash (on head, shoulders, breast, abdomen, legs): hyperemia of skin→papula→vesicle→crust

enanthema (on mucosal layer of cheek, tongue): little vesicles→erosions (sometimes-apthosis coated with gray fur)

Severe forms (bullas, pustulas, hemorrhages) t=38,5-39,5°C, chill, weakness, headache, mialgias

HERPES ZOSTER

Ganglia -skinal form→rash – by projection of nerves: hyperemia of skin→papula→vesicle→crust

Ocular form: very sever course, affection of n. trigemini; rash by projection of branches n. trigemini (on mucosa of eye, nose, on skin of face); specific viral keratitis, iritis, glaucoma

Hant syndrome (ears affection): affection of ganglion; rash – on auricle, paralysis of n. facialis is possible

Mortify (necrotic) form: deep affection of skin with formation of ckars

Meningoencephalitic form (rare): severe course, mortality more than 60%; ganglio-skinial affection→meningoencephalitis (ataxia, hallucinations, hemiplegia, meningeal signs)

Complications	postherpetic neuralgia	Pneumonia	Hepatitis
	Segmental myelitis	Ulcers of cornea	Myocarditis
Differential diagnosis	Herpetic infection	erysipelas	eczema
	impetigo	Allergic dermatitis	Contact dermatitis
		piodermia	Secondary syphilis
Laboratory diagnostics	Detection of virus: genome and antigens in content of vesicles, smears from mucosal layers, cerebro-spinal fluid, etc.	PCR and molecular hybridization	
		Reaction of immune fluorescence, electronic microscopy	
		IEA: detection of IgM (acute process) and IgG	
treatment	Specific therapy	Antiviral drugs: acyclovir, valacyclovir, famcyclovir, gancyclovir	
		Specific immunoglobulin	
		Interferon: laferon, viaferon, roferon, intron-A	
		Inductors of interferon: amixin, neovir, inozit, cycloferin	
	Supportive treatment	desintoxicattion	
		antibiotics	
		Local treatment	
prophylactics	Immunoglobulin for persons with immunodeficiency		

\*Additionally to structure and logical scheme in chapter 3.2 “Content of theme” can be used tables of differential diagnostics, thesis (depending of theme difficulty, its

novelty, presence of lecture material, etc.), normative documents of MHP regulating plans of diagnostics, prophylactics, treatment of infectious diseases, included in theme of lesson.

### **3.4 Materials for self-control**

#### **3.4.1. Questions for self-control:**

1. To what group by transmission belong herpetic infection, chicken pox and herpes zoster?
2. Routes of transmission of herpetic infection, chicken pox and herpes zoster.
3. Stages of pathogenesis of herpetic infection, chicken pox and herpes zoster.
4. Clinical classification of herpetic infection, chicken pox and herpes zoster.
5. Main symptoms of herpetic infection, chicken pox and herpes zoster depending from form.
6. Peculiarities of herpetic infection course in patients with AIDS.
7. Complications of herpetic infection, chicken pox and herpes zoster.
8. Main reasons of mortality after herpetic infection, chicken pox and herpes zoster.
9. Plan of diagnostics of herpetic infection, chicken pox and herpes zoster.
10. Spinal liquor count in case of affection of nervous system, caused by herpetic infection.
11. Methods of specific diagnostics of herpetic infection, chicken pox and herpes zoster.
12. Specific therapy of herpetic infection, chicken pox and herpes zoster: dosage, prescription, duration of treatment.
13. Principles of supportive treatment of herpetic infection, chicken pox and herpes zoster.
14. Rules of discharge a patients after herpetic infection from hospital.

#### **3.4.2 Tests for self-control**

**a=2**

1. The viruses of the Herpes simplex type 1 and 2 are related to the subfamily of:
  - A. Alphaherpesviruses;
  - B. Betaherpesviruses;
  - C. Gammaherpesviruses;

- D. 1<sup>st</sup> and 2<sup>nd</sup> types are related to the different subfamilies.
2. The chicken pox infection agent is:
- A. Cytomegalovirus;
  - B. Epstein Barr Virus;
  - C. Herpes Virus type 1;
  - D. Herpes Virus type 2;
  - E. Herpes zoster.
3. Specific methods of the herpetic infections diagnosis:
- A. Widal's test in the blood serum;
  - B. Immunoenzyme analysis;
  - C. Hemoculture;
  - D. Fluorescing antibodies reaction;
  - E. Culturing of faeces and urine.
4. The source of infection diseases, caused by herpes zoster are:
- A. Patient with infection mononucleosis;
  - B. Patient with herpes virus type 6;
  - C. Patient with chicken pox;
  - D. Patient with Herpes zoster infection;
  - E. Patient with Herpes virus type 8 infection.
5. Herpes Virus type 1 affects:
- A. Face;
  - B. Skin above the waist;
  - C. Mucous coat of oral cavity and lips;
  - D. Genitales;
  - E. Skin below the waist.
6. What is typical for clinical picture of chicken pox in adults:
- A. Rash is symmetrical;
  - B. Polymorphism of rash;

- C. Absence of phase character of rash;
  - D. Elements of rash on top of head;
  - E. Elements of rash mainly on plants and foots.
7. Eruption by the chicken pox includes such elements as:
- A. Vesicles;
  - B. Roseolas;
  - C. Papule;
  - D. Pustule;
  - E. Crust.
8. What drug is used for the treatment of the genital herpes:
- A. Amoxyclan;
  - B. Famciclovir;
  - C. Phenosal;
  - D. Aciclovir;
  - E. Valciclovir.
9. Unfavourable prognosis observed in case of chicken pox:
- A. Conjunctivitis;
  - B. Malignant form of the chicken pox;
  - C. Keratitis;
  - D. Severe form of encephalitis;
  - E. Vestigial (rudimentary) process of the disease.
10. The rules of the discharge of patient after the chicken pox from the hospital:
- A. After 3 times negative result of the culturing of faeces;
  - B. After the negative result of the Widal's reaction;
  - C. After clinical recovery;
  - D. Not earlier than 21 days after the temperature normal;
  - E. After the negative result of the hemoculture.

Etalons of the right answers:



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

To fill the table

a=2

**Drugs, which are used in treatment of infections, caused by  $\alpha$ -herpesviruses  
1<sup>st</sup> - 3<sup>rd</sup> types (acute period)**

Drugs	$\alpha$ - herpesviruses 1 <sup>st</sup> - 3 <sup>rd</sup> types
Cyphran	-
Acyklovir	+
Streptomycin	-
Lapheron	+
Prednisolone	-
Amyxine	+
Nonsteroid antiphlogistic	-
Cyclopherone`s liniment (locally)	+
Specific immunoglobulin therapy (IM, IV)	+
Ampycillin	-

3.4.3 Tasks for self-control

a=2

Task 1

a=2

23-years-old patient was hospitalized on the 5<sup>th</sup> day of his disease, which has begun with forerunners in the form of quick tiredness, headache, skin itching. Blood heat - 38°C. There were papules on the chest skin which are quickly reformed into vesicles. Objectively: medium-weight state. There are vesicles with serous contents on the chest skin and lateral surface of body on the left sight. Here and there are observed some pustules. Appearance of skin rush attend with strong pain along intercostals nerves which is keeping during examination.

1. Formulate previous diagnosis.
2. Plan of examination
3. Plan of treatment.

## **Task 2**

a=2

Patient turned to the doctor with complaints of painful sensation and eruption on his penis, which appeared after he had flu. OE: on the balanus observed integrated vesicles and erosions with scalloped edges, with distinct limit, soft by palpation, which are accompanied with painfulness.

1. Formulate previous diagnosis.
2. Plan of examination
3. Plan of treatment.

## **Task 3**

a=3

18-years-old patient hospitalized in severe condition. From the mother's words he is ill during 1st day. Disease began from chill, headache, blood heat increased until 39,9°C, emesis. OE: coma 1 stage, excitement, muscular rigidity of neck muscles, Kernig's symptom +++, pale hot skin, arterial pressure – 70/0, pulse – 140 beating. There is abundant herpetic eruption on the mucosal layer of lips.

1. Formulate previous diagnosis.
2. Plan of examination
3. Plan of treatment.

### **4. Materials for auditory self work**

4.1 List of educational practical tasks for practical lesson:

- to know the methodic of examination of patient with herpetic infection, chicken pox and herpes zoster;
- To perform examination of a patient with herpetic infection, chicken pox and herpes zoster;
- To perform differential diagnosis of herpetic infection, chicken pox and herpes zoster;
- To make a plan of laboratory diagnostics;
- To interpret results of specific tests for herpetic infection, chicken pox and herpes zoster;



			<p>the contacts to a chicken pox and zoster patient.</p> <p>To remember: presence, expression, symptoms' dynamic are conditioned by a term and an intensity of the process of the disease, its form and depend on the age of the patient and concomitant pathology.</p>
2.	To perform examination of a patient	<p>III. To perform an objective examination</p> <ol style="list-style-type: none"> <li>1. General examination <ul style="list-style-type: none"> <li>- common condition of the patient;</li> <li>- skin and mucous coats</li> <li>- lymphatic nodes</li> </ul> </li> <li>2. Digestive system <ul style="list-style-type: none"> <li>- tongue examination;</li> <li>- abdomen percussion;</li> <li>- abdomen palpation;</li> <li>- stool characterization.</li> </ul> </li> <li>3. Cardiovascular system: <ul style="list-style-type: none"> <li>- Pulse;</li> <li>- arterial tension;</li> <li>- heart auscultation;</li> </ul> </li> <li>4. Respiratory system <ul style="list-style-type: none"> <li>- lungs auscultation</li> </ul> </li> <li>5. Nervous system</li> <li>6. Vision</li> <li>7. Genitourinary apparatus</li> </ol>	<p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- temperature</li> <li>- pain syndrome</li> <li>- eruption (taking into account the hair covered part of the head, conjunctivitis, mucous coats of the oral cavity) their localization (along the nerve trunks etc.), eruption character and the terms of its appearance</li> <li>- itch</li> <li>- their possible increasing</li> </ul> <p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- eruption ( by the lesion of upper branch of n. trigeminus herpes zoster)</li> <li>- hepatolienal syndrome (by the general forms)</li> </ul> <p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- pulse matches the temperature</li> <li>- there is a tendency to hypotension</li> <li>- does not exclude the development myocarditis and endocarditis</li> </ul> <p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- the symptoms of the pneumonia among the part of the patients, especially by adults (dry and moist rales etc.)</li> </ul> <p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- encephalitis, meningitis, myelitis, ganglialitis, neuritis.</li> </ul>

			<p>neuralgia, arachnoiditis development possibilities</p> <p>To pay attention to:</p> <ul style="list-style-type: none"> <li>- eruption on the genitals</li> <li>- frequent, painful emiction.</li> </ul>
3.	To prescribe laboratory additional researches, interpret the results.	<ol style="list-style-type: none"> <li>1. Complete blood count</li> <li>2. Complete urine count</li> <li>3. Cerebrospinal fluid (in case of meningitis or local neurological symptoms)</li> <li>4. Microscopy of the smear from the eruption elements</li> <li>5. PCR</li> <li>6. Fluorescing antibodies reaction</li> <li>7. Serum tests: complement fixation test, indirect hemoagglutination reaction, radioimmune analysis. Immunoenzyme assay</li> <li>8. X-ray of the chest</li> <li>9. Ultrasound scan of abdominal cavity</li> </ol>	<p>To pay attention to: leucopenia, lymphocytosis, normal ESR Absence of the considerable changes by the typical process The picture looks like miliar tuberculosis Detecting antigen of Varicella zoster virus</p> <p>Taken twice: in the acute period of the disease and in the recovery period (in 7-10 days), the reaction is considered to be a positive one by the increasing of the titre of the antibodies not less than in 4 times more (by chicken pox) for the detection of the Herpes Simplex Virus's DNA</p> <p>Is usually lymphocytal, with a normal or increased protein content and the lymphocytes quantity, reducing Na and glucose quantity Found the polynuclear cells with eosinophils inclusions in the nucleus, characted for the infection Varicella zoster virus</p> <p>Detection of the antibodies of Ig M class to the antigens of Herpes Simplex type 1 and 2 for acute infection, Ig G - for chronic relapsing process for the detection of the antigens of the Herpes Simplex Virus in vesicles content Enlargement of lever in case of hepatitis</p>

\*\* Additionally in the part 4.2 may be used according to the cathedral choice the diagnostic algorithms, the instructions and the orders of the Ministry of Health towards to prophylactic measures, the examinations and help to the patients with the infected diseases, which were included into the lesson topic.

### **5. Materials for post-auditorium self- education**

Themes of Educational Research Work of Student (ERWS):

- Peculiarities of course of  $\alpha$ -herpetic infection, virus 1-3 type, in present time.
- Modern methods of specific diagnostics of  $\alpha$ -herpetic infection, virus 1-3 type;
- Problems of specific treatment of  $\alpha$ -herpetic infection, virus 1-3 type, in present time;
- Problems of prophylactics of relapses of  $\alpha$ -herpetic infection, virus 1-3 type

### **Theme: EBV CMV-INFECTIONS, MONONUCLEOSIS**

#### **Actuality of theme:**

High contamination and spreading among population of the world is the main problem of EBV and CMV infections, especially in developing countries. Infection is found in 80% children younger 3 years. Properties of viruses for lifelong persistence has described and their connection with slow infections and neoplastic diseases (EBV). EBV- CMV are pathological agents of opportunistic infections in patient with AIDS. EBV and CMV infection develop in 50% patients after organ transplantation. Donor blood has high risk of transmission CMV and EBV infection. Therefore EBV and CMV is important in different field of medicine.

#### **2. Study purpose of practical studies:**

##### **2.1. A student must know:**

**a-2**

- Etiology of EBV-infection and CMV-infection, characteristic of viruses;
- Epidemiology of EBV-infection and CMV-infection;
- Pathogenesis of EBV-infection and CMV-infection;
- Clinical features of EBV-infection and CMV-infection of typical clinical forms;
- Classification of clinical forms of CMV-infection;
- Classification of clinical form of EBV-infection;

- Peculiarities of clinical features of EBV-infection and CMV-infection in dependence of their forms;
- Peculiarities of clinical course of CNS complication caused by CMV;
- Pathogenesis and clinical features of complication in EBV-infection and CMV-infection;
- Conception of persistation in EBV-infection and CMV-infection;
- Laboratory diagnosis of EBV-infection and CMV-infection;
- Treatment principles;
- Prophylaxis principles;
- Tactic of treatment in patients in cases of emergency stages;
- Prognosis of EBV-infection and CMV-infection;
- Indications for hospitalization of patients with EBV-infection and CMV-infection;
- Rules of discharge from hospital;
- Rules of convalescent observation;

**2.2. A student has to perform:**

**a-3**

- to follow the main rules of work with EBV- and CMV- infections;
- to perform anamnesis morbi with estimation of epidemiological information ;
- investigate of patient and to distinguish the main
- to ground clinical diagnosis for timely reference of patient at hospital;
- to work out differential diagnosis EBV- and CMV- infections;
- on the basis of clinical investigation timely to diagnose possible complications and emergency care stages in EBV- and CMV- infections;
- to fill in medical documentation concerning fact of diagnosis “EBV- and CMV-infections” (urgent notification for epidemiology serves);
- to complete plan of laboratory and instrumental investigation;
- to explain a result of laboratory analysis;
- to analyse a result of special diagnostic methods in dependence of material and disease period;



- to prescribe individual plan of treatment under epidemiological data, stage of disease, appearance of complications, severity of disease, accompanied pathology;
- to carry out the first aid on outpatient period;
- to complete a plan of antiepidemic and prophylaxis measures in the nidus of infection;
- to make a recommendation concerning to regimen, diet, investigation, physical activity and acute and convalescence periods.

### 3. Material for auditorium independent work

#### 3.1 Basis knowledge, skills, which are necessary for study of topic (interdisciplinary integration)

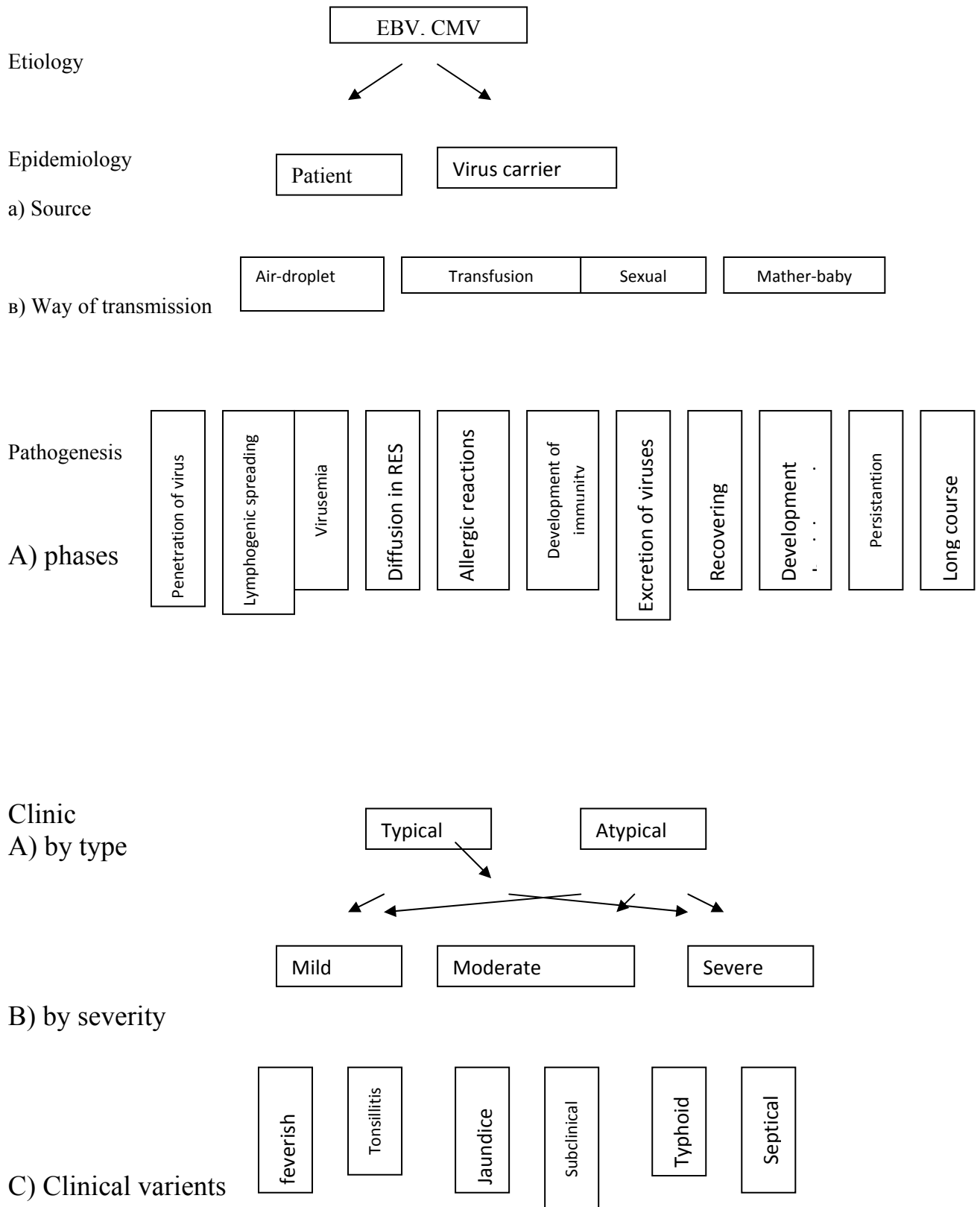
Discipline	To know	To be able
Previous disciplines		
Microbiology	Properties of Herpes simplex I and the II types; methods of specific	To estimate the results of specific methods of diagnostics of CMV EBV infection
Physiology	Parameters of physiological norm of organs and systems of human; indexes of normal values of laboratory tests (general analysis of blood, urine, CSF, biochemical blood test, parameters of acid-alkaline balance, electrolytes, etc.).	To estimate information of laboratory tests
Pathophysiology	Mechanism of disturbance of functions of different organs and systems in pathological states of	To assay pathological changes by results of laboratory tests at disturbances of functions of organs and systems of different
Immunology and allergology	Basic concepts of the object, role of the immune system in infectious process, influencing for the term of elimination of pathogen from the human organism. Immunological	To estimate data of immunological tests.

Epidemiology	Epidemic process (source, mechanism and ways of transmission) of CMV EBV infection; prevalence of pathology in Ukraine and in	To collect epidemiological anamnesis, conduct anti epidemic and prophylactic measures in a pesthole of CMV EBV infection.
Dermatology	Pathogenesis, clinical characteristic of exanthema and enanthema	To recognize rash in patient with herpetic infection.
Neurology	Pathogenesis and clinical signs of meningitis, meningoencephalitis and	To conduct a clinical examination of patient with affection of nervous system.
Ophthalmology	Pathogenesis and clinical signs of keratitis, conjunctivitis, iridocyclitis, choreorithinitis, uveitis and	To conduct a clinical examination of patient with affection of eyes.
Propaedeutics of therapy	Methods and basic stages of clinical examination of patient.	To collect anamnesis, conduct the clinical examination of patient, show pathological symptoms and syndromes. To
Clinical pharmacology	Pharmacokinetics and pharmacodynamics, side effects of antiviral and supportive drugs.	To prescribe treatment depending on age, individual features of patient, to choose the optimum mode of reception and dose of drug, write recipes.
Reanimation and intensive therapy	Urgent stages: brain edema.	To diagnose in good time and give the first aid
Next disciplines		
Family medicine	Pathogenesis, epidemiology, dynamics of clinical symptoms, possible complications of herpetic infection. Features of clinical course of CMV EBV infection. Principles of prophylaxis and treatment.	To conduct differential diagnostics of illnesses of different genesis with a herpetic infection. To recognize a herpetic infection, its complications; to estimate information of laboratory diagnostics. To hospitalize a patient in good time in
Interdisciplinary integration		

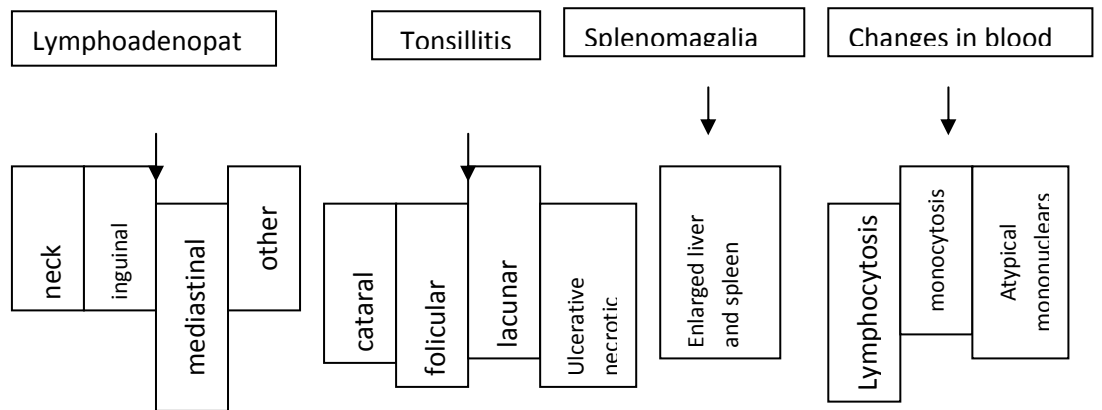
Infectious diseases	Features of infectious diseases. Principles of diagnostics, treatment and prophylaxis of infectious diseases. Pathogenesis, epidemiology, dynamics of clinical symptoms, laboratory diagnostics, possible complications of	To conduct differential diagnostics of herpetic infection with other infectious diseases. To recognize herpetic infection, its complications; to estimate information of laboratory diagnostics. To prescribe treatment. To give the first aid on outpatient stage.
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### 3.2 Contents of lesson topic

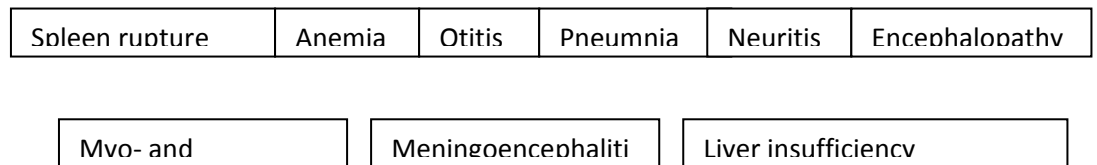
#### EBV-, CMV-infections. Mononucleosis.



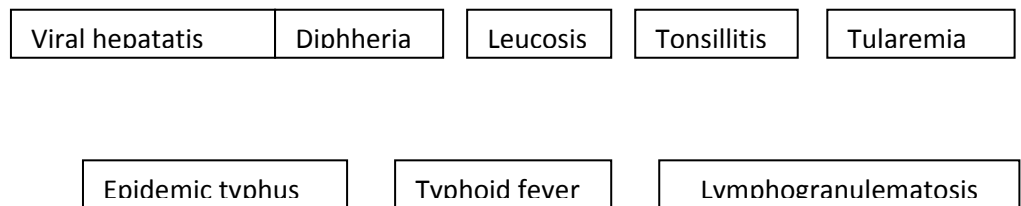
#### Д) The main clinical features



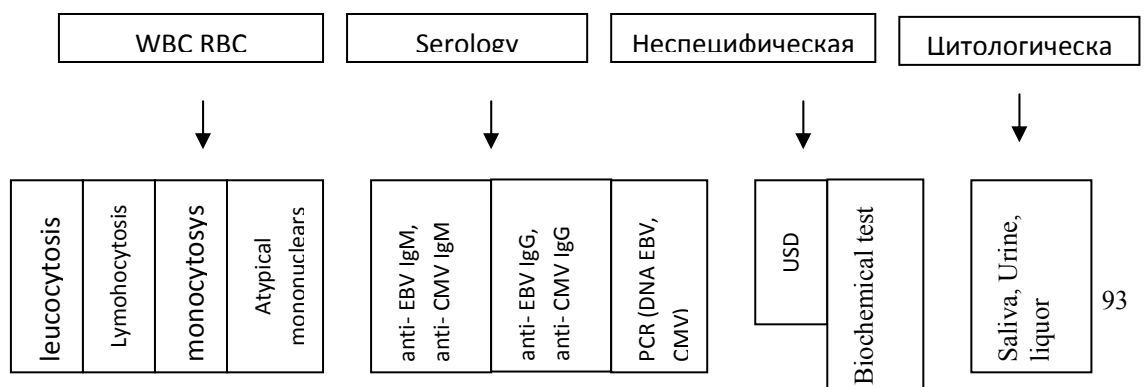
#### E) Complications



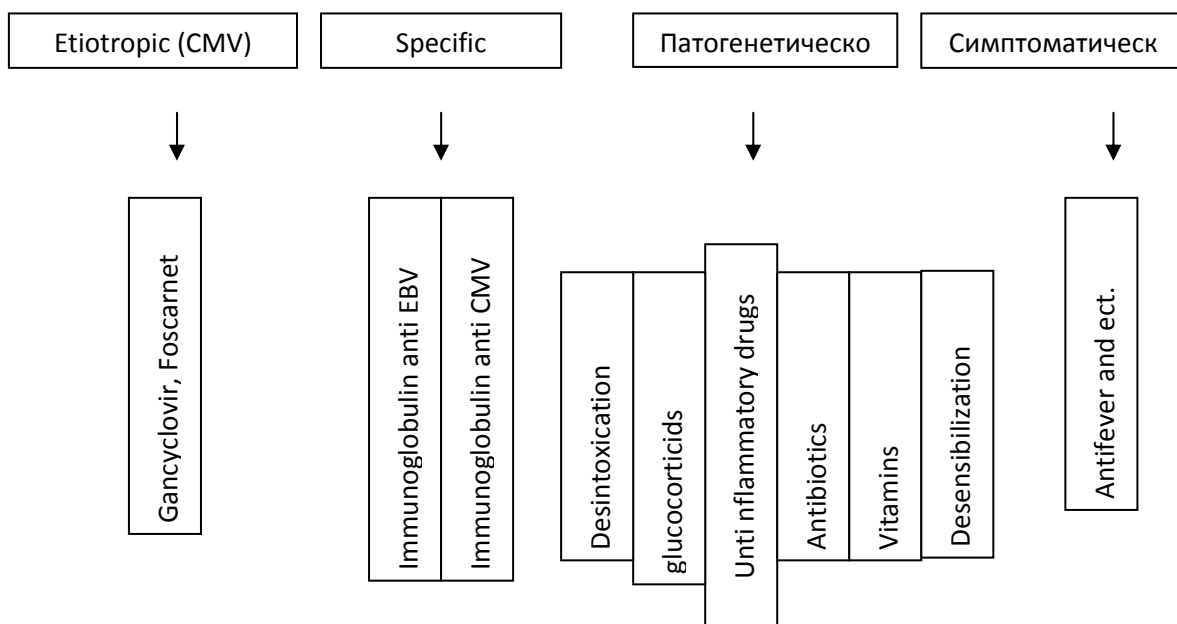
#### Differential diagnosis



#### Laboratory diagnosis



## Treatment



### 3.4.1 Questions for self control of students

1. What group of infection in classification of infectious diseases is EBV and CMV infection by source of infection?
2. What are ways of transmission for EBV and CMV-infections?
3. Characteristics of EBV and CMV?
4. Pathogenesis of EBV-infection?
5. Pathogenesis of CMV-infection?
6. What is clinical classification of EBV-infection?
7. What is clinical classification of CMV-infection?
8. Clinic of EBV infection in dependence of disease form.
9. Clinic of CMV infection in dependence of disease form.
10. What are the main clinical symptoms and syndromes of EBV-infection?
11. What are the main complications of EBV-infection?
12. What are typical complications of CMV-infection?
13. What are peculiarities of EBV and CMV infection in patients with AIDS?

14. Differential diagnosis EBV and CMV infection with other infectious and uninfected diseases.
15. WBC and RBC in patients with EBV infection
16. What kind of changes in peripheral blood on the 2-d weak of diseases is typical in EBV infection?
17. What is a plan of investigation patients with EBV- and CMV-infections?
18. Methods of specific diagnosis EBV and CMV infections. Interpretation of biochemical and serological investigation
19. What changes in immunogram the I and II types are typical in patients with EBV and CMV-infections?
20. The rules of hospitalization for patients.
21. What are principles of pathogenic treatment?
22. Schemes of antiviral therapy. The main ethiotropic medications
23. Indication and prescription of antibiotic in cases of EBV-infection.
24. Principles of prophylaxis of EBV and CMV-infection

#### **3.4.2 Tests for self control of students**

**$\alpha=2$**

1. EBV infects cells:
  - A. T-killers
  - B. B-lymphocytes
  - C. Macrophages
  - D. NK-cells
  - E. T-suppressors
2. Infection transmits by:
  - A. Air-droplets way
  - B. Sexual
  - C. Parenteral
  - D. Contact with animal
  - E. By insect's bites
3. What is average duration of incubation period in cases of mononucleosis:

- A. Some hours
  - B. 1-3 days
  - C. 4-15 days
  - D. Some weeks
  - E. Some month
4. Method of PCR can reveal :
- A. Atypical mononuclears in blood
  - B. Virus in blood
  - C. Antibody to virus antigens in blood
  - D. viral DNA in blood
  - E. Nuclear viral antigens
5. Choice typical exanthema in mononucleosis:
- A. monomorphic exanthema
  - B. vesicular
  - C. Polymorphic
  - D. Appears in all cases of mononucleosis
  - E. Appears in 25-50% cases
6. To find typical changes in WBC and RBC in patients with mononucleosis:
- A. Leucocytosis
  - B. Eosinophilia
  - C. Neutrophil leucocytosis
  - D. Lymphomonocytosis
  - E. Atypical mononuclear cells
7. Typical features in mononucleosis are:
- A. Sore throat
  - B. Stop breathing through a nose
  - C. Generalized lymphadenopathy
  - D. Hepatosplenomegaly
  - E. Fever
8. Typical features in mononucleosis without complications are:



- A. Increased level of creatinin and urea in blood
  - B. In WBC
  - C. In coprocytologic taste
  - D. ALT, bilirubin in blood
  - E. In SCF
9. Does antibiotic necessary in mononucleosis:
- A. Does not prescribe in any case
  - B. Prescribe in any case
  - C. Prescribe in dependence of indication
  - D. Ampicillin should be prescribed
  - E. Ampicillin is contraindicated
- 10 Peculiarities of clinical course of CMV-infection in patients with AIDS.
- A. Latent clinical course
  - B. Usually has mild form
  - C. Localized form often develops
  - D. Has long, relapsing, progressive course
  - E. Severe, generalized form develops

Right answers

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

**To fill up**

$\alpha=3$

**Treatment of infectious mononucleosis in dependence of disease severity**

Level of disease severity	mild	moderate	severe
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Medication			
Penicillin	-	+	+
Tetracyclin	-	-	-
Ampicillin	-	-	-
Chloramphenicol	-	-	-
Sulfanamids	-	-	-
Antiinflammation drugs	-	+	+
Steroids	-	-	+
Immunomodulators	-	-	+
Symptomatic therapy	+	+	+

### Task 1

$\alpha=2$

Patient, 19, was referred at a hospital with diagnosis “sore throat” with compliance on headache, general fatigue, pain in throat, increased temperature 37.5-38.5°C. The diseases began 6 day before admission. On examination: temperature – 38.3°C, sclera and skin are yellow. Enlarged all types of lymphatic nodules; 1-3 cm in diameter, tender. Liver is enlarged 2-2.5 cm. Spleen margin is palpated.

1. Make preliminary diagnosis?
2. What is a plan of investigation?
3. Prescribe a treatment for the patient?

### Task 2

$\alpha=2$

Patient C, 18, came to clinic on the 8-th day of diseases with complains of general malaise, headache, sore throat in swallowing, increased temperature, and sweating. Disease had begun from fatigue, subfebrile temperature, sore throat.

On examination: Temperature 37,7°C, pale skin, rash is absent. Tonsils are enlarged without pus. Palpation reveals enlarged submaxillar, posterior and anterior cervical, axillary lymphatic nodes in size 0,8×1 cm, which is painful and movable. Heart beat sound is clear, Ps – 94 b/min. In lungs – vesicular respiration. Abdomen – painless. Liver enlarged +1.0 cm. Spleen is palpated.

1. Make preliminary diagnosis?
2. What is a plan of investigation?
3. Prescribe a treatment for the patient?

### **Task 3**

**$\alpha=3$**

Patient, 21, was admission at a hospital on the 8<sup>th</sup> day of disease. In onset of diseases the patient experienced increased temperature 37,5-38,5°C, moderate headache, fatigue, pain in chest. In following days blocking nose breathing, abundant rose rash on trunk and extremities appeared. Patient did not keep regiment and take any medication. He drank alcohol. On the 7<sup>th</sup> day of disease severe headache developed, vomiting and disturbance appeared. On examination: temperature – 39,5°C, snuffling voice. Hyperemia of mucous tissue of throat and tonsils. There is membrane on tonsils. Posterior cervical, occipital, axillar, lymphatic nodules are palpated. Liver is enlarged 2-2.5 cm. Spleen margin is palpated. Rigid neck, positive Kernig symptom, strabismus, paresis of left hand.

1. Make preliminary diagnosis?
2. What is a plan of investigation?
3. Prescribe a treatment for the patient?

## **4. Materials for auditorium independent work.**

### **4.1 The list study and practical tasks, which are necessary performed in practical lesson:**

- To study methodic of examination patients with EBV and CMV infectious;
- To perform examination patient with EBV and CMV infectious;
- To make differential diagnosis EBV and CMV infectious;
- To estimate results of lab test in patients with EBV and CMV infectious;
- To diagnose possible complications in cases of EBV and CMV infectious;
- To plan treatment of patients with EBV and CMV infectious;
- To fill up medical documentation

## 4.2 The professional algorithm of the practical skills formation and the diagnostic skills of EBV- CMV - infections

№	Task	Performance sequence	Remarks, warnings as for the self-control
1.	To study the methods of clinical examination of the patients with EBV- CMV - infection	<p>I. To find out a patient's complaints</p> <p>II. To find out anamnesis</p> <p>1. Anamnesis morbi</p>	<p>To select the complaints, which characterize the syndromes</p> <p style="padding-left: 40px;">general intoxication</p> <p style="padding-left: 40px;">organs' affections</p> <p>To pay attention to the beginning (mononucleosis – acute etc.);</p> <p>To draw attention to the possible polymorphism manifestations of mononucleosis – in its process can be acute and gradual.</p> <p>To pay attention on term, time of appearing, dynamic</p> <p style="padding-left: 40px;">eruption</p> <p style="padding-left: 40px;">fever</p> <p style="padding-left: 40px;">pain syndrome</p> <p style="padding-left: 40px;">increasing of the lymphatic nodes</p>
2.	To supervise a patient	<p>2. Anamnesis vitae</p> <p>3. Epidanamnesis</p>	<p style="padding-left: 40px;">pharyngitis</p> <p style="padding-left: 40px;">tonsillitis</p> <p style="padding-left: 40px;">other symptoms</p> <p>To find out the previous diseases (especially followed by immune deficiency). To ask about pregnancy (in case of a</p>

		<p>III. To perform an objective examination</p> <p>General examination common condition of the patient</p> <p>skin and mucous tissue</p> <p>lymphatic nodes</p> <p>Digestive system</p>	<p>positive answer, to find out which term.)</p> <p>To find out the data as for the fact of contact, genital, airborne, percutaneous, placental mechanism of transmission, to pay attention to the contacts to a mononucleosis and flu patient.</p> <p>To remember: presence, expression, symptoms' dynamic are conditioned by a term and an intensity of the process of the disease, its form and depend on the age of the patient and concomitant pathology.</p> <p>To pay attention to:</p> <p>temperature pain syndrome eruption (taking into account the hair covered part of the head, conjunctivitis, mucous coats of the oral cavity) their localization (along the nerve trunks etc.), eruption character and the terms of its appearance itch their possible increasing</p>
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		<p>tongue examination</p> <p>belly percussion</p> <p>belly palpation</p> <p>stool characterization</p> <p>Cardiovascular system:</p> <p>pulse</p> <p>arterial tension</p> <p>heart auscultation</p> <p>Respiratory system</p> <p>lungs auscultation</p> <p>Nervous system</p> <p>Genitourinary apparatus</p>	<p>To pay attention to:</p> <p>characteristic of lymphadenopathy eruption</p> <p>hepatolienal syndrome (by the general forms)</p> <p>To pay attention to:</p> <p>pulse matches the temperature</p> <p>there is a tendention to hypotension</p> <p>does not exclude the development miocarditis and endocarditis</p> <p>To pay attention to:</p> <p>the symptoms of the pneumonia among the part of the patients, especially by adults (dry and moist rales etc.)</p> <p>To pay attention to:</p> <p>encephalitis, meningitis, myelitis, ganglialitis, neuritis. neuralgia, arachnoiditis</p> <p>development possibilities</p>
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3.	To prescribe laboratory additional researches, interpret the results.	<p>Complete blood count</p> <p>Complete urine count</p> <p>Roentgenogram of the chest</p> <p>Fluorescing antibodies reaction</p> <p>Serum researches: complement fixation test, indirect hemoagglutination reaction, radioimmune analysis.</p> <p>Cerebrospinal fluid (by having the meningeal)</p>	<p>To pay attention to: abortion</p> <p>To pay attention to: leucopenia, lymphocytosis, atypical mononuclears, normal ESR</p> <p>Absence of the considerable changes by the typical process</p> <p>The picture looks like miliar tuberculosis</p> <p>Lets discharging an antigen Varicella zoster virus</p> <p>Taken twice: in the acute period of the disease and in the recovery period (in 7-10 days), the reaction is considered to be a positive one by the increasing of the titre of the antibodies not less than in 4 times more (by chicken pox)</p>
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		<p>Microscopy of the smear from the eruption elements</p> <p>Immunoenzyme analysis</p> <p>PCR</p> <p>Immunoenzyme reaction</p>	<p>Is usually serosum, with a normal increasing of the protein content and the lymphocytes quantity, reducing Na and glucose quantity</p> <p>Found the polynuclear cells with eosinophils inclusions in the nucleus, characted for the infection EBV- CMV</p> <p>The determination of the antibodies of the Ig M classification towards the antigens of EBV - CMV - by an acute infection, Ig G by the chronic relapsing process Or antigens VCA EBNA for EBV</p> <p>for the determination of EBV, CMV DNA</p> <p>for the determination of the antigens of the EBV CMV in the</p>
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			secretion from vesicles
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## **5. Materials for extra auditorium independent work**

### **Topics for review reports**

- Modern methods of specific diagnosis of EBV and CMV infectious.
- Problems of treatment patients with EBV and CMV infectious and frequent relapses
- Immunological disturbances in patients with EBV and CMV infectious and their correction
- CMV-infection, as composition of TORCH-infection

### Tests for self control

1. Specify the biochemical indices, which indicates the increase in the development of cytolytic syndrome in patients with viral hepatitis:

- A. \* alanine aminotransferase,
- B. alkaline phosphatase,
- C. thymol test,
- D. total bilirubin,
- E. prothrombin index

2. Synthetic liver function reflects the following indicators:

- A. The level of aspartate aminotransferase
- B. total bilirubin level
- C. level of alanine aminotransferase
- D. \* level of prothrombin index
- E. alkaline phosphatase level

3. The main laboratory parameters cholestatic syndrome is the increase in blood

- A. direct bilirubin
- B. the level of bile acids
- C. alkaline phosphatase
- D. cholesterol
- E. \* All of the above is true

4. Jaundice in viral hepatitis:

- A. obstructive
- B. \* parenchymal
- C. above the hepatic
- D. hemolytic
- E. mechanical

5. For astenovegetativnogo syndrome predzheltushnogo period of viral hepatitis is characterized by:

- A. general weakness
- B. Malais
- C. decreased performance
- D. Fatigue
- E. \* all right

6. For dyspeptic version predzheltushnogo period of viral hepatitis is not typical:

- A. \* fever, chills
- B. decreased appetite
- C. nausea
- D. heaviness in the epigastric region
- E. heaviness in the right upper quadrant

7. For post-exposure prophylaxis of viral hepatitis A are used:

- A. interferon
- B. horse serum
- C. acyclovir
- D. penicillin
- E. \* Immunoglobulin

8. Artralgichesky syndrome predzheltushnogo period of viral hepatitis manifested such symptoms:

- A. restriction of joint movement
- B. hyperemia and swelling of the skin around the joints
- C. \* joint pain
- D. Violations walk
- E. all true

9. Specify the symptoms of influenza-like variant predzheltushnogo period of viral hepatitis:

- A. \* acute onset, fever, body aches, headache, dry cough, nasal congestion
- B. decreased appetite

C. plentiful rhinitis, weakness, normal body temperature

D. fever, laryngitis

E. conjunctivitis, headache

10. Specify a criterion that indicates the severity of viral hepatitis:

A. increasing intoxication, nausea, vomiting

B. decrease in prothrombin index

C. decrease in total protein

D. decrease in the activity of alanine on a background of increasing total bilirubin

E. \* all right

11. For the cyclic course of viral hepatitis characterized by the following period:

A. incubation

B. predzheltushnogo

C. jaundice

D. period of convalescence

E. \* all right

12. The causative agent of viral hepatitis A is:

A. \* picornavirus RNA

B. RNA herpesvirus

C. RNA flavivirus

D. DNA hepadnavirus

E. RNA adenovirus

13. Specify HAV resistance to heat:

A. \* at T + 1000C dies after 5 minutes

B. by boiling dies after 15 minutes

C. withstands boiling for several hours

D. Boiling does not destroy the virus

E. killed by boiling in 45 minutes

14. The genus Hepatovirus include:

A. pathogen hepatitis E

B. Hepatitis B

- C. Hepatitis A \*
  - D. pathogen hepatitis C
  - E. all of the above is true
15. The source of infection with viral hepatitis A:
- A. rodents
  - B. bird
  - S. pets
  - D. \* people
  - E. all true
16. The transmission mechanism, characteristic of viral hepatitis A:
- A parenteral
  - B. \* fecal-oral
  - C. airborne
  - D. transmissible
  - E. vertical
17. In what period of the disease in patients with hepatitis A is the most active virus isolation in the environment?
- A. The incubation period
  - B. \* at the end of the incubation period and the entire prodrome
  - C. during the height of the disease
  - D. convalescence
  - E. all true
18. Factors of transmission of hepatitis A is all except:
- A. water
  - B. \* blood
  - C. Food Products
  - D. dirty hands
  - E. household items
19. The causative agent of hepatitis E is:
- A. RNA picornavirus

- B. \* RNA gepavirus
  - C. RNA flavivirus
  - D. DNA hepadnavirus
  - E. RNA adenovirus
20. The mechanism of hepatocyte injury during HAV:
- A.\* A direct cytopathic effect of the virus
  - B. immunological processes
  - C. toxic-allergic reaction
  - D. damage due to the intensive synthesis of interferon
  - E. . all true
21. Replication HAV occurs in:
- A. macrophages
  - B. endothelial cells
  - C. \* hepatocytes
  - D. enterocytes
  - E. all true
22. Specify the syndrome, the most characteristic of the period prejaundice HAV:
- A. asthenovegetative
  - B. artralgichesky
  - C. dyspeptic
  - D. \* grippopodobnyy
  - E. mixed
23. Specify the most common form of HAV:
- A. \* anicteric
  - B. icteric
  - C. cholestatic
  - D. E. Fulminant
  - E. all right

24. In a patient with jaundice and general weakness prodromal period proceeded by flu-like variant. diagnosis:

- A. jaundice
- B. Toxic hepatitis
- C. \* viral hepatitis A

- D. leptospirosis
- E. Gilbert's syndrome

25. Viral hepatitis in the icteric period to differentiate:

- A hemolytic anemia
- B. infectious mononucleosis
- C. Toxic hepatitis
- D. leptospirosis
- E. \* all true

26. B. 20 years old, entered the 7th day of illness complaints single vomiting, loss of appetite. Three weeks ago with friends enjoyed the same dishes. On-but: skin and sclera icteric, the liver + 2 cm, dark urine, feces aholichny. The diagnosis?

- \* A viral hepatitis A
- B. leptospirosis
- C. intestinal yersiniosis
- D. hemolytic anemia
- E. infectious mononucleosis

27. A woman of 22 years, 7 months pregnant acutely ill 3 weeks after the arrival of Turkmenistan. After three days of fever, jaundice appeared, uncontrollable vomiting, pain in the right upper quadrant, lethargy. diagnosis:

- \* A virus hepatitis E
- B. acute fatty liver of pregnant
- C. cholestatic hepatotoxicity pregnant
- D. hepatitis A
- E. severe sepsis

28. What will develop immunity after suffering HEV:

- A. is not formed
  - B. short
  - C. type specific antitoxic
  - D. type-specific antibacterial
  - E. \* long lasting
4. With what disease
29. it is necessary to differentiate viral hepatitis A in predzheltushnogo period:
- A. \* influenza and other acute respiratory infections
  - B. polio
  - C. Malaria
  - D. typhoid
  - E. all true
30. Enter the reason that causes the darkening of the urine in viral hepatitis:
- A. proteinuria
  - B. increase in indirect bilirubin in the blood
  - C. hemolysis
  - D. \* increase in direct bilirubin in the blood
  - E. Oliguria
31. Complications such as hemolysis and acute renal failure, are characteristic
- A. Hepatitis A
  - B. \* Hepatitis E
  - C. Hepatitis B
  - D. Hepatitis C
  - E. all true
32. The patient blood detected anti - HAV IgM, anti - HBc IgG, anti - HBs. The diagnosis?
- A. \* acute hepatitis A, hepatitis B history
  - B. hepatitis B
  - C. Hepatitis C
  - D. hepatitis A, hepatitis C history



E. Acute hepatitis B

33. In the event of a marker can establish the diagnosis of acute hepatitis E:

A. \* RNA and HEV IgM HEV

B. IgG HEV

C. HAV RNA

D. IgM HAV

E. HAV IgG

34. For specific diagnosis of HAV used:

A virus isolation in chicken embryos

B. biological method

C. agglutination

D. \* immunosorbent assay

E. compliment fixation

35. Hepatitis HAV is found in:

A. saliva

B. sperm

S. urine

D. \* faeces

E. all true

36. Contact person with hepatitis A patients undergo clinical observation and biochemical examination within:

A. 7 days

B. 14 days

C. 21 days

D. \* 35 days

E. 60 days

37. Patient 18, complains of pain in the right hypochondrium, bitter taste in the mouth, jaundice. In the blood, mild leukopenia, defined Ig M anti-HEV. diagnosis:

\* A virus hepatitis E

B. leptospirosis

C. infectious mononucleosis

D. jaundice

E. viral hepatitis A

38. B. 18 found hepatomegaly, hyperbilirubinemia, elevated alanine aminotransferase determined Ig M anti-HAV

A hepatitis E

B. yellow fever

C. infectious mononucleosis

D. jaundice

E. \* viral hepatitis A

39. donor immunoglobulin for the prevention of hepatitis A contains

A. \* anti HAV IgG

B. anti HAV IgM

C. anti HbsAg

D. anti HbeAg

E. anti HEV IgG

40. B. 22 years old, complains of pain in the right hypochondrium, bitter taste in the mouth, jaundice. In the blood, moderate leukopenia, increased cholesterol levels, alkaline phosphatase determined Ig M anti HAV. Your diagnosis?

\* A viral hepatitis A

B. leptospirosis

C. infectious mononucleosis

D. jaundice

E. hepatitis E

41. B. 20 years, with complaints of weakness, anorexia, heartburn, nausea periodically. Considers herself sick about a week. A month ago, the family was sick with hepatitis child of five. What research be carried out to confirm the diagnosis?

A. HbsAg

B. \* anti-HAV IgM

C. liver function tests

- D. fibrogastroscopy
  - E. Ultrasonography of the gastrointestinal tract
42. Detection of blood donor anti HAV IgG indicates:
- A. Acute gepatiet E
  - B Acute hepatitis. A
  - C. Acute hepatitis B
  - D.A chronic hepatitis
  - E. \* a history of acute hepatitis A
43. A specific diagnosis of hepatitis E is based on the identification of
- A. \* anti - HEV IgM
  - B. increasing bilirubin
  - C. . increase tymol test
  - D. increase the activity of alanine aminotransferase
  - E. presence urobilinuria
44. Criteria dichgadge hospital in viral hepatitis A:
- A clinical recovery, but not before 21 days after the onset of jaundice
  - B. increase in the activity of alanine aminotransferase to 3 – standards
  - C. normalization of total bilirubin
  - D. normalization of liver size
  - E. \* all right
45. Clinical supervision for hepatitis A recover set for the term
- A. 2 weeks
  - B. 21 days
  - C. 2 months
  - D. 3 months
  - E. \* 6 months
46. The primary method of treatment of viral hepatitis A, within easy
- A. \* symptomatic
  - B. pathogenetic
  - C. immunomodulating

D. antiviral

E. antibacterial

47. Patient R., viral hepatitis A, moderate flow. Concerned about nausea, weakness, loss of appetite. What drug should be used for the correction of these symptoms:

A. colloid infusion therapy

B. glucocorticoids

C. interferon

D. Vitamin group "B"

E. \* sorbents

48. Treatment of viral hepatitis E:

\* A basic therapy (diet №5, mode, drinking plenty of fluids, sorbents, enzymes)

B. antibiotic therapy

C. immunomodulators

D. antispasmodics

E. glucocorticoids

49. Which of the drugs should be used with intrahepatic cholestasis:

A. \* ursodeoxycholic acid

B. prednisolone

C. essential fosflipidy

D. preparations based on silymarin

E. amino acids

50. For routine prevention of hepatitis A is used:

A. live vaccine

B. \* inactivated vaccines

C. ribavirin

D. interferon

E. acyclovir

51. The causative agent of viral hepatitis B belongs to the family:

A. \* hepadnavirus

B. orthomyxoviruses

C. picornavirus

D. herpesvirus

E. flavivirus

52. DNA - containing a virus is:

A. Hepatitis E virus

B. Hepatitis A virus

C. Hepatitis B virus SEN

D. Hepatitis C virus

E. \* hepatitis B virus

53. For transmission paths HDV belongs:

A. food

B. Water

C. contact-household

D. \* parenteral

E. all true

54. HBV nucleocapsid contains:

A. two-layer shell

B. DNA polymerase

C. DNA HBV

D. HbeAg

E. \* all right

55. The source of infection of viral hepatitis C:

A. rodents

B. \* people

C. fish

D. bird

E. All right

56. DNA HBV can be found in:

- A. blood
- B. urine
- C. saliva
- D. sperm
- E. \* all right

57. The main factor of transmission of hepatitis B:

- A. \* blood
- B. water
- C. vegetables
- D. fruit
- E. all true

58. Source of infection with viral hepatitis B:

- A person with subclinical
- B patients with acute icteric forms
- C patients with chronic
- D. patients with fulminant form
- E.\* all true

59. The most common mode of transmission of hepatitis C is:

- A. \* intravenous drugs
- B. vertical path
- C. food
- D. contact with animals
- E. waterway

60. What are the genotypes of hepatitis C virus are more common in Ukraine:

- A.\* 1, 3
- B. 1, 4
- C. 6, 5
- D. 4, 6

E. 1, 6

61. What is viral hepatitis often leads to the development of liver cirrhosis and hepatocellular carcinoma:

- A. chronic hepatitis D
- B. \* chronic HCV infection
- C. TTV virus
- D. Chronic Hepatitis B
- E. SEN virus

62. The pathogenesis of infection which is predominantly antibody-mediated immune cytolysis of hepatocytes:

- A. viral hepatitis A
- B. \* viral hepatitis B
- C. hepatitis C
- D. hepatitis E
- E. hepatitis G

63. What is the causative agent of viral hepatitis is characterized by the greatest variability in the genome:

- A. \* HCV
- B. HBV
- C. HAV
- D. HEV
- E. HGV

64. Hepatitis B virus replicates mainly in:

- A. T-helper
- B. cholangiocytes
- C. kupffer
- D. \* hepatocytes
- E. B lymphocytes

65. By the end of acute hepatitis B are:

- A recovery
- B. death
- C. Chronic hepatitis
- D. primary cancer
- E. \* all right

66. syndrome, the most characteristic pred jaundice period of hepatitis B:

- A. \* artralgi
- B. mixed
- C. flu
- D. asthenovegetative
- E. all true

67. Patient 19 years old, was admitted on the 4th day of illness. Vomiting, severe weakness, drowsiness, severe jaundice. After one day, the patient is not oriented in time, confused consciousness, intermittent agitation. Reduce the size of the liver. What complications developed:

- A. acute adrenal insufficiency
- B. hypovolemic shock
- C. \*Acute liver failure
- D. Acute hepatic colic
- E. jaundice

68. Reducing the size of the liver at the peak of viral hepatitis B indicates:

- A. cholangitis
- B. Liver abscess
- C. recovery
- D. \* massive hepatic necrosis



E. biliary dyskinesia

69. The symptom most characteristic of the initial stage of liver failure in acute hepatitis B:

A. \* nausea, vomiting, dizziness, feeling of "failure"

B. decreased appetite

C. itching

D. increase in body temperature

E. presence of focal symptoms

70. A woman who for 9 years been an active donor, within 6 months marked ALT - 2.6 mmol / lh. No complaints there. In the blood found anti-HCV. What is characteristic for the further course of the disease:

\* A chronic process in 80-85% of cases

B. chronic process in 10% of cases

C. lack of synchronization

D. frequent development of massive hepatic necrosis

E. presence of unfavorable prognosis if pregnancy occurs

71. Patient 18, was hospitalized with severe viral hepatitis B during the day fell sharply indicators transaminases and hyperbilirubinemia. What complications can occur:

A. electrolyte coma

B. brain coma

C. \* Acute liver failure

D. gastric bleeding

E. jaundice

72. A patient after acute hepatitis B within 6 months of clinical supervision registers allocation blood HBsAg. diagnosis:

- A. acute hepatitis B, prolonged duration
- B. Acute hepatitis B. In phase integration
- C. Acute hepatitis C. In the phase of replication
- D. In the recovery
- E. \* Chronic Hepatitis B

73. B. 28 years, injection drug addict, complains of a dull pain in the right upper quadrant, weakness, loss of appetite, pain in the joints, which are concerned for 2 weeks. On examination, the skin and sclera jaundice, enlarged liver and spleen, the urine is dark, discolored feces. The diagnosis?

- A Toxic hepatitis.
- B HIV
- C. chronic cholecystitis
- D. \* viral hepatitis B
- E. jaundice

74. B. complains of general weakness, a feeling of heaviness in the right upper quadrant. Skin pale - pink eye sclera subicteric. Liver 2.0 cm. The spleen 1.0 cm. In the history-parenteral administration of opiates (last 2 months ago). The diagnosis?

- A\*. Acute hepatitis C
- B. leptospirosis
- C. Malaria
- D. intestinal yersiniosis
- E. Gilbert's syndrome

75. At the doctor's intensive care unit during a routine inspection found increased ALT levels up to 3.4 mmol / L increase in the liver to 2 cm. Bilirubin within normal limits. What is viral hepatitis is most likely the patient:

- \* A viral hepatitis C
- B. Hepatitis B.
- C viral hepatitis A

D. hepatitis D

E. hepatitis E

76. Patient 24 years complaints of weakness, loss of appetite, heaviness in the epigastrium, after 10 days appeared ikteric sclera. OBJECTIVE: pale skin, sclera icteric, the liver + 2.0 cm, 1.0 cm + spleen. In the blood found anti-HCCor IgM, RNA HCV. The diagnosis?

\* A viral hepatitis C

B. Toxic hepatitis

C Hepatitis C.

D. jaundice

E. hepatitis D

77. An early sign of hepatic encephalopathy in viral hepatitis B is:

A. \* inversion of sleep

B. Front bradycardia

C. itchy skin

D. leukopenia

E. hepatomegaly

78. A 20-year-old drug addict appeared weakness, arthralgia, nausea, jaundice after 5 days the skin and sclera, dark urine, pale stools, liver enlargement + 2.0 cm, 1.0 cm + spleen. In the blood -leykotsity -  $3.5 \times 10^9 / L$  lymphocytes - 45%, total bilirubin 180 mg / dL, ALT, 7.2 mmol / tsp, prothrombin index - 76%. The diagnosis?

A. HIV infection

B. yellow fever

C. infectious mononucleosis

D. jaundice

E. \* viral hepatitis B

79. In which viral hepatitis are more common extrahepatic manifestations:

A. \* chronic hepatitis C

B Viral hepatitis A

C. Chronic hepatitis G

D. coinfection B + D

E. chronic hepatitis B

80. On the chronicity of acute hepatitis B indicates the presence of HBsAg in the blood of more than:

A. 1 month

B. 3 months

C. \* 6 months

D. 9 months

E. 1 year

81. Which of these markers is the criterion of early diagnosis of HBV:

A.\* HBsAg

B. anti HBsAg

C. anti HBcAg

D. anti HBcIgG

E. anti HBeAg

82. Persistence of HBeAg in serum for more than 3 months, indicating:

A. carriers of the virus

B. period of convalescence

C. during fulminant

D. \* the risk of chronic hepatitis B

E. all true

83. patients with viral hepatitis B is most contagious when it detects blood:

A.\* HBeAg

B. anti-HBsAg

C. anti-HBeAg

D. anti-HBCor IgG

E. anti-HCCor IgM

84. Name the antigen, which uses the delta - a virus for the completion of its own shell:

A. HBeAg

- B. HBcAg
- C.\* HBsAg
- D. HBxAg
- E. All true

85. Specify a marker that indicates the replication of hepatitis B, and is found in the blood:

- A.\* HBeAg
- B. anti-HBeAg
- C. HBcAg
- D. anti - HBc IgG
- E. anti-HBsAg

86. Specify a marker of viral replication of hepatitis C:

- A. anti - HCV IgG
- B.\* RNA HCV
- C. anti - HCV NS5 IgG
- D.anti - HCV NS3 IgG
- E. anti – HCV

87. For the detection of hepatocellular carcinoma is characterized in serum high content:

- A. cholesterol
- B. iron
- C.\*  $\alpha$ -fetoprotein
- D. triglycerides
- E. Alkaline phosphatase

88. In favor of acute viral hepatitis B indicates the presence of a high concentration in the blood:

- A. HBsAg
- B.\* anti-HBc IgM
- C. anti-HBsAg
- D. anti - HBc IgG

E. anti-HBeAg

89. For the diagnosis of acute HDV infection is a major marker:

A. \* anti-HDAg IgM

B. anti- HDAg IgG

C. anti-HBcIgM

D. DNA HBV

E. HBsAg

90. Which of these funds is not indicated in mild HBV infection:

A. enzymes

B. Diet №5

C. chelators

D. \* glucocorticoids

E. lactulose

91. For the treatment of hepatitis B virus in acute use all, except:

A. \* lamivudine

B. sorbents

C. enzymes

D. hepatoprotectors

E. detoxification therapy

92. A woman of 42 years, three years ago, had hepatitis C. In recent notes weakness ikterict sclera. Indicator ALT 2.0 mmol / lh. Detected HCV - RNA. Determine the most effective means of therapy:

A. ganciclovir

B. prednisolone

C. \* recombinant interferon -  $\alpha$ , ribavirin

D. lamivudine

E. acyclovir

93. A woman 28 years old, suffered a severe form of viral hepatitis B. After 3 months from the onset of the disease was noted the presence of HBV-DNA of high concentration. Determine the most effective means for the prevention of sexual

partner:

\* A. Vaccination against hepatitis B

B. spermicides

C. interferon

D. barrier contraception

E. lamivudine

94. surgeon during the year in the blood is detected HBsAg .. What measures in relation to members of the family of the patient should be taken?

A monthly survey of transaminases

B. Introduction anti-HBV immunoglobulin

C. appointment ribavirin

D. \* vaccine against HBV

E. ultrasonography of the liver and spleen

95. The specific routine prophylaxis of viral hepatitis in the wire:

\* A recombinant vaccine

B. Vaccine Adsorbed

C. toxoid

D. Chemical vaccine

E killed vaccine

96. In carrying out vaccination against hepatitis B in the blood appear:

A.\* anti- HBsAg

B. anti-HBeAg

C. anti –HBcorIgM

D. anti-HBcorIgG

E. All true

97. Specify the minimum protective titer of anti-HBsAg serum

A.0,5 ME/мл

B. 1,0 ME/мл

C. 5 ME/мл

D.\* 10ME/мл

E. 100 ME/мл

98. Observation of the contact persons with viral hepatitis B is set for the term:

A. 35 days

B. 60days

C. \* 180dney

D. 1 year

E. observation is not made

99. hyperimmune immunoglobulin for emergency prevention of hepatitis B virus is introduced:

A. \* within the first 24-48 hours after contact with the patient

B. during the first 14 days after contact with a patient

C. during the first 25 days after contact with a patient

D. During the first 6 months after exposure

E. within the first 35 days after contact with a patient

100. The dose immunoglobulin for the prevention of hepatitis B:

A. 0,01 MI/kg

B. 0,02 MI/kg

C. \* 0,06 MI/kg

D. 0,1 MI/kg

E. 10 MI/kg

101. Spleen laceration at infectious mononucleosis develops

A. After convalescence.

B. on 5 - 7 day of the disease.

C. not characteristic

D. \* On the 2<sup>nd</sup> - 3<sup>rd</sup> week of the disease.

E. In 1 year after convalescence.

102. Patient A., complains about the increase of T body to 38.5°, malaise, headache, sore throat when swallowing, rash. On examination - on the face, trunk, scalp elements abundant polymorphic rash in the form of macules, papules, vesicles. On the soft palate, the bow - isolated vesicles. Diagnosis.



- A. Smallpox
- B. Streptoderma
- C. Herpetic infection
- D. \* Chickenpox
- E. Zoster

103. Glucocorticosteroids administered varicella

- A. In severe disease
- B. \* When developing varicella encephalitis
- C. Do not prescribe
- D. Children and the elderly
- E. In the presence of comorbidity

104. Place fixation varicella virus in the body

- A. lymph nodes of the mesentery
- B. Macrophages
- C. hepatocytes
- D. \* Epithelial cells of the skin and mucous membranes
- E. Langerhans cells

105. A child of 4 years on the background of fever to 39.0 ° C and catarrhal symptoms on day 2 of the disease on the scalp, face, trunk and extremities appeared polymorphic rash, red spots, papules and vesicles with clear fluid. Your diagnosis?

- A. \* Chickenpox
- B. Measles
- S. Rubella
- D. Meningococcal disease
- E. Allergic reaction

106. The causative agent of infectious mononucleosis is

- A. Bacteria
- B. Elementary
- C. Rickettsia
- D. vibrio

E. \* Virus

107. Select the pathogen of infectious mononucleosis

A. \* V.Epshteyn Barr

B. V. Inaba

C. Herpesviridae

D.V. Cholerae

E. V. Parvoviridae

108. A boy of 10 years, has strep throat, in leucogram registered mononuclear cells in an amount of 30%. The diagnosis?

A. Streptococcal infection

B. Herpetic infection

C. \* Infectious mononucleosis

D. Viral Hepatitis

E. Diphtheria

109. Patient K., 19 years old, was admitted to the surgical department with rupture of the spleen. On examination - enlarged lymph nodes, liver. What disease can give a pathology?

Viral hepatitis A.

B. Leptospirosis

C. Diphtheria

D. \* Infectious mononucleosis

E. Salmonellosis

110. Patient N., 18 years old with a diagnosis of infectious mononucleosis complains of pain on swallowing. On examination - enlarged tonsils covered with purulent coating, hepatosplenomegaly. What medications must be assigned in the first place?

A. Vitamins

B. Serum

C. \* Antibiotics

D. vaccine

E. Saluretics

111. The patient K. 17 years, diagnosed with infectious mononucleosis, when viewed - increased lymphocytes, hepatosplenomegaly. What medications must be assigned in the first place?

- A. Antibiotics
- B. \* Corticosteroids
- C. Vitamins
- D. Serum
- E. The vaccine

112. What complication of infectious mononucleosis is typical?

- A. Myocarditis
- B. Renal failure
- C. Pneumonia
- D. Sepsis
- E. \* Infectious mononucleosis

113. What changes are most typical of homeostasis in infectious mononucleosis?

- A. Increased blood pressure
- B. Reduction of blood pressure
- C. Metabolic changes
- D. \* The change of cellular blood
- E. Change in the cellular composition of urine

114. Patient R., 17 years old, with a diagnosis of infectious mononucleosis, there are signs of a ruptured spleen. What is the cause of this complication?

- A. Swelling of the tissues of the spleen
- B. Renal failure
- C. Increased blood pressure
- D. Swelling of brain tissue
- E. \* Hyperplasia of lymphoid tissue

115. Which of the following clinical syndromes determines the severity of infectious mononucleosis

- A. Intoxication

- B. \* gepatolienalny
- C. Syndrome hematological changes
- D. Modification of the T wave on the ECG
- E. Psihoastenicheskie changes

116. Which of the following vaccines used to prevent infectious mononucleosis

- A. DTP
- Influenza B.
- C. The vaccine Sebino
- D. \* No
- E. Measles

117. Specify the mortality of patients with timely treatment infectious mononucleosis

- A. \* 0%
- B. 0.5%
- C. 1%
- D. 2%
- E. 5%

118. A woman of 25 years, there were itching and burning sensation in the lower lip, low-grade T body. On examination - pale pink skin, peripheral lymph nodes were not enlarged, the lower lip swollen, infiltrated with multiple Vesicular eruptions with clear content. Your diagnosis?

- A. Eczema
- B. \* Herpes simplex
- C. Chickenpox
- D. Infiltrate lips
- E. Furuncles

119. Patient I., aged 28, complains of pain in the rectum, mucus when defikatsii, as well as the appearance of itchy sores in the genital area. Your are diagnosis?

- A. \* Genital herpes
- B. Helminthiasis
- C. Furunkullez

D. Eczema

E. Dermatitis

120. Patient M. 36 years, complains of low-grade T body, the appearance of vesicular rash on the chest in 6 intercostal space, severe pain in the area of the rash. As a child suffered chicken pox. What is the causative agent of this disease?

A. Meningococcus

B. Staphylococcus

C. Protozoa

D. \* Herpes Zoster

E. B. antracis

121. A patient of 25 years diagnosed with herpes infection of skin and mucous membranes. The diagnosis was confirmed by PCR. What treatment is recommended to assign?

A. peroxide treated vesicular rash

B. \* Acyclovir in \ in, then orally

C. vidarabine for a month

D. physiotherapy

E. Immunomodulating

122. Patient V., aged 23, student, with increased sexual activity. Fell ill with acute increase of T up to 38 ° C body, muscle pain, weakness. On examination - pale skin, enlarged inguinal lymph nodes, spleen. In the blood of 10% of the mononuclear cells. Reaction Paulo Bunelya negative. What can you suggest a patient?

A. Infectious mononucleosis

Viral hepatitis B.

C. \* CMV

D. Viral Hepatitis

E. Tuberculosis

123. Patient D., 24 years old. Within last month disturb weakness, headache, loss of appetite, insomnia, muscle pain sometimes. He was treated on the subfebrile the

therapist and the urologist, but to no avail. CMV infection was suspected. some studies have confirmed this diagnosis?

- A. A single study RAC
- B. \* Blood, urine, buccal scraping with the PCR method
- C. Immunoblot
- D. Phragmites once
- E. Common an. blood

124. The patient 34 years old with latent CMV infection during the acute illness. Which therapy is necessary to appoint a patient?

- A.  $\alpha$ -interferon
- B. restorative therapy
- C. \* The specific Ig and Cymeven
- D. Diphenhydramine
- E. Analginum

125. Patient D., 43 years old, developed severe pneumonia. The disease is accompanied by high intoxication, fever with symptoms of lymphadenopathy and hepatomegaly. RAC with CMV infection dynamics gave rise to a titer of 4 times. The diagnosis?

- A. CMV
- B. lobar pneumonia
- C. HIV infection
- D. \* Generalized form of CMV infection
- E. Pneumonia

126. Patient Z., 19 years of injecting drug users have stomach pain, epigastric, later appeared dark stools, 1-fold vomiting with blood. There was apathy. Pale skin. When radiography detected ulcer of the esophagus, stomach and colon. What disease should be excluded?

- A. ulcer disease
- B. HIV infection
- C. Hepatitis C virus

D. \* CMV infection, HIV infection

E. Viral hepatitis B

127. Patient N., 17 years old, a drug addict, was admitted to the hospital with complaints of weakness, malaise, low-grade temperature, yellowing of the skin and sclera. Was suspected viral hepatitis, as elevated ALT, bilirubin. Blood markers of viral hepatitis and HIV negative. Your possible diagnosis?

A. Gilbert's syndrome

Alcoholic hepatitis B.

C. Infectious mononucleosis

D. \* CMV hepatitis

E. Leptospirosis

128. At patient with fever to 39,0 °C and the catarrhal phenomena on the 2nd day of disease there appeared a polymorphic rash on the skin of pilar part of head, face, trunk and extremities: red macules, papules and vesicles with a transparent liquid.

The agent of this infection belongs to:

A. \*Herpesviridae of the 3rd type,

B. Herpesviridae of the 2<sup>nd</sup> type,

C. Epstein-Barr virus,

D. Enterovirus,

E. β-Herpesvirus.

129. Glucocorticoids at a chicken-pox are prescribed in such cases like:

A. at the severe clinical course,

B.\* at development of varicella encephalitis,

C – not prescribed,

D - to the children and elderly,

E - at presence of concomitant pathology.

130. Patient A., complains of temperature rise to 38.5°, malaise, a headache painful swallowing, rash. At examination - on the skin of the face, the trunk, the pilar part of the head there are elements of a plentiful polymorphic rash like macules, papules, vesicles. On the soft palate and arches there are the solitary vesicles. The diagnosis.

- A. Smallpox
- B. Streptococcal impetigo
- C. Herpetic infection
- D. \*A chicken pox
- E. Shingles

131. A place of fixing of Varicella-zoster virus in an organism exists in

- A. Mesenteric lymph nodes
- B. Macrophages
- C. Hepatocytes
- D. \*Skin epitheliocytes and mucous membranes
- E. Langerhans cells

132. At the child of 4 years on a background of a fever up to 39,0 °C and catarrhal signs on the 2nd day of disease there appeared the polymorphic rash on the skin of pillar part of the head, the face, a trunk and extremities: red macules, papules and vesicles with a transparent liquid. Your diagnosis?

- A. \*Chicken pox
- B. Measles
- C. German measles (Rubella)
- D. Meningococcosis
- E. Allergic reaction

133. At the patient of 56 years there appeared attacks of a pain in the left half of thorax, general weakness, fever, headache two days ago. At examination: on the course of 4-5 intercostals intervals at the left there are the elements of vesicular rash of 2-4 mm in diameter, filled with the transparent liquid, located on hyperemic and edematous background. The diagnosis.

- A. Chicken pox
- B. Measles
- C. A scarlet fever
- D. \*Herpes zoster
- E. Streptococcal impetigo



134. The patient of 45 years consulted the doctor with complaints on appearance of skin rash, temperature rise up to 37,6°C. At objective examination: there is the isolated polymorphic rash as maculae's, papules, vesicles on the skin of the face, trunk, hands, legs, pillar part of the head. The diagnosis?

- A. Scarlet fever
- B. Measles
- C. Pseudotuberculosis
- D. Enterovirus infection
- E. \*Chicken pox

135. The classic triad of symptoms in infectious mononucleosis

- A. subfebrilitet, pain in the right upper quadrant, ikterichnost skin
- B. normal temperature, myalgia, sore throat
- C. regional lymphadenitis, sore throat, headache
- D. fever, meningeal syndrome, otitis
- E. \* fever, lymphadenopathy, sore throat

136. The occurrence of complications in infectious mononucleosis contributes

- A. \* formation of autoantibodies
- B. primary immunodeficiency
- C. secondary immunodeficiency
- D. Age of patients
- E. presence of chronic diseases

137. gepatolienalny syndrome in infectious mononucleosis is caused

- A. hepatocyte cytolysis
- B. cytopathic effect of the virus
- C. \* infiltration of atypical mononuclear cells, the occurrence of focal necrosis
- D. addition of secondary infection
- E. degeneration of hepatocytes

138. Patient S., aged 9. High fever for 5 days. Complains of pain in the throat, shortness of nasal breathing. Sluggish. In the oropharynx - the tonsils swollen, almost

merge on line Wednesday, gaps are filled with pus. There is an increase in all groups of lymph nodes, hepatolienalny syndrome. In liver samples total bilirubin increased in 3 times, ALT - 4 times. Your diagnosis.

Viral hepatitis A.

B. leptospirosis

C. \* infectious mononucleosis

D. lacunar tonsillitis

E. flu

139. The rash in infectious mononucleosis

A. mottled papuloleznaya

B. haemorrhagic

C. urticaria

D. \* polymorphic

E. absent

140. The characteristic changes in the blood in infectious mononucleosis

A. \* stab neytrofillez, limfomonotsitoz, the presence of atypical mononuclear not less than 10%

B. stab neytrofillez, limfomonotsitoz, the presence of atypical mononuclear less than 10%

C. neytrofillez, lymphopenia, the presence of atypical mononuclear cells less than 10%

D. neutropenia, lymphocytosis, absence of atypical mononuclear cells

E. absent

141. Changes in the oropharynx in infectious mononucleosis

A. \* posterior pharyngeal wall mucosa hyperemic, granular, edematous, hypertrophied follicles. The tonsils are enlarged, friable, easily covered with whitish coating

B. moderate hyperemia of the mucosa of the oropharynx with cyanotic hue, tonsils swollen and covered with a smooth, dirty-gray patina with clear edges (+) fabric. When you try to remove the plaque is not removed, the bleeding

C. moderate hyperemia of the oropharynx, tonsils one of the moderately puffiness. In the crater deepening edematous tonsils dirty – gray raid

D. oropharyngeal mucosa bright hyperemic, swollen tonsils, lacunae filled with easily removable yellow pus

E. absent

142. For the laboratory diagnosis of infectious mononucleosis uses all except:

A. \*Bacteriological blood cultures

B. Hoff-Bauer Reaction

C. The reaction of Paul-Bunnelya

D. Total blood

E. ELISA

143. Infectious mononucleosis is characterized by all except:

A. Hepatic Insufficiency

B. Fever

C. tonsillitis

D. Poliadenopatii

E. Hepato-splenic syndrome

144. For a typical infectious mononucleosis is all the above, except

A. Lymphopenia

B. Leukocytosis

C. Neutropenia

D. lymphocytosis with monocytosis

E. The atypical mononuclear cells

145. Indicate where persists herpes zoster

A. \*In the nerve ganglia

B. In the cells of the reticuloendothelial system

C. In erythrocytes

D. In monocytes

E. In eosinophils

146. For the treatment of infectious mononucleosis shows all except:

A. \*immunostimulants

B. Antibiotics

C. Antihistamines

D. Antivirals

E. Glucocorticoids

147. CMV infection is characteristic:

A. \*Formation of giant cells

B. Reduction T cells

C. Reduction of B-cells

D. Thrombocytosis

E. Neutropenia

148. Please indicate what changes are characteristic of infectious mononucleosis

A. \*Generalized lymphadenopathy

B. Lymph nodes are tight and painful

C. Lymph nodes are soldered with subcutaneous fat

D. The skin over the lymph nodes hyperemic

E. All of the above is true

149. Specify the clinical signs of herpes zoster

A. All of the above is true

B. pain syndrome

C. Ganglionevrit

D. \*Grouped vesicles on the basis of the infiltrated

E. Fever

150. The patients have fever up to 39.0 ° C and catarrhal symptoms, on the 2nd day of the disease on the scalp, face, trunk and extremities appeared polymorphic rash, red spots, papules and vesicles with clear fluid. Specify diagnosis.

A. Chickenpox

B. measles

C. rubella

D. Meningococcal disease

E. allergic reaction.

151. Immunodeficiency virus belongs to:

A paramyxovirus;

B \* - Retroviruses;

C- herpesviruses;

D- flaviviruses;

E arboviruses.

152. How many structural genes has immunodeficiency virus:

A one;

B two;

C \* - three;

D- five;

E-eight.

153. HIV genes encoding the envelope glycoproteins include:

A p17;

B p24,

C- endonuclease;

D \* - gp 120;

E all of the above is true.

154. HIV genes encoding internal proteins of education include:

A \* - p24;

B gp120;

C- gp41;

D- endonuclease;

E all of the above is true.

155. HIV 1 envelope glycoproteins structurally subdivided into subtypes:

A-A-B;

B-C-B;

C-A-D;

D \* - A-J;

E-A-K.

156. The gene encodes all HIV enzymes except:

A- endonuclease;

B. Proteinase;

C.reverse transcriptase;

D \* - cholinesterase;

E- all true.

157.Kontrol for viral replication provides:

A p24;

B gp120;

C \* - gene «tat»;

D- endonuclease;

E p17.

158.Virus inactivated for 1 minute with:

\* A -boiled;

B ultraviolet irradiation;

C-ionizing radiation;

D- freezing;

E- all true.

159.Slow infections include:

A malaria;

B encephalitis;

C \* - HIV infection;

D- Lyme disease;

E all of the above is true.

160.HIV infection refers to diseases:

A \* - anthroponotic;

B- zooantroponoznym;

C- sapronotic;

D- zoonotic;

E endemic.

161. HIV-infected virus is contained in:

A blood;

B- semen;

C- vaginal secretions;

D- saliva;

E \* - all true.

162. The most amount of virus in HIV-infected patients is contained in:

A \* - semen;

B- sweat;

C- tears;

D- breast milk;

E- spinal fluid.

163. Mechanism immunodeficiency virus transmission:

A \* - pin;

B- airborne;

C- transmissible;

D- fecal-oral;

E- all true.

164. Hemokontak route of HIV transmission is possible when:

A blood transfusion;

B red cells;

C- infected transplant organs;

D- parenteral manipulations;

E \* - all true.

165. Antigenic complex CD4 + are:

A- Langerhans cells;

B cells oligodendroglial;

C- alveolar macrophages;

D- T Helpers;

E \* - all true.

166. Virus immunodeficiency may penetrate:

A monocytes;

B- macrophages;

C glial cells of the brain;

D- T Helpers;

E \* - all of the above is true.

167. HIV has a direct cytopathic effect on

A- cardiocytes;

B- hepatocytes;

C \* - cells of the nervous system;

D- nephrocytes;

E all of the above is true.

168. In healthy person helper/suppressor index is equal to:

A-0.3;

B-0.5;

C-1.0;

D \* - 1,7;

E 3.0.

169. In the construction of DNA -code immunodeficiency virus involved:

A- endonuclease;

B \* - reverse transcriptase;

C-phosphatase;

D- cholinesterase;

E- all true.

170. Virus integrates viral DNA code into the host cell genome:

A \* - endonuclease;

B- reverse transcriptase;

C-phosphatase;

D- cholinesterase;



E- all true.

171. HIV striking:

A- endothelial cells of the vascular plexus of the brain;

B reduces the production of neuropeptides - hormone-epiphyseal gipotalemicheskogo complex;

C has a cytopathic effect on neural cells;

D- T Helpers;

E \* - all of the above is true.

172. Immunodeficiency in HIV infection is caused by:

A activation of infected helper;

B- syncytium formation;

C- autoaggression;

D- death of healthy helper CD4 on the shell which merged with gp120;

E \* - all true.

173. Synchronicity HIV - infection is formed by:

A \* - capture HIV-infected healthy helper helpers;

B activation of T-killers;

C decrease the number of T helper cells;

D- autoaggression;

E- polyclonal activation of antibodies.

174. Immunoregulatory index of CD4 + / CD8 + in the acute phase of HIV reduced by:

A decrease in CD4 lymphocytes;

B reducing the amount of CD8 lymphocytes;

C increase in CD4 lymphocytes;

D \* - increasing the number of CD8 lymphocytes;

E- increase the number of plasma cells.

175. Immunoregulatory index CD4 / CD8 in the terminal phase of HIV reduced by:

A \* - reduce the number of CD4-cells;

B increasing amounts CD4-cells;

C increase the amount CD8-cells;

D- reduce the amount CD8-cells;

E- reduce the number of plasma cells.

176. HIV infected appeared complaints fever, headache, vomiting. Positive meningeal signs (Kernig's sign, Brudzinskogo). Cerebrospinal fluid is transparent cell count of 30 cells by lymphocytes. In blood CD4 lymphocyte counts of 550 cells / mm. Stage of HIV infection:

A \* - acute stage;

B latency,

C DIGE;

D- AIDS-related complex;

E AIDS.

177. Bolnoy for 5 weeks fever T-37,5-38,0 °. Complaints of pain in the throat. The tonsils are hypertrophied, enlarged submandibular, zadnesheynyh, subclavian, inguinal lymph nodes. Hepatosplenomegaly. Immunoblotting revealed antibodies to HIV-1 in CD4 lymphocyte immunological - 520 cells / mm. Determine the stage of HIV infection:

A primary latent period;

B- secondary latent period;

C \* - generalized lymphadenopathy;

D- AIDS-related complex;

E AIDS

178. The stage of persistent generalized lymphadenopathy in HIV can be diagnosed with the condition:

A patient complaints at elevated temperature, sweating;

B- an increase of at least 2 groups of lymph nodes (excluding inguinal)

C- presence of the CD4 lymphocytes in an amount not less than 500 cells / mm;

D- possible increase in liver:

E \* - all of the above is true.

179. Patient for 3 weeks low-grade fever, fatigue, abdominal pain, increased stool 8-10 times a day, weight loss. PCR - HIV RNA. In immunological CD4 lymphocytes - 150 cells / mm. CD8 lymphocytes - 150 cells / mm. Diagnosis:

A acute stage of HIV infection;

B- secondary latency;

C- HFRS;

D- AIDS related complex;

E \* - AIDS.

180. Lokal Kaposi's sarcoma in HIV-infected observed in the period:

A secondary latency;

B- acute stage;

C- with generalized lymphadenopathy;

D \* - AIDS-related complex;

E AIDS.

181. Etiology of PCP are:

A mycoplasma;

B \* - yeast-fungus;

C- chlamydia;

D- simple;

E rickettsiae.

182. Source of infection are infected with Pneumocystis carinii pneumonia:

A rodents;

B \* is a man of;

C pigs;

D- birds;

E- all true.

183. Mechanism of transmission pneumocystis pneumonia:

A pin;

B- fecal-oral;

C \* - airborne;

D- transmissible;

E- all true.

184. For the treatment of Pneumocystis carinii pneumonia in AIDS patients use:

A Biseptol;

B- clindamycin;

C- pentamidine;

D- dapson-trimetaprim;

E \* - all true.

185. The diagnosis of AIDS is entitled under:

A \* - candidiasis of the esophagus, bronchus, lung;

B- candidiasis colon;

C- vulvovaginal candidiasis;

D- streptoderma;

E- all true.

186. Dlya treatment of candidiasis can be used:

A miconazole;

B- ketoconazole;

C- fluconazole;

D- amphotericin B;

E \* - all true.

187. Etiology of cryptococcosis are:

A- bacteria;

B- rickettsiae;

C \* - mushrooms;

D- simple;

E viruses.

188. Osnovnoy transfer mechanism with cryptococcosis:

A pin;

B \* - airborne dust;

C- transmissible;

D- transplacental;

E intrapartum.

189. In the treatment of cryptosporidiosis are used:

A \* - azithromycin;

B- acyclovir;

C- fluconazole;

D- ganciclovir;

E foscarnet.

190. Etiology toxoplasmosis are:

A viruses;

B bacterium;

C- rickettsiae;

D \* - simple;

E fungi.

191. Prevention of cytomegalovirus infection in HIV-infected held at keeping blood CD4-lymphocytes in number:

A \* - 100 cells / mm;

B- 300 cells / mm;

C-400 cells / mm;

D-500 cells / mm;

E- all true.

192. For treatment cytomegalovirus infection used:

A \* - ganciclovir;

B- dapsone;

C- pyrimethamine;

D- fluconazole;

E amphotericin.

193. Diagnosis HIV infection laboratory confirmed the presence in the blood of antibodies by ELISA:

A gp120;

B- gp 41;

C \* - immunoblotting;

D- immunogram;

E- all true.

194. Diagnosis HIV infection can be confirmed by laboratory:

A PCR - detection of HIV RNA;

B PCR - detection of HIV proviral DNA;

C-ELISA - definition of antibodies;

D- ELISA - definition of fragments of the virus;

E \* - all true.

195. Methods of laboratory diagnosis of HIV infection, confirming the stage of the disease is:

A CD8 lymphocytes;

B \* - CD4 lymphocytes;

C  $\beta$  cells (CD19 +);

D- immunoglobulins;

E- all true.

196. Nucleoside reverse transcriptase inhibitors (NRTIs) are:

A- efavirenz;

B \* - zidovudine;

C- nevirapine;

D- lopinavir;

E- all true.

197. Non-nucleoside reverse transcriptase inhibitors (NNRTIs) are:

\* A -ifavirents;

B- zidovudine;

C- lamivudine;

D- lopinavir;

E- all true.

198. Protease inhibitors (PIs) are:

- A- efavirenz;
- B \* - lotenavir;
- C- zidovudine;
- D- lamivudine;
- E- all true.

199. Antiretroviral therapy is indicated for HIV-positive:

- A acute stage of HIV infection
- B the presence of HIV RNA greater than 100,000 copies / mL;
- C- generalized mycobacteriosis;
- D. PCP;
- E \* - all true.

200. Prior initial examination of HIV-infected should be investigated:

- A general analysis of blood, urine;
- B- biochemical parameters (bilirubin, ALT, creatinine, blood urea, sugar, protein and albumin levels);
- C- cal for helminth eggs and protozoa;
- D- complex CD4 lymphocytes;
- E \* - all true.

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