INTRODUCTION TO INFECTOLOGY.
INFECTIONS DISEASES WITH FECAI-ORAL TRANSMISSION

MANUAL FOR PRACTICAL TRAINING AND INDEPENDENT WORK

Of students for the 5th year of the Medical Faculty
On Infectious Diseases to the module №1)

2014

Manual for practical training and independent work of students for the 5th year of the Medical Faculty on Infectious Diseases to the Infections diseases with fecal-oral transmission. In manual aid theoretical bases of infectious diseases are expounded with the fecal-oral mechanism of transmission. The questions of etiology, epidemiology, classification of pathogenetic mechanisms, clinics, diagnostics, differential diagnostics, treatment and prophylaxis of intestinal diseases are presented in every part of manuals. In every division theoretical part is presented by charts, algorithms, tables.

A manual contains methodical recommendations for independent preparation of students to practical employments, control questions, situational tasks and tests with the standards of answers to every theme of practical lessons. This manual was written for the medical student and presumes a basic knowledge of biology, biochemistry, immunology, therapy, surgery.
Introduction

Manual for practical training and independent work of students for the 5th year of the Medical Faculty on Infectious Diseases to the module №1 «Introduction to infectology. Infections diseases with fecal-oral transmission» it is worked out by the collective of department of infectious diseases of ЗГМУ on the basis of the program of discipline infectious diseases ZSMU for speciality 7.12010001 is curative business, taking into account the requirements of state educational standard.

A manual is presented by 5 divisions and infectious diseases correspond to the 1- th module of discipline. In manual aid theoretical bases of infectious diseases are expounded with the fecal-oral mechanism of transmission. The questions of etiology, epidemiology, classification of pathogenetic mechanisms, clinics, diagnostics, differential diagnostics, treatment and prophylaxis of intestinal diseases are presented in every part of manuals. In every division theoretical part is presented by charts, algorithms, tables.

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It is recommended, as manual for the students-foreigners of higher educational medical establishments of IV of levels of accreditation.
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TYPHOID FEVER. PARATYPHOID A. PARATYPHOID B

1. Actuality of theme

Typhoid fever is caused by a Gram-negative organism Salmonella enterica - either serovar Typhi (S. typhi) or serovar Paratyphi (S. paratyphi). The latter (paratyphoid) is divided into three subtypes (A, B and C) and is generally similar but usually less severe. Typhoid fever is common, but it is most widely spread in African, South American and Asian countries. Each year about 33 million people have typhoid fever. Spread of the disease is caused by the absence of effective methods of sanation of S. typhi carriers, which are the main source of infection, resistance of agents in the environment, fecal and oral transmission mechanism, tendency of the disease for epidemic spread, especially in the regions with low social, hygiene and sanitary level, in the context ecological problems. About 3% of untreated patients, referred to as chronic enteric carriers, harbor organisms in their gallbladder and shed them in stool for > 1 yr. Some carriers have no history of clinical illness. Epidemiologic data indicate that typhoid carriers are more likely than the general population to develop hepatobiliary cancer.

Epidemiology. Typhoid bacilli are shed in stool of asymptomatic carriers or in stool or urine of people with active disease. Inadequate hygiene after defecation may spread S. typhi to community food or water supplies. In endemic areas where sanitary measures are generally inadequate, S. typhi is transmitted more frequently by water than by food. In developed countries, transmission is chiefly by food that has been contaminated during preparation by healthy carriers. Flies may spread the organism from feces to food. Occasional transmission by direct contact (fecal-oral route) may occur in children during play and in adults during sexual practices. Rarely, hospital personnel who have not taken adequate enteric precautions have acquired the disease when changing soiled bedclothes.

Symptoms and Signs. The incubation period (usually 8 to 14 days, maximum to 21 days) is inversely related to the number of organisms ingested. Onset is usually gradual, with fever, headache, arthralgia, pharyngitis, constipation,
anorexia, and abdominal pain and tenderness. Less common symptoms include dysuria, nonproductive cough, and epistaxis.

Without treatment, the temperature rises in steps over 2 to 3 days, remains elevated (usually 39.4 to 40°C) for another 10 to 14 days, begins to fall gradually at the end of the 3rd wk, and reaches normal levels during the 4th wk. Prolonged fever is often accompanied by relative bradycardia and prostration. CNS symptoms such as delirium, stupor, or coma occur in severe cases. In about 10% of patients, discrete pink, blanching lesions (rose spots) appear in crops on the chest and abdomen during the 2nd wk and resolve in 2 to 5 days. Splenomegaly, leukopenia, anemia, liver function abnormalities, proteinuria, and a mild consumption coagulopathy are common. Acute cholecystitis and hepatitis may occur.

Late in the disease, when intestinal lesions are most prominent, florid diarrhea may occur, and the stool may contain blood (occult in 20% of patients, gross in 10%). In about 2% of patients, severe bleeding occurs during the 3rd wk, with a mortality rate of about 25%. An acute abdomen and leukocytosis during the 3rd wk may suggest intestinal perforation, which usually involves the distal ileum and occurs in 1 to 2% of patients. Pneumonia may develop during the 2nd or 3rd wk and may be due to secondary pneumococcal infection, although S. typhi can also cause pulmonary infiltrates. Bacteremia occasionally leads to focal infections such as osteomyelitis, endocarditis, meningitis, soft-tissue abscesses, glomerulitis, or GU tract involvement. Atypical presentations, such as pneumonitis, fever only, or, very rarely, symptoms consistent with UTI, may delay diagnosis. Convalescence may last several months.

Complications. The two most common complications are haemorrhage (including disseminated intravascular coagulation) and perforation of the bowel. Before antibiotics, perforation had a mortality of around 75%. Jaundice may be due to hepatitis, cholangitis, cholecystitis, or haemolysis. Pancreatitis with acute renal failure and hepatitis with hepatomegaly are rare. Toxic myocarditis occurs in 1-5% of patients (ECG changes may be present). It is a significant cause of death
in endemic areas. Toxic confusional states and other neurological and psychiatric disturbances have been reported.

Diagnosis.

Cultures. Other infections causing a similar presentation include other Salmonella infections, the major rickettsioses, leptospirosis, disseminated TB, malaria, brucellosis, tularemia, infectious hepatitis, psittacosis, Yersinia enterocolitica infection, and lymphoma. Early in its clinical course, typhoid fever may resemble malaria. Cultures of blood, stool, and urine should be obtained. Blood cultures are usually positive only during the first 2 wk of illness, but stool cultures are usually positive during the 3rd to 5th wk. If these cultures are negative and typhoid fever is strongly suspected, culture from a bone marrow biopsy specimen may reveal the organism.

Serology. The traditional serological test is Widal's test. It measures agglutinating antibodies against flagellar (H) and somatic (O) antigens of S. typhi. In acute infection, the O antibody appears first, rising progressively, falls later, and often disappears within a few months. H antibody appears slightly later but persists longer. High or rising O antibody titres generally indicate acute infection, whereas H antibody is used to identify the type of infection. The test is positive on admission in between 40 and 60% of patients.

Treatment. Chloramphenicol, Ceftriaxone, sometimes a fluoroquinolone, levofloxacin, moxifloxacin for 10 to 14 days.

Corticosteroids may be added to antibiotics to treat severe toxicity.

Nutrition should be maintained with frequent feedings. While febrile, patients are usually kept on bed rest. Salicylates (which may cause hypothermia and hypotension), as well as laxatives and enemas, should be avoided. Diarrhea may be minimized with a clear liquid diet; parenteral nutrition may be needed temporarily. Fluid and electrolyte therapy and blood replacement may be needed.

Intestinal perforation and associated peritonitis call for surgical intervention and broader gram-negative and anti–Bacteroides fragilis coverage.
Relapses are treated the same as the initial illness, although duration of antibiotic therapy seldom needs to be $> 5$ days.

Patients must be reported to the local health department and prohibited from handling food until proven free of the organism. Typhoid bacilli may be isolated for as long as 3 to 6 mo after the acute illness in people who do not become carriers. Thereafter, 3 stool cultures at weekly intervals must be negative to exclude a carrier state.

Prevention. In countries in which typhoid is endemic, the most important action is attention to safe drinking water and disposal of sewage. Mass vaccination with typhoid vaccine is also effective. Travellers to endemic areas should also take precautions with regard to hygiene but they must receive vaccination too (there is no vaccine for S. paratyphi A).

2. Study purpose of practical studies:

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

• have an idea: a place in the structure of typhoid fever infections, the study of history, scientific contribution of scientists during an epidemics of typhoid fever in the world.

• read: the statistical data on the prevalence of typhoid fever, mortality, frequency of complications in the world.

2.2. Student should know:

• etiology agents, epidemiology, pathogenesis of typhoid fever;
• clinical aspects of typhoid fever with typical disease course;
• clinical and epidemiological characteristics of paratyphoid A and paratyphoid B;
• pathogenesis, term of onset and clinical manifestations of typhoid fever complications;
• laboratory diagnostics of typhoid fever;
2.3. Student should be able to:

- keep basic rules at the bedside;
- prepare medical history with epidemiologic evidence estimation;
- examine a patient and detect basic typhoid fever symptoms and syndromes, prove clinical diagnosis for timely referral to treatment;
- make differential diagnostics of typhoid fever;
- identify possible typhoid fever complications and emergencies on basis of clinical examination;
- make patient’s examination plan;
- interpret laboratory examination results;
- make individual treatment plan
- make recommendations concerning regimen, diet, examination, observation during convalescent period.

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 typhoid fever.
- 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. Basic knowledge, skills which are necessary for studying of topic (interdisciplinary integration)

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<th>Discipline</th>
<th>To know:</th>
<th>To be able to:</th>
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<td><strong>Previous disciplines</strong></td>
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<td>Characteristics of S. typhi, S. paratyphi A, S. paratyphi B</td>
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<td>Obtain epidemiologic medical history, take antiepidemic and prophylactic measures in the focus of infection.</td>
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<td><strong>Dermatology</strong></td>
<td>Pathogenesis, clinical characteristics of exanthema.</td>
<td>Detect skin rash of patients with typhoid fever</td>
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<td><strong>Surgery</strong></td>
<td>Signs of small bowel ulcer perforation, enterorrhagia, first-aid approach.</td>
<td>Timely diagnose complications and give first aid.</td>
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<td><strong>Internal Diseases Propedeutics</strong></td>
<td>Basic stages and methods of patient’s clinical examination.</td>
<td>Obtain medical history, make clinical examination, detect pathological symptoms and syndromes.</td>
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<td><strong>Clinical</strong></td>
<td>Pharmacokinetics and</td>
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<td>Pharmacology.</td>
<td>Pharmacodynamics, side effects of chloramphenicol, ciprofloxacin, means of pathogenetic therapy.</td>
<td>accordance with the age, individual features of the patient, choose optimal medicine intake and dose, issue prescription.</td>
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| Resuscitation and Intensive Care | **Emergencies:**  
  - infectious and toxic shock  
  - enterorrhagia  
  - enterobrosia  
  - infectious and toxic encephalopathy | **Timely diagnose and give first aid in case of emergency:**  
  - infectious and toxic shock  
  - enterorrhagia  
  - enterobrosia  
  - infectious and toxic encephalopathy |
3.2 Theme contents.

**Typhoid fever. Paratyphoid A, Paratyphoid B**

**Ethiology**

**Epidemiology**
- Source – sick person and carrier (Paratyphoid B – human and cattle). Fecal-oral mechanism; water, food, contact and domestic route, high susceptibility, durable postinfectious immunity.

**Pathogenesis**
- Hypersensibility of slow type → Ulceration → Surgical complications
- Penetration
- Degree of lymph protection
- Bacteriemia
- Toxemia
- Paranchyma diffusion
- Agent removal
- Immune reaction forming
- Recovery, agent elimination
- Chronic carrier state forming

**Clinic**
- Fever, intoxication
- Status typhosus
- Rash
- Skin pallor
- Baked tongue
- Meteorism, constipation
- Hypotonia
- Relative bradycardia
- Muffled heart
- Hypotonia

**Complications**
- Infectious and toxic shock
- DIC-syndrome
- Enterorrhagia
- Perforation of typhoid fever ulcer, peritonitis

**Specific diagnostics**
- Hemoculture
- Copro-urinal culture
- Antibody-mediated methods: hemagglutination reaction, indirect hemagglutination reaction, immune-enzyme analysis IgM

**Treatment**
- Etiotropic: chloramphenicol,
- Disintoxication
- Hemostatic therapy
- Surgical intervention

**Preventive measures**
- Planned
- Emergency

**Specific**
3.3 Literature recommended:

**Main sources:**

1. Lectures of Professor.

**Additional sources:**

3.4. Self-control materials

3.4.1. Questions for self-control

1. Ways of typhoid fever, paratyphoid A, paratyphoid B transmission.
2. S. typhi pathogenicity factors and stages of typhoid fever pathogenesis.
3. Structural (morphological) changes of small bowel wall subject to the period of disease.
4. Stages of cyclic clinical course of a typhoid fever.
5. Main signs of a typhoid fever
7. Type of fever of patients with typhoid fever.
8. Characteristic of paratyphoid A, paratyphoid B course
9. Specific typhoid fever complications.
10. Hemogram of patients with typhoid fever at the height of disease.
11. Plan of examination of patients with typhoid fever.
12. Methods of specific typhoid fever diagnostics. Interpretation of results subject to the period of disease and examined material.
13. Etiotropic therapy of typhoid fever
15. Treatment of typhoid fever complications.
16. Rules of convalescents’ discharge from the hospital.
3.4.2. Tests for self-control

Choose correct answers: $\alpha=2$

Variant 1

1. The causative agent of typhoid belongs to the family Enterobacteriacea, genus:
   A. Shigella
   B. Salmonella
   C. Yersinia
   D. Escherichia
   E. Everything is wrong.

2. The causative agent of typhoid on adverse conditions can form:
   A. spores
   B. capsules
   C. L-forms
   D. everything is right
   E. everything is wrong.

3. Antigenic structure of the causative agent of typhoid is characterized by presence of:
   A. a somatic (O) antigens
   B. a flagellar (H) antigen
   C. a virulence (Vi) antigen
   D. everything is right
   E. everything is wrong.

4. Who can be source of typhoid fever? :
   A. infected human
   B. infected human and cows
   C. pigs
   D. cockroachs
   E. cats and dogs.

5. The factors of the transmission of typhoid fever may be:
   A. water and air
   B. medical instruments
   C. soil
   D. insects
   E. water and food.

6. What plays the main role in pathogenesis of typhoid fever?:
   A. exotoxin
   B. endotoxin
C. hemolysin
D. neurominidase
E. endotoxin and exotoxin.

7. What phase of pathogenesis coincides with the first clinical signs of typhoid fever?:
A. penetration of the causative agent
B. development of lymphadenitis and lymphangitis
C. bacteremia
D. parenchymatous diffusion
E. discharge of the agent from the organism.

8. What is underlying in development of “status typhosus”?:
A. endotoxins’s toxic action on nervous centres (with development of processes of inhibition)
B. development of meningitis
C. exotoxin’s toxic action on nervous centres (with development of processes of excitation)
D. development of encephalitis
E. development of sepsis due to action causative agent’s poison.

9. What postinfectious immunity forms after typhoid fever in the most cases?:
A. stable non-specific
B. unstable specific
C. stable specific
D. artificial
E. unstable non-specific.

10. What is the average incubation period of typhoid fever?:
A. 10-14 days
B. 20-30 days
C. 3-5 days
D. from a few hours till 2-3 days
E. 1-6 months.

Keys:

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Variant 2

1. What sign isn’t typical for typhoid fever?:
A. high temperature
B. skin eruption
C. hepatosplenomegaly
D. icteritiousness of skin
E. abdominal pain.

2. What symptom is typical while examination of patient with typhoid fever?:
   A. short sound on percussion in the iliocaecal area
   B. disappearence of liver dullness on percussion
   C. incompletely closed anus
   D. rigid neck
   E. exophthalmos.

3. When the typical rash appear in patients with typhoid fever?:
   A. on the second day of the disease
   B. after 2 weeks of the disease
   C. in the incubation period
   D. before the rising temperature
   E. on the 8-10th day of the disease.

4. Where is usual localization of skin rash in patients with typhoid fever?:
   A. all over the body
   B. on the lower part of trunk and thighs
   C. on upper region of abdomin and lower region of chest
   D. on face and neck
   E. on scalp.

5. The following complications are typical for typhoid fever, except:
   A. intestinal perforation
   B. infection-toxic shock
   C. intestinal bleeding
   D. chronic carrier
   E. liver insufficiency.
6. What method of laboratory diagnostics is the most important for making a diagnosis of typhoid fever?:
   A. bacteriological investigation of blood
   B. bacteriological investigation of urine and feces
   C. the Widal reaction
   D. microscopy of blood smear
   E. biological method.

7. What laboratory method isn’t used for early diagnostics of typhoid fever?:
   A. immune-enzyme analysis
   B. polymerase chain reaction
   C. bacteriological investigation of blood
   D. the Widal reaction
   E. everything is used.

8. What titre is diagnostical in the Widal reaction?:
   A. 1:100
   B. 1:200
   C. 1:300
   D. 1:400
   E. the titre isn’t important.

9. What material is used for bacteriological diagnostics of typhoid fever?:
   A. urine
   B. blood
   C. bile
   D. feces
   E. everything is right.

10. What antibacterial medicine is the most effective in treatment of typhoid fever?:
    A. ciprofloxacin
    B. penicillin
    C. chloramphenicol
D. cephalosporins
E. interferon.

Keys:

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3.4.3. Situational tasks of the second level learning

Task 1
A patient, age – 54, came to a district doctor on the sixth day of disease complaining of strong headache, general weakness, absence of appetite, insomnia, raise of temperature from 37,5°C at the very first day to 39,5°C at the day of visit. Objectively: significant skin paleness, no rash. A tongue is off-white; on the lateral surfaces, which are free from fur, there are teeth marks. Pulse - 74 beats/minute, blood pressure - 110/60. An abdomen is moderately bloated, painless. 1,5 sm of liver are seen from under the costal margin edge. Padalka's symptom is positive. No bowel emptying for 2 days.

1. Make preliminary diagnosis.
2. Plan of examination
3. Treatment.

Task 2
The patient turned to the doctor on the 8th day of illness. His temperature was increased during the week. He had headache, weakness, decreased appetite. On examination: temperature 39.5, answered questions sluggish, slightly retarded, pale skin, tongue with brown fur, blood pressure 100/60, pulse 74 beats/minute. Flatulence, a few roseola are on the skin of abdominal. Hepatolienal syndrome is determined. Stool is delayed during several days.

1. Make preliminary diagnosis.
4. Materials for the class of independent work

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

- To master methods of examination of a patient with typhoid fever, paratyphoid A, paratyphoid B
- To examine a patient with typhoid fever
- To make differential diagnostics of typhoid fever
- To make laboratory examination plan
- To interpret results of specific examination of a patient with typhoid fever
- To diagnose typhoid fever complications
- To make up a treatment plan of a patient with typhoid fever.
- To determine a policy of treatment in emergency cases.
- To draw up medical documentation after diagnosing typhoid fever.

4.2. Professional algorithm of obtaining knowledge and skills of typhoid fever, paratyphoid A, paratyphoid B diagnostics.

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<th>Tasks</th>
<th>Sequence of actions</th>
<th>Notices and warnings concerning self-control</th>
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<td>1.</td>
<td>To master methods of examination of a patient with typhoid fever,</td>
<td>I. Determine patient’s complaints.</td>
<td>Sort complaints characterizing different</td>
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<td>paratyphus A, paratyphus B</td>
<td>II. Determine history:</td>
<td>syndromes:</td>
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<td>1. Case history</td>
<td>- general intoxication</td>
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<td>2. Patient’s history</td>
<td>- affection of organs</td>
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<td>- accessory affections</td>
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<td>Pay attention to a gradual beginning;</td>
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<td>period, sequence of appearance and</td>
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<td>treatment</td>
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2. To examine a patient with typhoid fever

II. Make proper examination.

1. General examination:
   - general condition of a patient;
   - skin, pharyngeal glands;

2. Digestive system:
   - tongue examination;
   - abdomen percussion;
   - abdomen palpation;

3. Epidemic history
   - dynamics of fever;
   - headache;
   - sleep disturbance;
   - delay of bowel emptying;
   - rash;
   - other symptoms

Detect past illnesses.
Find out information about fecal and oral mechanism of transmission, pay attention to patient’s stay at the regions with the increased risk of typhoid fever, paratyphus A and paratyphus B contamination.

Remember: presence, intensity and dynamics of symptoms are caused by the period and severity of clinical course and depend on the patient’s age and concomitant pathology.

Pay attention to:
- patient’s slackness, adynamia and lethargy;
- characteristics of bowel emptying.

3. Cardiovascular system:
- pulse;
- blood pressure;
- heart auscultation.

Pay attention to:
- „baked tongue”;
- Hepatosplenomegaly (Banti's syndrome);
- meteorism;
- positive Padalka’s syndrome;
- symptoms of peritonitis (if any, there is small bowel ulcer perforation);
- susceptibility to constipation (melena is an evidence of enterorrhagia);
- relative bradycardia (tachycardia is an evidence of complication!);
- dicrotic pulse;
- moderately low blood pressure.
<table>
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<tr>
<th>Prescribe</th>
<th>Pay attention to typical changes:</th>
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<td>(significantly low blood pressure is an evidence of complication!);</td>
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<td>- moderately hollow heart tones</td>
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<td>Pay attention to:</td>
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<td>- presence of the signs of bronchitis among some patients.</td>
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<td>If clinical course is poor a patient may have status typhosus, delirium, hallucinations</td>
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3. laboratory and additional examinations, interpret their results

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<td><strong>1. General blood analysis.</strong></td>
<td>leucopenia, lymphomonocytosis, aneosinophilia, thrombocytopenia (leukocytosis, anemia, increase of erythrocyte sedimentation rate appear in case of complications).</td>
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<td><strong>2. General urine analysis.</strong></td>
<td>Absence of significant changes in case of typical clinical course.</td>
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<td><strong>3. Abdominal ultrasound</strong></td>
<td>Hepatosplenomegaly (Banti's syndrome)</td>
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<td><strong>4. Hemoculture</strong></td>
<td>Prescribed irrespective of a disease period in case of fever (as soon as possible).</td>
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<td><strong>5. Coprourinal culture</strong></td>
<td>Becomes positive from the end of the first week of disease.</td>
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<tr>
<td><strong>6. Antibody-mediated methods:</strong> hemagglutination reaction, (Widal reaction), indirect hemagglutination reaction - Immune-enzyme analysis (IgM)</td>
<td>Prescribed in paired blood serum with 10 days interval; <strong>Diagnostic titer – 1:200.</strong></td>
<td></td>
</tr>
</tbody>
</table>
Dynamics of clinical symptoms of typhoid fever

<table>
<thead>
<tr>
<th>Term of the disease</th>
<th>1&lt;sup&gt;st&lt;/sup&gt; week of the disease</th>
<th>2&lt;sup&gt;nd&lt;/sup&gt; week of the disease</th>
<th>3&lt;sup&gt;rd&lt;/sup&gt; week of the disease</th>
<th>4&lt;sup&gt;th&lt;/sup&gt; week of the disease</th>
<th>5&lt;sup&gt;th&lt;/sup&gt; week of the disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body temperature rise</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Headache</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Insomnia</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Status typhosus</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>General weakness</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Meteorism</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Rash</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Padalka's symptom</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Filipovych symptom</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Hepatolienal syndrome</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Relative bradycardia</td>
<td>-</td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

4.3. Materials methods of the final phase of occupation

4.3.1. Objectives of the 3rd level

Task

Patient I., 19 years old, entered the hospital with 9.03. on the 7th day of illness. Sick 4.03.: Increased body temperature to 38,0 °C, there were chills,
headache, and weakness. In the following days, the body temperature was kept at 38.2 - 39.6 °C, was growing headache, receiving antipyretics and analgesics did not give effect. Twice was mushy stool. He ate almost nothing, greatly weakened. Arrived 10 days ago with her mother from the focus of military operations on the border of the Chechen Republic and Dagestan.

On admission the patient's condition is serious. Body temperature - 39.2 °C. Consciousness is preserved, but the patient is very sluggish, indifferent, meets with reluctance, in monosyllables, with a delay. Hardly tells history of the disease. Pale skin on the anterior abdominal wall single pale pink rash elements diameter not exceeding 5 mm. Language increased, thickly coated with a thick dirty brown touch dry, with imprints of teeth. Pulse 64 beats/min., weak filling. BP - 90/50. Cardiac sounds are muffled. Harsh breath without rales. abdomen is distended, during palpation is soft and painless, heard rumbling in the right iliac region. Padalkas symptom is positive. Defined edge of the liver 1.5 cm below the costal arch, clearly palpable spleen edge. Mushy stools was twice without impurities.

1. Highlight the main clinical syndromes.
2. Formulate a preliminary diagnosis.
3. Is there a need to differentiate these diagnosis with other infectious and non-communicable diseases?
4. Specify the possible complications.
5. What are the indications for hospitalization.
6. Make a plan for examination of the patient.
7. Assign the necessary treatment.
8. Specify the probable source mechanisms of infection, ways of transmission.
9. Make the plan of anti-epidemic measures in the focus.

**The right answers**
1. An intoxication - general weakness, lethargy, apathy, headache, chills, loss of appetite, prolonged febrile fever, bradycardia, hypotension; B)
hepatosplenomegaly; C) roseolous rash; D) mesenteric lymphadenitis - bloating, positive symptom Padalka.

2. Diagnosis: Typhoid fever, a severe form, the crisis period.

3. Intoxication syndrome associated with bacteremia, endotoxemia and exposure to toxins on the central nervous and cardiovascular systems; hepatosplenomegaly - naturally caused generalized infectious process and specific (with the formation of granulomas typhoid) defeat these bodies; roseolous rash - the result of dissemination of the pathogen in the superficial layers of the skin with the development productively - inflammatory changes; mesenteric lymphadenitis - result hyperplasia inflammatory lymph nodes the mesentery.

In the wall of the small intestine is legitimate STAGED changes - medullary swelling of the lymphoid tissue of the small intestine, swollen lymph necrotisation formations rejection of necrotic masses of lymphoid tissue and the formation of "dirty" ulcers, ulcers cleansing of necrotic tissue, the period of "clean" ulcers, ulcer healing.

4. The disease must be differentiated from typhus, malaria, brucellosis, sepsis, tuberculosis, Hodgkin's disease.

5. Possible specific complications: toxic shock, intestinal bleeding, perforation of typhoid ulcers, ruptured peritonitis.

6. For suspected typhoid a mandatory provisionally hospitalization. Indications for hospitalization: epidemiological and clinical.

7. Hemogram: can detect leukopenia with neutrophilic shift aneozinofiliya, relative lymphocytosis, thrombocytopenia, a mild increase in ESR. Urinalysis: proteinuria may, cylindruria, microscopic hematuria. To verify the diagnosis: bacteriological examination - sowing Covey, urine, feces and duodenal contents on media containing bile (cond. Rappoport).

Expected results:
- Finding in blood culture pathogen S. Typhi abdominalis from first day of illness;
- Finding in urinoculture from 2nd week of illness;
- Finding in coproculture from 2-3rd week of illness;
- Sowing duodenal contents conducted to assess the bacteriological sanitation body.

Serological methods: Phragmites with Salmonella antigen complex with 5 - 7th day of illness and later in paired sera. A positive response is considered at a titer of 1:200 or higher (titer increase in 4 times)

8. Bed rest until the 10th day of normal temperature. Diet number 4 - mechanically and chemically gentle. Antibiotic therapy is conducted throughout the febrile period and within 10 days of the period apyrexia (ciprofloxacin, ofloxacin, chloramphenicol). Detoxification therapy - Ringer, hemodez, reopoliglukin, 5% glucose solution. Vitamins, Enzymes.

9. The disease belongs to a group of bacterial intestinal infections. Anthroponosis. Source of infection - a sick man or MBT. Transmission mechanism - fecal-oral. Transmission path - water, nutritional and contact-household.

10. In the focus final disinfection is made, control of contact persons for 21 days with daily thermometry and once bacteriological examination of stool (feces sowing typhoid - paratyphoid group.)

Specific prevention in the outbreak include the appointment of bacteriophage to all contact persons.

At the outbreak of typhoid also carry sanitary supervision of water supply and sanitation.

5. Materials of after-work

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

"Prospects for early diagnosis of typhoid fever »
"Clinical and epidemiological characteristics of typhoid fever"
"Differential diagnosis of typhoid fever"
"Pathogenesis of complication of typhoid fever"
"Toxic shock. Etiopathogenesis, clinical manifestations, intensive treatment"
DIARRHEA SYNDROME IN INFECTIOUS DISEASES CLINIC.  
SALMONELLOSIS, FOOD TOXICOINFECTION, BOTULISM,  
ENTEROVIRAL INFECTION, ROTAVIRAL INFECTION

1. Actuality of theme

Gastroenteritis is inflammation of the lining of the stomach and small and large intestines. Acquisition may be foodborne, waterborne, or via person-to-person spread. Gastroenteritis is usually uncomfortable but self-limited. Electrolyte and fluid loss is usually little more than an inconvenience to an otherwise healthy adult but can be grave for people who are very young, elderly, or debilitated or who have serious concomitant illnesses. Worldwide, an estimated 1.5 million children die each year from infectious gastroenteritis; although high, this number represents one half to one quarter of previous mortality. Improvements in water sanitation in many parts of the world and the appropriate use of oral rehydration therapy for infants with diarrhea are likely responsible for this decrease.

Infectious gastroenteritis may be caused by viruses, bacteria, or parasites. Many specific organisms are discussed further in the Infectious Diseases section. A great place among acute virulent infections of digestive system is taken by such diseases like salmonellosis and food toxicoinfections. Today, salmonellosis is one of the most widely spread anthropooneses in developed countries. There is a trend for care rate increase; in particular it concerns big cities with centralized system of food supply. It is caused by ubiquitous nature of germs, their resistance to ambient conditions and ability to accumulate beyond living organisms. Social aspect plays an important part - expansion of public food network, deviations in manufacturing technologies and storage of food products, failure to follow rules of personal care and low level of sanitary culture of some branches of population. These diseases, especially in terms of an epidemic, results in great economic damage and can contribute to development or recrudescence of chronic diseases, and in some cases lead to patient’s death. Apart from influence on digestive tract, germs can lead to sepsis development; especially in terms of congenital or secondary
immunodeficiency (septic and pyemic progress of salmonellosis is referred to HIV-marked disease). Vast prevalence of salmonellosis and FTI, quick dehydratation and possible contamination during indoor treatment makes doctors of any profile obliged to study this pathology. The botulism is often in the centre of doctor’s and explorer’s attention in spite of its rare register. It is because of hard movement, of insufficient knowledges of pathogenes, high death rate.

In the world botulism has the main importance because of use the conserved products prepared at home without the observance of peculiar technology. During last years the information about flare of illness has been caused as a result of eating the salt and smoke fish with fabric production.

Viruses are very common cause of gastroenteritis. They infect enterocytes in the villous epithelium of the small bowel. The result is transudation of fluid and salts into the intestinal lumen; sometimes, malabsorption of carbohydrates worsens symptoms by causing osmotic diarrhea. Diarrhea is watery.

**Rotavirus** is the most common cause of sporadic, severe, dehydrating diarrhea in young children. Rotavirus is highly contagious; most infections occur by the fecal-oral route. Adults may be infected after close contact with an infected infant. The illness in adults is generally mild. Incubation is 1 to 3 days. In temperate climates, most infections occur in the winter.

Symptoms and Signs. The character and severity of symptoms vary. Generally, onset is sudden, with anorexia, nausea, vomiting, borborygmi, abdominal cramps, and diarrhea (with or without blood and mucus). Malaise, myalgias, and prostration may occur. The abdomen may be distended and mildly tender; in severe cases, muscle guarding may be present. Gas-distended intestinal loops may be palpable. Persistent vomiting and diarrhea can result in intravascular fluid depletion with hypotension and tachycardia. In severe cases, shock, with vascular collapse and oliguric renal failure, occurs. If vomiting is the main cause of fluid loss, metabolic alkalosis with hypochloremia can occur. If diarrhea is more prominent, acidosis is more likely. Both vomiting and diarrhea can cause hypokalemia. Hyponatremia may develop, particularly if hypotonic fluids are used.
in replacement therapy. In viral infections, watery diarrhea is the most common symptom; stools rarely contain mucus or blood. Rotavirus gastroenteritis in infants and young children may last 5 to 7 days. Vomiting occurs in 90% of patients, and fever > 39°C occurs in about 30%.

Diagnosis. Rotavirus can be diagnosed using commercially available rapid assays that detect viral antigen in the stool, but these assays are usually done only to document an outbreak. General tests: Serum electrolytes, BUN, and creatinine should be obtained to evaluate hydration and acid-base status in patients who appear seriously ill.

Treatment. Oral or IV rehydration. Supportive treatment is all that is needed for most patients. Bed rest with convenient access to a toilet or bedpan is desirable. Oral glucose-electrolyte solutions, broth, or bouillon may prevent dehydration or treat mild dehydration. Even if vomiting, the patient should take frequent small sips of such fluids; vomiting may abate with volume replacement. Carbonated beverages and sports drinks lack the correct ratio of glucose to Na. If vomiting is protracted or if severe dehydration is prominent, IV replacement of volume and electrolytes is necessary. When the patient can tolerate fluids without vomiting and the appetite has begun to return, food may be gradually restarted. There is no demonstrated benefit from restriction to bland food (eg, cereal, gelatin, bananas, toast). Some patients have temporary lactose intolerance. Antidiarrheal agents are safe for patients > 5 yr with watery diarrhea (as shown by heme-negative stool). Effective antidiarrheals include loperamide. If vomiting is severe and a surgical condition has been excluded, an antiemetic may be beneficial. Drugs useful in adults include prochlorperazine and promethazine. Although probiotics appear to briefly shorten the duration of diarrhea, there is insufficient evidence that they affect major clinical outcomes (eg, decrease the need for IV hydration and/or hospitalization) to support their routine use in the treatment or prevention of infectious diarrhea.

Prevention. Two oral rotavirus vaccines are available that are safe and effective against the majority of strains responsible for disease. Rotavirus
immunization is part of the recommended infant vaccination schedule. Prevention of infection is complicated by the frequency of asymptomatic infection and the ease with which many agents, particularly viruses, are transmitted from person to person. In general, proper procedures for handling and preparing food must be followed. Travelers must avoid potentially contaminated food and drink. To prevent recreational waterborne infections, people should not swim if they have diarrhea. Infants and toddlers should have frequent diaper checks and should be changed in a bathroom and not near the water. Swimmers should avoid swallowing water when they swim. Breastfeeding affords some protection to neonates and infants. Caregivers should wash their hands thoroughly with soap and water after changing diapers, and diaper-changing areas should be disinfected with a freshly prepared solution of 1:64 household bleach. Children with diarrhea should be excluded from child care facilities for the duration of symptoms.

**Enteroviral infection.** Enteroviruses are distributed worldwide and are influenced by season and climate. Infections occur in summer and early fall in temperate areas, while tropical and semitropical areas bear the brunt all year. Enteroviruses include Coxsackieviruses, Echoviruses, Enteroviruses, Polioviruses. Enteroviruses are picornaviruses (pico, or small, RNA viruses). All enteroviruses are antigenically heterogeneous and have wide geographic distribution. Enteroviruses are shed in respiratory secretions and stool and sometimes are present in the blood and CSF of infected patients. Infection is usually transmitted by direct contact with respiratory secretions or stool but can be transmitted by contaminated environmental sources (eg, water). Enteroviral diseases or epidemics occur in summer and fall. Infection transmitted by a mother during delivery can cause severe disseminated neonatal infection, which may include hepatitis or hepatic necrosis, meningoencephalitis, myocarditis, or a combination. Intact humoral immunity and B-cell function are required for control of enteroviral disease. Severe enteroviral infections (often manifesting as a slowly progressive meningoencephalitis) occur in patients with agammaglobulinemia but usually not in those with other immune deficiencies. Enteroviruses are transmitted
predominantly via the fecal-oral route. However, there are some exceptions, including coxsackievirus, which is spread mainly by respiratory secretions and enterovirus, which is shed in tears and spread via fingers and fomites. Upon entry into the oropharynx, the virus replicates in submucosal tissues of the distal pharynx and alimentary tract. Viral particles are shed in the feces and in upper respiratory tract secretions for days prior to symptom onset. The average incubation period is 3-10 days, during which the virus migrates to regional lymphoid tissue and replicates. Minor viremia results, which is associated with the onset of symptoms and viral spread to the reticuloendothelial system (spleen, liver, bone marrow).

Dissemination to target organs follows, and viral replication in target organs produces the major viremia with possible secondary seeding of the CNS. Potential target organs include the skin and CNS. Infectious virus is shed from the upper respiratory tract for 1-3 weeks and from the feces for 3-8 weeks.

Enteroviruses cause various syndromes. Epidemic pleurodynia, hand-foot-and-mouth disease, herpangina, viral exanthems, gastroenteritis and poliomyelitis are caused almost exclusively by enteroviruses. Other disorders (eg, aseptic meningitis, myopericarditis) may be caused by enteroviruses or other organisms.

Physical examination findings in enteroviral disease vary greatly depending on the type of illness and etiologic agent. Diagnosis of enterovirus infections is often clinical. Laboratory diagnosis can be achieved with serological tests, viral isolation by cell culture, and polymerase chain reaction (PCR).

Unfortunately, no specific antiviral medication or treatment is available for an enteroviral infection. The best care is provided through supportive measures. Fluid hydration and antipyretics are the mainstays of care for a viral syndrome.

Prevention. Hygienic measures such as hand washing and adequate disposal of infected secretions help prevent the spread of enteroviral infections.

2. Study purpose of practical studies:

2.1. The student must have an idea (read):
1. have a general idea about position of Infectious gastroenteritis in the structure of virulent diseases, prevalence in the world; study statistic data related to case rate, case mortality, event frequency and bacteria carriage as for today.
2. get familiar with history of scientific study of Infectious gastroenteritis, have an idea of scientific contribution of native scientists, in the history of scientific research in this field.

**2.2. The student is should know: α - 2**

1. causation of salmonellosis, food toxicoinfection (FTI), botulism, viral gastroenteritis;
2. pathogenesis of salmonellosis, food toxicoinfection (FTI), botulism, viral gastroenteritis;
3. clinical signs of salmonellosis, food toxicoinfection (FTI), botulism, viral gastroenteritis;
4. pathogenesis, genesis term and clinical aspects infectious gastroenteritis complications;
5. laboratory diagnostics Infectious gastroenteritis;
6. the ways of treatment;
7. principles of prophylaxis;
8. medical approach in case of emergencies;
9. rules of discharge of recovered patients from in-patient hospital.

**2.3. The student should be able to: α-3**

1. Follow the main rules of behavior by sickbed.
2. Make up medical history estimating epidemiological data.
3. Examine the patient and find out the main symptoms and syndromes of infectious gastroenteritis, justify the clinical diagnosis, and solve the issue of necessary inpatient treatment.
4. Based on clinical examination define possible complications of Infectious gastroenteritis, emergencies.
5. Fill in medical documentation based on previously stated diagnosis “Infectious gastroenteritis” (emergency call to regional epidemiological department).
6. Make up a plan of patient’s laboratory and instrumental examination.
7. Analyze the results of laboratory examination.
8. Give a proper estimate to the results of specific methods of diagnostics proceeding from material and period of examination.
9. Make up an individual treatment plan taking into account epidemiological data, stage of disease, available complications, severity of the condition, allergic anamnesis, morbidity, provide rescue emergency care.
10. Make up a preventive measures plan for the centre of infections.
11. Provide recommendations related to mode of treatment, diet, examination and medical supervision during recovery period.

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

<table>
<thead>
<tr>
<th>Discipline</th>
<th>Know</th>
<th>Be able to</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microbiology</td>
<td>Features of opportunistic microorganisms and</td>
<td>Take samples of material for</td>
</tr>
<tr>
<td>Subject</td>
<td>Description</td>
<td>Methodology</td>
</tr>
<tr>
<td>----------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Propedeutics of medical diseases</strong></td>
<td>Main stages and methods of patient clinical examination</td>
<td>Make up medical history, perform clinical examination of the patient by different organs and systems, define clinical symptoms of pathology</td>
</tr>
<tr>
<td><strong>Epidemiology</strong></td>
<td>Epidemiological process of salmonellosis and food toxicoinfections</td>
<td>Make up an epidemiological history, perform antiepidemic and preventive measures in the centre of infection</td>
</tr>
<tr>
<td><strong>Immunology and allergology</strong></td>
<td>Role of immunity system in infectious process, influence on the term of germ elimination from human organism. Immunological aspects of Salmonella carriage</td>
<td>Analyze data of immunological examinations</td>
</tr>
<tr>
<td><strong>Physiology</strong></td>
<td>Aspects of physiological standards of human organs and systems; aspects of laboratory examination in standard condition</td>
<td>Estimate data of laboratory examination</td>
</tr>
<tr>
<td><strong>Clinical pharmacology</strong></td>
<td>Pharmacological properties, adverse effects of means of nosotropic therapy</td>
<td>Prescribe treatment with regard to age, individual symptoms of the patient, chose an optimum mode of drug intake and dosage, provide prescriptions</td>
</tr>
<tr>
<td>Nevrology</td>
<td>The pathogenesis, the clinical symptoms of illness</td>
<td>To make the clinical examination for patient with failure of nervous system</td>
</tr>
<tr>
<td>---</td>
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<td>---</td>
</tr>
</tbody>
</table>
| Reanimation and intensive care | Emergencies:  
  - TSS  
  - Dehydration shock | Make due diagnosis of and provide rescue care in emergencies |
| Other disciplines | | |
| Family practice | Pathogenesis, epidemiology, intensiveness of clinical signs, possible complications of salmonellosis and food toxicoinfections. Principles of prophylactics and treatment. | Perform differential diagnostics of diseases with various genuses at salmonellosis and food toxicoinfections. Find out salmonellosis and food toxicoinfections and possible complications; analyze results of laboratory examination. Admit the patient to contagious isolation ward in due time. Fill in the emergency notice. Provide rescue emergency care if required |
| Integration between subjects | | |
| Virulent diseases | Features of infectious diseases. Methods of diagnostics, treatment and prophylactics of infectious diseases. Pathogenesis, epidemiology, intensiveness of clinical signs, laboratory diagnostics, possible complications specific features of clinical progress of salmonellosis | Perform differential diagnostics of salmonellosis and food toxicoinfections with other infectious diseases. Define salmonellosis and food toxicoinfections, their complications; analyze results of laboratory examination. Prescribe treatment. Provide rescue emergency care in pre- |
3.2 Theme contents.

**Salmonellosis**

<table>
<thead>
<tr>
<th>Causation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Salmonella, except germs of typhoid fever and paratyphoids</td>
</tr>
<tr>
<td>Endotoxin, enterotoxin (disturbs synthesis of citidine monophosphate)</td>
</tr>
</tbody>
</table>

Prophylactics and treatment methods in hospital stage.
**Food toxico infection**

**Causation**
Opportunistic flora: Aerobic bacteria: Proteus, E.coli; gram-positive cocci: Staph., Str. Sporogenous anaerobes: Cl.perfringens
Resistant to ambient conditions, able to accumulate in food products and generate toxins

**Epidemiology**
Way of transmission – food.
Infection source – person suffering from pyoinflammatory process of various locations, germ carriers, cows suffering from blue bag

**Pathogenesis**
- Introduction of bacteria and/or toxins
- Bacteria destruction
- Toxemia
- Metabolic processes failure
- Generalization
- Pathology of organs
- Toxin elimination
- Death
- Recovery
- Collapse
- Intoxication
- Fever
- Hypotonia
- Stomach-ache
- Vomiting
- Diarrhea
- Thirst
- Convulsions
- Dehydration
- Dehydratation shock

**Clinics**
- Intoxication
- Fever
- Hypotonia
- Stomach-ache
- Vomiting
- Diarrhea
- Thirst
- Convulsions
- Dehydration
- Dehydratation shock

**Complications**
- Dehydratation shock

**Specific diagnostics**
- Bacterial inoculation of biological material
- RHT, AT with autohemagglutinin

**Treatment**
- Deintoxication
- Dehydratation
- GТ lavage
- Absorbs
- Rehydration

**Prophylactics**
Non-specific
Botulism

- **The etiology**
  - Clostridium botulinii

- **The epidemiology**
  - eating products, especially can without sufficient thermical processing
  - the presence of wounds infected by ground
  - the nonobservance of sanitary and hygienic norms during the separation of animals

- **The pathogens**
  - Botulotoxin
    - The oppression of parasympathetic nervous system
      - The paresis of intestines
      - The transgression of heart-vessels system
      - The transgression of production in nervous-muscle synaps
    - The pharyngoplegic syndrome
    - The ophthalmoplegic syndrome

- **The definition of seriousness, the type of exciter, complications**
  - Yes, diagnosis is confirmed
  - The differential diagnosis with poisoning by metilen spirit, belladonna, mushrooms, the bulbous form of polyomyelit
  - No

- **The treatment**
  - irrigation of stomach and intestines
  - antibotulinum anatoxin (A,B,C types)
  - desintoxication
  - antibacterial treatment
  - medicines opposite cholinesterase
  - symptomatic therapy

- **The recovery; the discharging**
  - The disappearance of clinical symptoms
  - The dispensarisation

- By clinical evidences the doctor for eyes and nervous system make the inspection
3.3 Literature recommended:

**Main sources:**

1. Lectures of Professor.

**Additional sources:**


3.4. Self-control materials

3.4.1. Questions for self-control
1. Source of infection at salmonellosis and food toxicoinfections.
2. Ways and main aspects of salmonellosis and food toxicoinfections transmission.
4. Pathogenesis of the main salmonellosis clinical symptoms.
5. Basic symptoms of local forms of salmonellosis.
6. Main symptoms of generalized forms of salmonellosis.
7. Basic symptoms of food toxic infections caused by opportunistic germs.
8. Salmonellosis clinical classification.
10. Specific complications of salmonellosis.
11. Reasons for generalized forms of salmonellosis development.
12. Biochemical aspects to be examined in patients with salmonellosis and FTI.
13. Examination plan for patient with supposed salmonellosis and FTI.
15. Factors of FTI specific diagnostics.
16. Indications for bacteriological blood testing at salmonellosis and FTI.
17. Diagnostics of salmonellosis carriage.
19. Emergency care at local forms of salmonellosis and FTI.
21. The peculiarity of botulism’s exciter
22. The characteristic of botulotoxin
23. The cause of infection and the factor of transmission
24. The pathogenes of botulism
25. The classification of botulism
26. The main clinical syndromes of botulism
27. The clinic according to the seriousness of condition
28. The specific complications of botulism
29. The differential diagnosis of botulism
30. The plan of inspection for patients with botulism
31. The methods of specific diagnostic of botulism
32. The analysis of results of laboratory exploration
33. The specific therapy for botulism: the doses, the ways, of putting into, the longing of treatment
34. The urgent help for patients with botulism
35. The principles of writing out the patients from hospital
36. The prevention and the actions in the focus
37. Ways of enteroviral infection, rotaviral infection transmission.
38. Enterovirus and rotavirus pathogenicity factors and stages of enteroviral infection, rotaviral infection pathogenesis.
39. Stages of cyclic clinical course of enteroviral infection, rotaviral infection.
40. Main signs of enteroviral infection, rotaviral infection.
41. Specific complications of enteroviral infection, rotaviral infection.
42. Hemogram of patients with enteroviral infection, rotaviral infection.
43. Plan of examination of patients with enteroviral infection, rotaviral infection.
44. Methods of specific enteroviral infection, rotaviral infection diagnostics.
45. Etiotropic therapy of enteroviral infection, rotaviral infection
46. Principles of pathogenetic enteroviral infection, rotaviral infection therapy.
47. Prevention of enteroviral infection, rotaviral infection.
3.4.2. Tests for self-control

Choose correct answers:

**Variant 1**

1. Patient N. in 15 hours after consuming tinned mushrooms complains about appearance of muscular weakness, dry mouth, sickness, vomit, malfunction of eyesight. What investigation is needed urgently?:
   A. immune-enzyme analysis
   B. polymerase chain reaction
   C. bacteriological investigation of blood
   D. the Widal reaction
   E. biological test

2. Patient complains about weakness, dry mouth, diplopia, difficulty of swallowing. He has ptosis, light reaction of eyes is sluggish. The day before he ate dried fish. What do you begin treatment with?:
   A. antibacterial therapy
   B. antiviral therapy
   C. introduction of serum
   D. desintoxication therapy
   E. gastric lavage

3. Patient V. has Salmonellosis, localized form. All of following materials may be used for bacteriological investigation, except:
   A. blood
   B. feces
   C. vomit mass
   D. lavage liquid of stomach
   E. all of them may be used

4. Food toxicoinfection can be caused by:
   A. Proteus vulgaris
   B. Enterobacter
C. Citrobacter
D. Clostridium perfringens
E. everything is right

5. Patient was admitted to the hospital with suspicion about intestinal infection. The day before he ate uncooked egg. He complains about sickness, multiple vomit, frequent thin (watery) foamy stool green color. On examination: the patient is slack, his skin is pale, his temperature is 39.2°C, abdominal swelling, painfullness in ileocecal and periumbilical area. What is the most probable diagnosis?:
   A. typhoid fever
   B. salmonellosis
   C. botulism
   D. enterovirus infection
   E. rotavirus infection

6. What toxic factor, which Cl. botulinum form, is the main?:
   A. exotoxin
   B. endotoxin
   C. hyaluronidase
   D. neurominidase
   E. streptolysin

7. What is the reason of respiratory malfunction in patients with botulism?:
   A. edema-swelling of brain
   B. destruction of neurons of respiratory center
   C. lesion of motoneurons of chest and neck parts of spinal cord by toxin
   D. pneumothorax
   E. aspiration

8. What method of investigation is needed for early diagnostics of rotavirus infection?:
   A. bacteriological test of blood
   B. bacteriological test of feces
   C. immune-enzyme analysis for disclosure of rotaviral antigen in feces
D. serological methods for disclosure of rotaviral antibodies
E. all these tests are needed

9. Who can be source of rotavirus infection?:
A. infected human
B. cattle
C. birds
D. fish
E. everything is right

10. All of them can be source of salmonellosis, except:
A. infected human
B. cattle
C. birds
D. fish
E. everything can be so.

Keys:

<p>| | | | | |</p>
<table>
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</thead>
</table>

**Variant 2**

1. What antibacterial medicine is the most effective in treatment of salmonellosis?:
A. interferon
B. penicillin
C. chloramphenicol
D. cephazolin
E. ciprofloxacin

2. Generalized (septic) form of disease can be characterized for:
A. food toxicoinfection
B. salmonellosis
C. botulism
D. enterovirus infection
E. rotavirus infection.

3. Who can be source of enterovirus infection?:
   A. infected human
   B. cattle
   C. birds
   D. fish
   E. everything is right.

4. What clinical form of enterovirus infection is made out?:
   A. herpangina
   B. epidemic myalgia
   C. encephalitis
   D. enterovirus diarrhea
   E. everything is right.

5. What is the average incubation period of food toxicoinfection?:
   A. from 30 minutes till 24 hours
   B. 20-30 days
   C. 3-5 days
   D. 10-14 days
   E. 1-6 months.

6. Botulism is belonged to:
   A. Food toxic infections
   B. Blood infections
   C. Intestinal infections
   D. Virus infections
   E. Food intoxication

7. The exciter of botulism:
   A. The pale treponema
   B. The spindled stiek
   C. Vibrio coma
   D. Clostridium perfringens
   E. Clostridium botulinum

8. What toxins doe the exciter botulism secretion?
   A. Endotoxin
   B. The toxin for nervous system
   C. The anatoxin
   D. There is no secretion
   E. The exotoxin and the endotoxin

9. The cause of inspection for botulism
A. The food  
B. Peoples  
C. The water  
D. The animals with such food as grass  
E. The conserve products

10. The factors of transmission for botulism
A. Products with sports of exciter with anaerobe conditions  
B. The unqualitative vegetables  
C. The unwashed fruits  
D. All answers are right  
E. The milk products

**Keys:**

|---|------|------|------|------|------|

**3.4.3. Situational tasks of the second level learning  α -3**

**Task 1**

The patient N, 31 years old was delivered to the hospital by the ambulance. The state is serious, the patient is not dynamic. Ptosis is present. The skin is pale with bluish shade, the tonuses of skeleton muscles are lower, the heart tonus is a little, and there is extrasistoly, the frequency of heart contractions is 130 in minute, the frequency of breath is 40 in minute, the breath is superficial. The patient eated the clinic of food poisoning and upsetting of eyesight.

1. To formulate the preliminary diagnosis  
2. The plan of patient’s inspection  
3. The treatment

**Task 2**

Patient, 42 years old, has an acute form of the disease. He was complaining of rigor, hot flash, temperature increase up to 39°C, nausea, vomiting, frequent bulky and nauseous stool without any pathological tap, and ache in epigastric and
mesogastric areas. His sister had the same disease. 6 hours before sickness the patient ate a boiled duck, which had been stored for 12 hours at room temperature.

Results of examination: moderate severe condition, body temperature – 39,2°C, tissue tension is reduced, dry tongue with rich brown deposit. AT – 90/60 mm of mercury column, beat – 100 strikes/minute, very weak. Tenderness in epigastric zone, close to omphalus.

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

4. Materials for the class of independent work
4.1. List of study practical tasks to be performed in the practice:
4.1. List of practical training tasks to be done during the practical class:
- Study methods of examination of patient with infectious gastroenteritis
- Perform differential diagnostics of infectious gastroenteritis
- Make up a plan of laboratory examination
- Study the results of specific examination of patients with infectious gastroenteritis
- State the complications of infectious gastroenteritis.
- Make up a treatment plan for the patient with infectious gastroenteritis.
- Define medical approach in different complications of infectious gastroenteritis.

4.2.1. Professional algorithm of obtaining knowledge and skills of infectious gastroenteritis.

<table>
<thead>
<tr>
<th>№</th>
<th>Task</th>
<th>Sequence of action</th>
<th>Notices and warnings concerning self-control</th>
</tr>
</thead>
</table>


| 1. Study the methods of examination of patient with infectious gastroenteritis | I. Define the complaints of the patient.  
II. Define the history:  
1. Medical history  
2. Patient’s life history  
3. Epidemic history  
II. Perform proper examination.  
1. General examination:  
   1. General blood analysis.  
   2. General urine analysis  
   3. Feces culture and | Divide complaints attributable to syndromes of:  
- total toxicosis  
- organs attack  
- additional influences  
Pay attention to acute start, period, sequence of symptoms and intensiveness of  
- fever;  
- nausea  
- stomach-ache  
- vomiting  
- diarrhea  
- other symptoms  
Define pervious diseases  
Specify data related to fecal-oral mechanisms of transmission, pay attention to consumption of products that can constitute factors of germ transmission.  
Remember: presence, intensity and dynamics of the symptoms is related to the period and severity of disease progress, and they depend on age of the patient and comorbidity.  
Pay attention to:  
- weakness, adynamia and retardation of the patient;  
Pay attention to standard changes: leucocytosis, neutrocytosis, and IPT acceleration | 2. Examine the patient | 3. Prescribe laboratory and other testing, estimate their results |
4. Serological methods:

- Sensitivity, vomited matter, lavage water and food products

Increase of specific weight

Prescribed regardless of disease period during fever period, but, recommended as soon as possible

Positive since the first day of the disease.

Prescribed in paired serums with an interval of 10 days

At FTI – with autohemagglutinin– titer increase

At rotaviral infection – ELISA (Ag in feces)

---

4.2.2. Professional algorithm of obtaining knowledge and skills of botulism.

<table>
<thead>
<tr>
<th>№</th>
<th>The assignment</th>
<th>The order of doing</th>
<th>The remarks and warning for self-control</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>To master the methodic of clinical inspection for patients with botulism</td>
<td>The complains</td>
<td>To detachment complains which characterized such syndromes as - the dyspepsia - the ophtalmoplegic - the phagoplegic - phonolaryngoplegic</td>
</tr>
<tr>
<td></td>
<td></td>
<td>The anamnesis of illness</td>
<td>To disregard to beginning, time, the gradual of appearance next symptoms, it’s dynamic</td>
</tr>
</tbody>
</table>
| **Anamnesis of life** | - the diplopia  
- the disphagia  
- the disartria  
- the transgression of salivation  
- the transgression of breath  

To discover the facts about mechanisms of transmission with the help of evacuation and mouth. The patient’s food such as conserved products smoke sausages, the salt fishes |
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td><strong>2 To make the curation for patient</strong></td>
<td><strong>To make the objective inspection</strong></td>
</tr>
<tr>
<td><strong>The general inspection</strong></td>
<td>The expression, the dynamics of symptoms are caused by the period, the seriousness of movement illness</td>
</tr>
<tr>
<td><strong>To disregard to</strong></td>
<td>To disregard to</td>
</tr>
</tbody>
</table>
| - squint-eyes  
- the ptos  
- the anizokoria  
- the nistagm  
- the snuffle voice, the disartria  
- the voice is not expressive |
| **The digestive system** | To disregard to |
| - the slime cover in the match  
- the swilling  
- the bolt  
- the absence of peristaltic noise |
| **The nervous system** | To disregard to |
| - the bifurcating in eyes  
- «net», «fog» before eyes  
- the midriaz  
- the strobism  
- the transgression of swallow  
- the transgression of talking  
- the miastenia  
- the transgression of breath |
| **The system of heart and vessels** | To disregard to |
| - tones are deaf a little  
- the tachycardia  
- the widening of heart borders |
4.3. Materials methods of the final phase of occupation

4.3.1. Objectives of the 3rd level

Task 1

The patient K, 38 years old became to the hospital with complains of headache, weakness, giddiness, «ness», «fog», the bifurcating of objects, the deterioration of eyesight. Then the hard breath and the dryness in the mouth appeared. The patients’ eated the conserved mushrooms the days before. There are the pots, midriaz anizokoria, the snuffcle voice, the unexpressive talk. During the auscultation there are the hard breaths. The frequency of breath is 28 in minute; the tones are the deaf a little. There is the widening of heart borders bluntness in the left. The frequency of heart contractions is 95 in minute. The artery pressure is 140/95 mm of mercury column.
1. To formulate the preliminary diagnosis
2. The plan of patient’s inspection
3. The treatment

**The right answers**

1. The food botulism, the ophthalmologic, the pharyngoplegic and the phonologic syndromes, the serious movement.
2. The blood for reaction of neutralization on white mousses.
3. The irrigation of stomach and intestines by sode solution. The wheys opposite botulism (10000 ME for types A, E, 5000 ME for type B in veins with the internal of 12 hours. The desintosication therapy, the levomicetin for 0,5g times a day during 5 days, the oxygen therapy, the symptomatic treatment).

**Task 2**

Patient, 40 years old, has an acute form of the disease. He was complaining of rigor, hot flash, temperature increase up to 39°C, nausea, vomiting, frequent bulky and nauseous stool without any pathological tap, and ache in epigastric and mesogastric areas. His sister had the same disease. 6 hours before sickness the patient ate a boiled duck, which had been stored for 12 hours at room temperature.

Results of examination: moderate severe condition, body temperature – 39,2°C, tissue tension is reduced, dry tongue with rich brown deposit. AT – 90/60 mm of mercury column, beat – 110 strikes/minute, very weak. Tenderness in epigastric zone, close to omphalus. Rear convulsions of calcaneal muscles.

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

**The right answers**

1. Methods of laboratory diagnostics of generalized forms of salmonellosis
   - bacteriological: blood, bile and urine culture and sensitivity, if required – spinal liquid, apostem content;
   - serological: agglutination test, PHT with antigen of salmonellosis
2. Diagnostics of salmonellosis subclinical form
- separation of Salmonella from feces and presence of diagnostic titers of anti-Salmonella antigens in serological reactions when clinical signs of the disease are absent

3. Specific diagnostics of food toxicoinfections, caused by opportunistic flora
- simultaneous sampling of material from the patient and suspicious product;
- testing of one germ attributable to all patients;
- reduced amount of germ in feces in the course of recovery;
- increased antiserum activity with autohemagglutinin

5. Materials of after-work

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

- "Prospects for early diagnosis of infectious gastroenteritis »
- "Clinical and epidemiological characteristics of infectious gastroenteritis "
- "Differential diagnosis of infectious gastroenteritis "
- «Pathogenesis of complication of infectious gastroenteritis "
- "Toxic shock. Etiopathogenesis, clinical manifestations, intensive treatment"
- "Dehydratation shock. Etiopathogenesis, clinical manifestations, intensive treatment".
CHOLERA.

DEHYDRATION SHOCK IN PATIENTS WITH CHOLERA

1. Actuality of theme

Cholera is one of the oldest human diseases. It is second only to the plague in the history of human disasters tends to pandemic spread is quarantine infections. From 1817 to 1925 recorded 6 pandemics caused by classical Vibrio cholerae. The seventh pandemic, which began in 1961 and caused biovar El Tor, covered more than 180 countries and continues to the present. During this period, the disease has penetrated from Asia to Africa, Europe, the Americas, the number of patients reached 2 million.

Cholera registered in Ukraine since 1965, with the greatest activity in the early 70's and re-rise of incidence in 1994.

Relevance of cholera caused by common susceptibility, ease of transmission, the need for early diagnosis and prompt adequate therapy in the absence of any disease progresses rapidly, leading to death of the patient. Lack of sustained immunity after the disease does not exclude re-infection, in the event of epidemics requires large material costs and a complex of preventive measures. Lack of awareness of physicians regarding cholera and experience recognition of the disease leads to frequent diagnostic mistakes, especially in sporadic cases, and low hygienic standard of living, migration, shortcomings in public services can contribute to the spread of infection.

The emergence in the 90s cholera outbreaks caused by serogroup O139 Bengal vibrios (Asia ) and the importation of the disease in other countries , in particular Russia and genetic diversity of vibrio cholera epidemic and the possibility of selection of significant strains can not hope soon to prosperity by cholera in the world.

Etiology: Vibrio cholerae, 2 biotypes - classical (true) Vibrio cholerae classica and Vibrio cholerae El Tor.
Epidemiology: the source of the cholera vibrio - only people (patients and carriers), the mechanism of transmission - fecal - oral (through contaminated water, food).

Pathogenesis: the gate of infection - the digestive tract. Vibrio overcome the gastric barrier, reach the small intestine and begin to multiply rapidly, highlighting the exotoxin (choleragen) and neuraminidase. Cholera toxin binds to a specific receptor enterocytes - ganglioside. Neuraminidase enhances the action of cholera, by modifying the receptor. The complex choleragen - specific receptor activates adenylate cyclase, which, with the participation of prostaglandins increases the formation of cyclic adenosine monophosphate (AMP). AMP regulates the pump by ion secretion of water and electrolytes from the cells into the lumen. As a result of the small bowel mucosa begins to secrete a lot of isotonic fluid, which does not have time to absorb the large intestine. It starts with profuse diarrhea isotonic fluid. Fluid loss reaches 1 1 hour. As a result of decreased blood volume develops intracellular dehydration and the subsequent development of shock and acute renal failure.

Clinic: incubation period usually 1-5 days.

The forms of the disease: subclinical, mild, moderate, severe and very severe, are determined by the degree of dehydration (I degree - loss of fluid volume equal to 1-3 % of the mass of the body - worn and mild forms , II degree - 4.6 % - moderate , grade III - 7.9 % - severe and level IV - over 9 % - very severe). 

a) worn shape - can only be a single loose stools in a good state of health of patients and the absence of dewatering .

b) mild - acute onset of fever and without prodromal phenomena. The first clinical signs are sudden urge to defecate and discharge of mushy, or from the beginning, watery stools. In subse - ponding these repeated urgency; they are not accompanied by pain. The stools are easily distinguished, the intervals between bowel movements are reduced, and the amount of feces each time increases. The stools have a kind of «rice water »: translucent, unclear and white color, sometimes with floating flakes of gray color, without odor or the smell of fresh water. Patient
notes rumbling and discomfort in the umbilical region. Defecation is repeated at most 3-5 times a day, the general state of health is satisfactory, slight feeling weak STI, thirst, dry mouth. Duration of illness is limited to 1-2 days.

c) the average weight - the disease progresses to diarrhea vomiting attached form "congee", increasing in frequency, not accompanied by any strain and nausea. With the addition of vomiting dehydration (exsicosis) is progressing rapidly. Thirst is a painful, dry tongue with a «touch of chalk", skin and mucous membranes of the eyes and pale oropharynx, skin turgor decreases, the amount of urine decreases to anuria. Chair up to 10 times a day, a rich, increases in volume. There are single leg cramps, hands, feet, chewing muscles, unstable cyanosis of the lips and fingers, hoarseness. Developing moderate tachycardia, hypotension, oliguria, hypoxia. Disease lasts 4-5 days.

d) severe - pronounced signs exsicoses because of the very rich (1-1.5 liters per defecation) chair that becomes so from the very first hours of the disease, and the same abundant and repeated vomiting. Patients concerned about the painful muscle spasms of the limbs and abdominal muscles that go from rare to frequent clonic and even mixtures tonic convulsions. Voice weak, thin, often barely audible. Decreased skin turgor, pleated leather long straightened. The skin of the hands and feet becomes wrinkly - "hand laundress." A person takes a characteristic of cholera form sharpened features, sunken eyes, cyanosis of the lips, ears, earlobes, nose. On palpation of the abdomen are determined by the liquid from the intestines, increased rumbling, and splashing liquid. The liver and spleen were not enlarged. Appears tachypnea, tachycardia increases to 110-120 beats/ min. Pulse weak filling ("thready"), cardiac deaf, blood pressure progressively falls below 90 mm /Hg. Art. first maximum, minimum, and then pulse. The body temperature is normal, decreased urine output, and soon stopped. Thickening of the blood is expressed moderately. The relative density of the plasma, the index hematocrit and blood viscosity at the upper limit of normal or moderately increased. Expressed hypokalemia plasma and red blood cells, hypochloremia, reasonable compensatory hypernatremia plasma and red blood cells.
d) very severe (algidnaya) - differs sudden rapid disease progression, starting with the massive uninterrupted stools and copious vomiting. After 3-12 hours in a patient develops a serious condition algida, which is characterized by lower body temperature 34-35,5 ° C, very dehydrated (patients lose up to 12 % of body weight - dehydration IV degree), shortness of breath, and impaired hemodynamics anuria the type of hypovolemic shock. At the time of admission of patients to the hospital when they develop paresis of the muscles of the stomach and intestines, as a result of which the patients stopped vomiting (replaced convulsive hiccups), and diarrhea (gaping anus, free flow "of intestinal water" from the butt-hole no go with a light pressure on the abdominal wall). Diarrhea and vomiting occur again in the background or after rehydration. Patients are in a state of prostration, drowsiness goes into stupor, then to come. Disturbance of consciousness coincides with breathing - from the surface to the frequent pathological memory - minute breathing (Cheyne-Stokes, Biot). Color of the skin gets ashy hue (cyanosis of the total), there are "sunglasses around the eyes", the eyes sunken, the sclera appear dull, eyes unblinking, his voice is absent. The skin is cold and clammy to the touch, the body is reduced seizures (posture "champion" or "Gladiator" as a result of general tonic convulsions). Stomach in, determined by palpation twitching rectus abdominis. Painful cramps worse even with mild abdominal palpation, which is a concern of patients. There is a pronounced hemoconcentration - leukocytosis (up to 20 x 10^9 / l), the relative density of the plasma reaches 1,035-1,050 index hematocrit 0,65-0,7 l / l. The level of potassium, sodium and chlorine is reduced significantly (hypokalemia 2.5 mmol/l), decompensated metabolic acidosis.

Diagnosis: Epidemiological anamnesis (cholera outbreak), clinical, bacteriological study (feces and vomit), serological methods (mainly used for retrospective diagnosis) and agglutination titer determination of vibriocidal antibodies, immunofluorescence, IHA, etc.

Treatment: The main principles: a) restoration of circulating blood volume, b) reducing the electrolyte composition of tissues, c) the impact on the pathogen.
1. It starts in the early hours of the disease, severe hypovolemia should be carried out once the primary rehydration - intravascular infusion therapy warmed polyionic solutions (Trisol, laktosol, Disol, hlosol, "WHO solution" - to 1 liter of pyrogen-free water 4 g of sodium chloride, 1 g of potassium chloride, 5, 4 g of sodium lactate and 8 g glucose). Introduction of pressor amines (epinephrine, phenylephrine, etc.) is contraindicated. In mild rehydration - oral.

2. Secondary rehydration is carried out on the basis of loss of fluid from the patient vomit, feces, urine, through the skin, breathing (requires the collection and measurement of emissions).

3. When the pyrogenic reactions (chills, fever), the introduction of the solution does not stop, this is added to a solution of 1% dimedrol (1-2 ml) or pipolfen. At the very pronounced reactions prescribed prednisolone (30-60 mg/daily).

4. Termination criterion is water-salt therapy - the appearance of stool fecal character in the absence of vomiting and the prevalence of incontinence of bowel movements over the last 6-12 hours.

5. AB-therapy: tetracycline for 0.3-0.5 g every 6 hours for 3-5 days or doxycycline 300 mg once daily, may also be quinolones (ofloxacin)

Discharge criteria: clinical recovery and three negative bacteriological feces, bile study once. In the food industry, water supply, children's and medical institutions stool tested five times (for five days) and bile once.

2. Study purpose of practical studies:

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

• have an idea: a place in the structure of cholera infections, the study of history, scientific contribution of domestic scientists, including members of the Department of Infectious Diseases KNMU during an outbreak of cholera in the Crimea, in the history of research in this area.

• read: the statistical data on the prevalence of cholera, mortality, frequency of complications in Ukraine and in the world.
2.2. **Student have to know:**

- etiology of cholera, pathogenicity factors of the pathogen;
- epidemiology of cholera and features of the sixth pandemic;
- pathogenesis of cholera, dehydration shock;
- classification and clinical forms of cholera;
- clinical manifestations of cholera in various stages of dehydration;
- complications of cholera;
- clinical features and laboratory examination of patients with cholera;
- research methods used for the specific diagnosis of cholera;
- rules for fencing material for laboratory studies of patients with cholera;
- clinical and laboratory diagnosis of dehydration shock;
- principles of treatment of cholera;
- clinical management of patients in case of dehydration shock;
- prognosis of cholera;
- principles of prevention of cholera;
- rules for convalescents discharge from hospital;
- rules of the observation convalescents;
- principles of cholera hospital.

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

Follow the rules of work at the bedside of cholera;

- Collect history of the disease with the evaluation of epidemiological data;
- examine the patient and determine symptoms and syndromes cholera;
- Determine the degree of dehydration substantiate the clinical diagnosis;
- to provide differential diagnosis of cholera;
- on the basis of clinical examination time to recognize possible complications;
- draw medical documentation for the establishment of preliminary diagnosis of "cholera" with

  Compliance with all regulations concerning quarantine infections;
- a plan of laboratory examination of patients with cholera;
• interpret the results of laboratory examination with assessment ionograms;
• draw up a treatment plan based on the degree of dehydration, comorbidity, presence of complications;
• provide pre-hospital emergency care to the stage;
• a plan of preventive and prophylactic measures at the source of infection;
• make recommendations about treatment, diet, surveillance convalescence period.

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

<table>
<thead>
<tr>
<th>Discipline</th>
<th>To know</th>
<th>To able</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microbiology</td>
<td>Features classic cholera and El Tor vibrio, methods of specific diagnostics of infectious cholera</td>
<td>Borrow material for research, interpret the results of specific methods for diagnosis of cholera</td>
</tr>
<tr>
<td>Physiology</td>
<td>Parameters of physiological norm of human organs and systems, rates</td>
<td>Assess data of laboratory inspection.</td>
</tr>
<tr>
<td>Field</td>
<td>Content</td>
<td>Interpretation</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Laboratory tests are normal</td>
<td>(total blood count, urinalysis, blood chemistry, KLS parameters, electrolytes, etc.).</td>
<td></td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>The mechanism of dysfunction of organs and systems in the development of dehydration in cholera.</td>
<td>Interpret pathological changes on the results of laboratory examination in violation of the organs and systems of various origins.</td>
</tr>
<tr>
<td>Immunology and Allergology</td>
<td>Basic concepts of the subject, the role of the immune system in infectious process, influence at the time of elimination of the pathogen from the human body.</td>
<td>Rate data immunological studies.</td>
</tr>
<tr>
<td>Epidemiology</td>
<td>Epidemic process (source, mechanism of infection, routes of transmission) with cholera, the concept of the epidemic, pandemic, prevalence of cholera in the world and in Ukraine.</td>
<td>Collect epidemiology history, provide preventive and anti-epidemic measures in the focus of infection, follow the rules of cholera in the hospital.</td>
</tr>
<tr>
<td>Propaedeutics Internal Medicine</td>
<td>Methods and main stages of clinical examination of the patient.</td>
<td>To collect anamnesis, conduct the clinical examination of the patient, to detect pathological symptoms and syndromes.</td>
</tr>
<tr>
<td>Clinical pharmacology</td>
<td>Pharmacokinetics and pharmacodynamics, side effects of doxycycline, erythromycin and others. Of pathogenic (rehidratatiynoyi) therapy.</td>
<td>Prescribe treatment depending on the degree of dehydration, individual characteristics, age of the patient, to choose the optimal method, the rate and extent of drug administration, write recipes.</td>
</tr>
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<td>-------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Urology</td>
<td>Clinical and laboratory signs of ARF, ARF stage, causes of ARF in cholera</td>
<td>Diagnose, assess the damage, assign the appropriate assessment and treatment</td>
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<tr>
<td></td>
<td></td>
<td>Subsequent disciplines</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Internal integration</td>
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</tbody>
</table>
3.2 Theme contents.

**Dehydration syndrome**

**Etiology**
- Shigellosis, cholera, salmonellosis, ptomaine, colibacillosis, etc.

**Pathogenesis**
- Exotoxin, endotoxin, enterotoxin
- Microcirculation abnormality
- DIC
- Cyclic adenosine monophosphate activation
- Hypoxia acidosis
- Liquid outlet into the bowels
- Vomiting, diarrhea
- Isotonic dehydration

**Clinical presentation**
- Thirst, xeromyceteria and xerostomia, vomiting, diarrhea, spasms, tachycardia

**Multiple organ failure**
- Traffic noise index

**Diagnosis**
- Clinical blood analysis
- pH of the capillary blood
- Electrolytes of the blood plasma

**Treatment**
- The identification of the dehydration degree, reduction of the fluid balance and permanent control of its condition
- The maintenance of the fluid balance, reduction of the fluid loss
- The prescription of the antimicrobial medicine internally
- The supply of adequate food for a patient
3.3 Literature recommended:

Main sources:
1. Lectures of Professor.

Additional sources:


### 3.4. Self-control materials

#### 3.4.1. Questions to be answered

1. What group of infectious diseases at the source of infection is cholera?
2. The mechanism of infection, ways and factors of transmission of cholera.
3. The etiology of cholera, pathogenicity factors of the pathogen.
4. The stages of the pathogenesis of cholera.
5. Pathological changes in organs and tissues with cholera.
6. Features of exotoxin - choleragen.
7. The degree of dehydration in cholera.
8. Supporting clinical symptoms of cholera.
9. Atypical forms of cholera.
10. Clinical characteristics of light and deleted forms of cholera.
11. Clinical characteristics of cholera moderately.
12. Clinical characteristics of severe cholera.
13. Cholera clinical hidu.
14. Peculiarities of cholera in children, the elderly and persons with severe concomitant diseases (cardiovascular disease).
15. Complications of cholera.
17. The prognosis for cholera.
18. Algorithm examination of patients with suspected cholera.
19. Methods of specific diagnostics of cholera.
20. The main stages of the treatment of cholera.
22. Solutions used for oral and parenteral rehydration.
23. Methods for calculating the volume of rehydration therapy.
24. Terms of rehydration at different stages of dehydration.
27. Terms of convalescents discharge from hospital.
28. Clinical supervision for those who recover from cholera.
29. Main areas of prevention of cholera.
30. What diseases are attended by a dehydration shock.
31. Dehydratation syndrome pathogenesis stages.
32. The basic clinical symptomatology of a dehydration shock.
33. Name the dehydration degrees depending on fluid loss.
34. The main lethality causes a dehydration shock.
35. The examination plan of the patient with a dehydration shock.
36. Hemogram of the patient with a dehydration shock.
37. The differential diagnosis of the dehydrationous shock.
38. The definition of the dehydrationous shock.
39. The clinical picture of the dehydrationous shock.
40. A dehydration degree estimation of the patient with a diarrhea at a pre-admission.
41. The stages of conducting the patient with dehydration.
42. A complex of medical measures at a dehydration shock.
43. What solutions are needed to be used for the rehydration?

3.4.2. Self-control tests

**Variant 1**

1. The incubation period for cholera (in days):
A. 1-5,
2. The main cause of death of patients with cholera:
   A. infectious-toxic shock
   B. dehydration shock
   C. acute renal failure
   D. edema and swelling of the brain
   E. acute hepatic failure.

3. The main cause of diarrhea in patients with cholera:
   A. increased vascular permeability in the intestine
   B. hypercatharsis
   C. high osmotic pressure in the lumen of the intestine
   D. disruption of intestinal enzyme systems (adenylate cyclase)
   E. increased production of water and electrolytes into the lumen of the intestine.

4. The main factor in the pathogenicity of cholera:
   A. flagella
   B. mutsinaza
   C. neuraminidase
   D. exotoxin
   E. all true.

5. A typical complication for cholera:
   A. edema and brain swelling
   B. pulmonary edema
   C. an infectious-toxic shock
   D. dehydration shock
   E. all true.

6. The material can be identified Vibrio cholerae:
   A. blood
B. urine
C. sputum
D. liquor
E. feces and vomit.

7. Relative density of the plasma in the norm:
   A. 1,022-1,023
   B. 1,024-1,025
   C. 1,027-1,028
   D. 1,030-1,035
   E. 1,020-1,030

8. Which drug should be used for urgent rehydration therapy?
   A. disol
   B. isotonic sodium chloride solution
   C. trisol
   D. chloramphenicol
   E. 5% glucose solution.

9. For emergency prevention of cholera for the contact persons in the hearth is used:
   A. ampicillin
   B. furazolidone
   C. quinine
   D. ftalazol
   E. doxycycline.

10. For laboratory confirmation of cholera is used:
    A. bacteriological examination of stool
B. blood culture
C. bacteriological examination of urine
D. study coprological
E. determination of electrolytes in the blood.

**Keys:**

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**Variant 2**

1. Criteria for discharge of patients with cholera:
   A. clinical recovery
   B. 3 negative fecal bacteriology
   C. 3 negative bacteriology of bile
   D. 3 negative fecal bacteriology, once the gall, clinical recovery
   E. 5 negative bacteriological studies of feces.

2. Duration of isolation of contact persons?
   A. 5 days
   B. 6 days
   C. 10 days
   D. 14 days
   E. 21 days.

3. For emergency prevention of cholera for the contact persons in the hearth is used:
   A. ampicillin
   B. furazolidone
   C. quinine
   D. ftalazol
   E. doxycycline.
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   A. bacteriological examination of stool
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   C. bacteriological examination of urine
   D. study coprological
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   A. blood
   B. urine
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6. Relative density of the plasma in the norm:
   A. 1,022-1,023
   B. 1,024-1,025
   C. 1,027-1,028
   D. 1,030-1,035
   E. 1,020-1,030.
7. The incubation period for cholera (in days):
   A. 1-5,
   B. 7-14,
   C. 10-21,
   D. 6-10,
   E. 45.
8. The main cause of death of patients with cholera:
   A. infectious-toxic shock
   B. dehydrationous shock
   C. acute renal failure
   D. edema and swelling of the brain
   E. acute hepatic failure.
9. The main cause of diarrhea in patients with cholera:
A. increased vascular permeability in the intestine
B. hypercatharsis
C. high osmotic pressure in the lumen of the intestine
D. disruption of intestinal enzyme systems (adenylate cyclase)
E. increased production of water and electrolytes into the lumen of the intestine.

10. The main factor in the pathogenicity of cholera:
A. flagella
B. mutsinaza
C. neuraminidase
D. exotoxin
E. all true.

**Keys:**

|---|------|------|------|------|------|

**Variant 3**

1. The material can be identified Vibrio cholerae:
A. blood
B. urine
C. sputum
D. liquor
E. feces and vomit.

2. Relative density of the plasma in the norm:
A. 1,022-1,023
B. 1,024-1,025
C. 1,027-1,028
D. 1,030-1,035
E. 1,020-1,030
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   D. dehydration shock
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10. Which drug should be used for urgent rehydration therapy?
   A. disol
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   D. chloramphenicol
   E. 5% glucose solution.

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

**Task 1**

Patient K., 30 years old, acutely ill after returning from Pakistan. The disease began with frequent watery stools without pathological impurities and odors that came without any painful events of the abdomen.

T 36,4°C body, BH -18 / min, BP - 120/70 mm Hg., Pulse - 80 beats / min. Above the lungs vesicular breathing, cardiac sonorous, rhythmic. Abdomen soft, smooth.
It is noted rumbling in the periumbilical area. Sections of intestine is not spasmodic, painless.

1. Preliminary diagnosis
2. Pattern of examination.

**Task 2**

Patient M., 32 years old, suffering from the first day, complained of vomiting, frequent, copious stools, dry mouth, cramps in the lower extremities. The disease began suddenly with uncontrollable diarrhea and vomiting. Before eating tomatoes bought at the market.

T body – 36,2\(^0\) C , BP - 90/60 mm Hg , pulse - 100 beats / min. His facial features are sharp, dry tongue, voice - hoarse, the skin is taken in the fold that slowly crushes. Acrocyanosis, cold extremities. Abdomen smooth. From the onset of the disease was not urine.

1. Preliminary diagnosis
2. Pattern of examination.

**Task 3**

Patient P., 22 years old, admitted to the infectious diseases hospital on the first day of illness with complaints of general weakness, once vomiting, and diarrhea. It was found that her brother and mother are in hospital with infectious intestinal infection.

T – 36,8\(^0\) C., pulse - 82 beats / min , BP - 110/80 mm Hg. century. Normal skin color, turgor of low, wet tongue, slight thirst. Palpation pain along the intestine is not defined. Stool as "rice," and odorless.

1. Preliminary diagnosis
2. Pattern of examination.
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
<table>
<thead>
<tr>
<th>№</th>
<th>Task</th>
<th>Execution sequence</th>
<th>Annotation, notices for self-checking</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>To seize a technique of cholera patient clinical examination, a patient with a dehydration shock</td>
<td>I To find out the patient complaints. &lt;br&gt;II To find out an anamnesis: 1. Medical history 2. Patient history. 3. Epidanamnesis.</td>
<td>To separate complaints that characterised cholera, dehydration syndrome. &lt;br&gt;To pay attention to: &lt;br&gt;Origin succession, symptoms dynamic peculiar for a dehydrationous shock, an enterorrhagia. &lt;br&gt;To find out commemorative diseases. &lt;br&gt;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.</td>
</tr>
<tr>
<td>2</td>
<td>To carry out a patient curation</td>
<td>II To carry out an objective examination. &lt;br&gt;1. Common examination: - patient general condition; - skin, fauces mucous tunic</td>
<td>Remember: presence, evidence, symptoms dynamic, provided with term and severity disease course, depend on patient age, accompanying pathology. &lt;br&gt;To pay attention for: &lt;br&gt;- slackness, adynamy, the patient block; &lt;br&gt;- a body temperature; &lt;br&gt;- the skincyanosis, acute lowered turgor. &lt;br&gt;To pay attention for: &lt;br&gt;- tongue is furred with white incrustation; &lt;br&gt;- dry tongue. &lt;br&gt;- Peritonitis symptoms (presence...</td>
</tr>
</tbody>
</table>
2. Digestive system:
- tongue examination;
- abdomen percussion;
- abdomen palpation;
- excrements characteristic.

3. Cardiovascular system:
- pulse;
- blood pressure;
- heart auscultation;
- quantity, character, propensity to constipation (occurrence melena testifies to an enterorrhagia).

To pay attention for:
- a tachycardia;
- moderately lowered blood pressure (considerable decrease testifies to complications);
- moderate dullness of heart sounds

To set laboratory and additional researches, to interpret the results:
1. Complete blood count
2. Clinical urine analysis

To pay attention of typical changes:
- leukopenia, or leukocytosis, lymphocytosis, aneosinophilia, thrombocytopenia (leukocytosis, anemia, ESR, appear in case of development enterorrhagia).

The absence of significant changes in typical course

<table>
<thead>
<tr>
<th>Dynamics of clinical symptoms of cholera</th>
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<tbody>
<tr>
<td>Index / degree of dehydration</td>
</tr>
<tr>
<td>the decrease in body</td>
</tr>
<tr>
<td>weight</td>
</tr>
<tr>
<td>--------</td>
</tr>
<tr>
<td>sonorous voice</td>
</tr>
<tr>
<td>cyanosis of the skin and mucous</td>
</tr>
<tr>
<td>T of body</td>
</tr>
<tr>
<td>pulse</td>
</tr>
<tr>
<td>blood pressure</td>
</tr>
<tr>
<td>diuresis</td>
</tr>
<tr>
<td>frequency of vomiting</td>
</tr>
<tr>
<td>frequency of stool</td>
</tr>
</tbody>
</table>

**Laboratory criteria depending on the degree of dehydration**

<table>
<thead>
<tr>
<th>Index / degree of dehydration</th>
<th>I</th>
<th>II</th>
<th>III</th>
</tr>
</thead>
<tbody>
<tr>
<td>hematocrit levels</td>
<td>before 54%</td>
<td>before 65%</td>
<td>more than 65%</td>
</tr>
<tr>
<td>relative density plasma</td>
<td>before 1,029 gr/sm3</td>
<td>before 1,035 gr/sm3</td>
<td>more than 1,035 gr/sm3</td>
</tr>
<tr>
<td>pH blood, unit</td>
<td>before 7,36</td>
<td>before 7,30</td>
<td>less than 7,30</td>
</tr>
<tr>
<td>electrolyte shifts</td>
<td>hypokalemia +</td>
<td>hypokalemia ++</td>
<td>hypokalemia, Hyponatremia ++ +</td>
</tr>
</tbody>
</table>
4.3. Materials methods of the final phase of occupation

4.3.1. Objectives of the 3rd level

Task 1

Two girls came to a hospital, because they had 38 °C fever, a headache, weakness, dizziness, and a pain in epigastry and round a navel, a nausea, vomiting 3 times, excrements 4 times per a night, watery diarrhea, foamy, fetid, with mucus impurity. It is known from the history that the day before the girls ate pastries with cream which were not stored in a refrigerator. Objectively: a tongue is dry, furred by white touch, the stomach is bloated moderately, rumbles in palpation, painful in epigas, pulse is 80 bpm, and the blood pressure is 110/70 mm mercury column

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

Task 2

Patient A, 40 years old, is admitted to an infectious hospital. Objectively: he is apathetic, adynamic, the consciousness is dulled. The tongue has teeth prints on the lateral surfaces and is furred up with grey-brown incrustation. The stomach is bloated, painful in palpation, liver and spleen increase. According to his wife words her husband is being ill for 4 days. He fell ill acute, the temperature had raised to 38,5 °C, repeated bile vomiting, he felt a pain in epigastry and paraumbilical areas, then he had a diarrhea, excrements were to 10 times per a day, diarrhea is watery, foamy, fetid, with slime impurity. The day before illness the man ate the soft-boiled goose eggs and mayonnaise.

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

Patient C, 18 years old, complains of frequent excrements. The diarrhea has begun acute, unexpectedly. He has 20-25 watery excrements per a day, reminding "rice water". Then repeated vomiting without a previous nausea appeared. Vomiting also reminds "rice water". The weakness, dry mouth, and thirst are observed. A body temperature is 36,5 °C. The epidanamnesis found, that 2 days ago the patient had visited his grandmother in a village where he took water from an open reservoir. On the view: the skin gets a bluish tinge, cold by touch, turgor is lowered. He has painless retracted abdomen. The patient has a tachypnea, a tachycardia, a low blood pressure, an oliguria, and gastrocnemius muscles spasms.

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

Task 4

Patient K, 38 years old, is delivered by an ambulance car in a grave condition in 4 hours from the illness beginning. Disease has begun with nausea, repeated vomiting (10 times), a plentiful watery diarrhea (8 times). The body temperature is 37,8 °C. Disease development connects with the duck eggs eating boiled a week ago. While being hospitalised he felt thirsty. The skin pallor attracted attention, sharp features, and hoarse voice. A skin is cold by touch, pulse of poor volume, 120 bpm. The patient body temperature at the moment of arrival to a department was 36,0 °C. The blood pressure is 80/40 mm mercury column. The breath is 24 per minute. Skin turgor is lowered, the folds unknit slowly. An acute pain and gastrocnemius muscles spasms. It is a diffuse rumble in stomach palpation. The continuation of intensive diarrhea and vomiting, vomit masses are watery, whitish, and turbid. The patient condition has considerably improved after the massive rehydratic therapy.

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

Task 5

Patient K, 20 years old, complains of a diarrhea without a stomach pain which has begun acute a day ago. The stool is more than 20 times per a day, watery feces, reminding "rice water", and repeated vomiting without a previous nausea. Weakness, dry mouth, thirst are observed. A body temperature is 36,0 °C. The epidamnesis found, that 2 days before disease the patient communicated with another patient who had similar clinic. On the view: a skin is pale with a bluish tinge, turgor is lowered, he has washerperson's effect, sharp features, hoarse voice, and gastrocnemius muscles spasms. His blood pressure is 70/50 mm mercury column, pulse of poor volume, 130 per mines, tachypnea.

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. A clinical salmonellosis, gastro-intestinal form, gastroenterocolitic variant, moderately severe, the I degree dehydration.
2. Bacteriological research of feces, vomit masses, food debris (pastry); RNGA with salmonellosis diagnosticum, coprogramm.
3. Diet 5, regidron to 1 l per a day, 1 tablespoon of a polifepan in an hour before food intake three times per a day, enzymes (mezim, pankreatin, pepzim, etc.) during food intake.

Task 2

1. A salmonellosis, generalized form, typhoid variant with intestinal manifestations, a severe clinical course.
2. Bacteriological research of feces, stomach percolates, blood, food debris (goose eggs, mayonnaise).
3. Disintoxication therapy (salt solutions, reopoliglukin, 5 % glucose), chloramphenicol succinite to 6 g per a day, 4,0-6,0 ampicillin per a day.

**Task 3**
1. A cholera, the typical form, gastroenteritis, a severe clinical course. The III degree dehydration.
2. Bacteriological research of feces, vomit masses. Conglutination reaction, RNGA, IFA.
3. Salt solutions intravenously (trisalt, acesalt, quatrasalt, etc.) before the stopping of vomiting, a diarrhea, haemodynamics of restoration and diuresis. In due course - 1,2 g tetracycline per a day or 0,2 g doxycycline per a day over a 5-day period.

**Task 4**
1. A salmonellosis, gastro-intestinal form of severe degree, the III degree dehydrationous shock.
2. The clinical blood analysis with hematocrit and plasma density definition, the biochemical blood analysis with ionogram; bacteriological research of feces, vomit masses, onglutination reaction with salmonellosis diagnosticum.
3. Salt solutions intravenously introduction (trisalt, acesalt, quatrasalt, etc.) to 6 % of a patient weight, heparin 100 ED/KG, prednisolone 3 mg/kg per a day, trasilol, 1,0 intramuscularly chloramphenicol succinite 4 times per a day.

**Task 5**
1. A cholera, the typical form, a heavy current. Dehydration III degrees.
2. Bacteriological research of feces, vomit masses. Conglutination reaction, RNGA, IFA. The accelerated methods: immobilization and microagglutination 0-vibrionov by anticholeraic serum, the RIF.
3. Rehydratic fluid maintenance by salt solutions (trisalt, disalt, quatrasalt, etc.) before the diarrhea and vomiting stopping, then antibacterial therapy (0,3 tetracycline 4 times per a day or 0,1 doxycycline 2 times) throughout 5 days is possible.
5. Materials of after-work

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

"Prospects for early diagnosis of cholera»

"Clinical and epidemiological characteristics of cholera"

"Differential diagnosis of cholera"

«Pathogenesis of dehydration shock"

"Dehydration shock. Etiopathogenesis, clinical picture, intensive care"
SHIGELLOSIS. AMEBIASIS.

PSEUDOTUBERCULOSIS. INTESTINAL YERSINIOSIS

Actuality of theme

Shigellosis is one of the most widespread intestinal infections all over the world. In the countries of Africa, Latin America, Asia it is one of the principal reasons of children’s under 5 mortality. The tendency towards morbidity growth has been lately marked not only in these countries but also in the countries of East Europe, states of CIS, including Ukraine.

Fight against Shigella is difficult due to polymorphism of clinical manifestations, variety of agent transmission factors, high receptivity at short duration of innate and specific immunity after illness as well as owing to high adaptation ability of shigellea and their resistance to antimicrobial agents. Shigellosis is found everywhere as concrete sporadic cases and epidemic outbreaks, thus it requires knowledge of this infection.

The problem of amoebiasis is of special significance in connection with contacts expansion with different countries, increase of tourist and business trips to the regions with hot climate. The real conditions are thus created for contamination in connection with the high morbidity level of native population. According to WHO findings (1988), amoebiasis is one of the major medical and social problems in the western and south-east regions of Africa, South-east Asia, China, Latin America, where the number of persons with dysenteric amebic invasion is 20-70%.

In the areas of temperate climate amoebiasis is registered as sporadic cases, however absence of doctors’ alertness to this disease and knowledge of this pathology leads to unreliability of statistical data about amoebiasis morbidity in these regions; especially as the number of dysenteric amoeba carriers in the case of careful inspection is 5-15% of inspected cases here. In our country the sporadic cases of amoeba dysentery are found mainly on a south. Amongst the countries of the CIS the most unfavorable are the states of Middle Asia and Transcaucasia, where carriers are found among 15-35% of habitants.
Acute enteric infections are one of the topical issues of health care in all countries. According to WHO, during the latest decades, incidence rate of diseases caused by Yersinia Enterobacteriactae (pseudotuberculosis and enteric yersiniosis) has raised. They are observed everywhere, both in developed and in developing countries. In the structure of enteric infections registered in developed countries yersinioses are in 3-4 place after shigellosis and salmonellosis. A number of causes stimulates propagation of these diseases: homioothermal animals are sensitive to Yersinia Enterobacteriactae including domestic animals, birds and humans; urbanization, development of large cattle farms, extension of public catering enterprise network, expansion in the number of synanthropic rodents; rather high resistivity of pathogens to environmental factors, preservation and accumulation of (Yersinia Enterobacteriactae) at low temperatures, high occurrence of dysbacteriosis in animals and humans due to uncontrolled administration of antibiotics causes penetration and preservation of pathogenic and opportunistic flora in intestines; absence of specific safety arrangements, fecal-oral route, high sensitivity of humans to this disease.

Clinical urgency of yersinioses is associated with the absence of alarm to this pathology, probability of development of severe generalized forms, complexity of diagnostics due to polymorphism of clinical presentations and specific conditions of isolation of the etiologic agent, low level of diagnostics of sporadic cases and mild forms of the disease, risk of development of surgical implications such as enterorrhagia, enterobrosia, peritonitis, appendicitis, and complications of allergic and autoimmune genesis.

2. Study purpose of practical studies:

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

To have general knowledge about shigellosis, amoebiasis place in the structure of infectious diseases, history of study, scientific contribution of native and foreign scientists to the history of scientific researches in this field. About the place of in the structure of infectious diseases, disease incidence in different
regions of Ukraine and in the world, about the mechanism and factors of introduction of infection, main clinical representations, to get current information about incidence of complications and bacteria carrying.

To study statistical data concerning shigellosis, amoebiasis prevalence, complications frequency, bacteria carrying in Ukraine and in the world nowadays. The history of scientific investigations of diseases caused by Yersinia; to have general knowledge about scientific contribution in this field made by native scientists; to get acquainted with current methods for diagnostication of these diseases.

### 2.2. Student have to know:  
\(\alpha -2\)
- shigellosis, amoebiasis etiology, factors of agent pathogenicity;
- epidemiology of shigellosis, amoebiasis, peculiarities of modern epidemiological process;
- pathogenesis;
- classification of shigellosis, amoebiasis clinical forms;
- peculiarities of shigellosis clinical course, depending on clinical form and agent;
- clinical manifestations of amebic dysentery and extraintestinal amoebiasis;
- shigellosis complications and their onset terms;
- amebic dysentery and extraintestinal amoebiasis complications;
- shigellosis, amoebiasis laboratory diagnostic;
- principles of treatment;
- therapeutic approach in case of emergency states;
- principles of prophylaxis;
- rules of discharging from the hospital;
- rules of the dispensary system.
- main pathogenic factors of Yersinia Enterobacteriactae.
• epidemiology of pseudotuberculosis and enteric yersiniosis: transmission mechanism, routes and main transmission factors, sources of infection.
• main stages of pathogenesis of pseudotuberculosis and enteric yersiniosis.
• clinical classification of pseudotuberculosis and enteric yersiniosis.
• main clinical representations of typical forms of pseudotuberculosis and enteric yersiniosis.
• pathogenesis of main clinical symptoms.
• intestinal and extraintestinal implications of pseudotuberculosis and enteric yersiniosis.
• pathogenesis, origin time and clinical representations and clinical implications of pseudotuberculosis and enteric yersiniosis.
• clinical approach to treatment of pseudotuberculosis and enteric yersiniosis.
• disease management of patients with surgical implications.
• preventive measures for pseudotuberculosis and enteric yersiniosis
• rules for discharging and health assessments of convalescent patients

2.3. Student have to be able: \( a -3 \)
• to keep the basicsanitary antiepidemic rules working with shigellosis, amoebiasis patient;
• to take the medical history with the estimation of epidemiological data (consumption of food products without thermal handling, contact with shigellosis patient or bacteria carrier, stay in endemic to amoebiasis regions;
• to examine patient and findout basic symptoms and syndromes of shigellosis, amoebiasis, to make the substantiation of presumptive diagnosis;
• to recognize the presence of specific complications;
• to carry out differential diagnostics of shigellosis, amoebiasis with diseases which have similar clinical manifestations;
• to draw up medical documents as far as the formulation of presumptive diagnosis “shigellosis”, “amoebiasis” is concerned (an urgent to the sanitary epidemiological station (SES);
• to work out a plan of laboratory and additional examination of patient;
• to interpret the results of laboratory examination, including specific methods of diagnostics;
• to work out an individual plan of treatment taking into account epidemiological data, clinical form of illness, severity of clinical process, presence of complications, allergy in anamnesis, concomitant pathology;
• to render the first aid in the case of ITSh, hypovolemic shock;
• to work out a plan of antiepidemic and preventive measures in the nidus of infection;
• to give recommendations concerning the regimen, diet, examination, supervision to convalescents.
• Adhere to the rules of the work at the bedside.
• To file information on medical history and to evaluate epidemiologic data.
• To examine a patient and determine main symptoms and syndromes of pseudotuberculosis and enteric yersiniosis, to substantiate clinical diagnosis for early hospitalization of the patient.
• To define the variant of pseudotuberculosis and enteric yersiniosis course, in particular, the generalized forms, on the basis of clinical symptoms.
• To define in time probable implications of pseudotuberculosis and enteric yersiniosis and emergency conditions on the basis of clinical examination.
• To prepare clinical documentation upon the provisional diagnosis of pseudotuberculosis and enteric yersiniosis (emergency report to regional epidemiologic department).
• To prepare a plan for laboratory and additional examination of the patient with pseudotuberculosis, enteric yersiniosis.
• To give an interpretation for laboratory results.
• To give correct evaluation for the results of specific diagnostic investigation depending on biological material and duration of examination.
• To work out an individual treatment plan depending on the clinical form, clinical stage, existence of implications, severity, allergic anamnesis, co-morbidity.
• To work out preventive measures in the center of infection.
• To provide recommendations on regimen, diet, examination and supervision during convalescence.

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 shigellosis, amoebiasis, pseudotuberculosis and enteric yersiniosis.
• 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) Basic knowledge, skills which are necessary for studying of topic (interdisciplinary integration)

<table>
<thead>
<tr>
<th>Subject</th>
<th>To know</th>
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<tbody>
<tr>
<td>Previous subjects</td>
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<tr>
<td>Normal anatomy</td>
<td>Anatomical structure of human gastro-intestinal tract</td>
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<tr>
<td>Microbiology</td>
<td>Taxonomic properties of Shigellae, their antigenic structure, growth,</td>
<td>To carry out necessary extent of laboratory examinations (inoculation</td>
</tr>
<tr>
<td>Multiplication, cultivation, agent’s identification. Properties of dysenteric ameba, forms of its existence in human organism, methods of amoebiasis specific diagnostics</td>
<td>To interpret the results of diagnostics specific methods.</td>
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<tr>
<td>Physiology</td>
<td>Parameters of human organs and systems physiological norm; standard laboratory examination indexes (total blood count, clinical urine analysis, biochemical blood analysis, parameters of AOS, electrolytes etc).</td>
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<tr>
<td>To estimate the laboratory examinations data.</td>
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<tr>
<td>Pathological Physiology</td>
<td>Mechanism of organs and systems dysfunctions with pathological conditions of different genesis.</td>
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<tr>
<td>To interpret pathological changes according to the results of laboratory examination on organs and systems dysfunction of different genesis.</td>
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<tr>
<td>Immunology and Allergology</td>
<td>Basic terms of subject, role of immunity system in the infectious process, influence on agent elimination term from human organism.</td>
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<tr>
<td>To estimate immunological researches findings.</td>
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<tr>
<td>Surgery</td>
<td>Clinical-laboratory signs of intestinal hemorrhage, peritonitis, first aid approach.</td>
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<tr>
<td>To diagnose these complications timely, to administer proper examination and render the first aid.</td>
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<tr>
<td>Propaedeutics of General Medicine</td>
<td>Methods and basic stages of patient’s clinical examination.</td>
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<tr>
<td>To take the history, to examine the patient, find out pathological symptoms and syndromes. To analyse findings.</td>
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<tr>
<td>Clinical Pharmacology.</td>
<td>Pharmacokinetics and pharmacodynamics, side effects.</td>
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<td>To prescribe treatment depending on patient’s age</td>
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<tr>
<td>Effect of preparations for shigellosis, amoebiasis, drugs of pathogenetic therapy. and individual peculiarities, to choose the optimum mode of drugs administrations and dosage, give prescriptions.</td>
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<tr>
<td><strong>Following subjects</strong></td>
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<tr>
<td><strong>Family medicine</strong></td>
<td>Etiology, pathogenesis, epidemiology, clinical manifestations, possible complications of shigellosis, amoebiasis. Prevention and treatment principles. Therapeutic approach, indications for hospitalisation.</td>
<td>To carry out differential diagnostics of shigellosis, amoebiasis with diseases of similar clinical symptomatology. To recognize disease, to interpret laboratory examinations findings. To work out a plan of antiepidemic measures in the nidus of infection. To determine the necessity of patient’s hospitalization. To give recommendation of follow-up supervision and treatment. To fill in the urgent report to SES. To render emergency in case of need.</td>
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<tr>
<td><strong>Intra-subject integration</strong></td>
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</tbody>
</table>
| **Infectious diseases.** | Peculiarities of infectious diseases. Principles of diagnostics, treatment, infectious diseases prevention. Pathogenesis, epidemiology, clinical manifestations dynamics, laboratory diagnostics, shigellosis and amoebiasis treatment and prevention, their complications. Peculiarities of clinical course depending of clinical form. Principles of treatment and prevention. | To carry out differential diagnostics of shigellosis, amoebiasis with other infectious diseases. To diagnose disease, its complications; to interpret laboratory examinations data. To prescribe etiotropic, patogenetical, symptomatic treatment. To work out recommendations to the patient concerning diet, regimen, professional action, rest and other elements of individual
## Pseudotuberculosis, enteric yersiniosis

<table>
<thead>
<tr>
<th>Discipline</th>
<th>To know</th>
<th>To be able to carry out</th>
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<tr>
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<tr>
<td>Microbiology</td>
<td>Properties of Yersinia Enterobacteriactae; rules and time for collecting biologic material for specific diagnostics, to learn principles of bacteriologic and serologic investigations.</td>
<td>To collect biologic material for bacteriologic investigation, to evaluate the results of serologic tests (agglutination test, reaction of indirect hemagglutination, complement fixation test, immunofluorescence test);</td>
</tr>
<tr>
<td>Physiology</td>
<td>Parameters of physiological standard of human organs and systems, results of laboratory investigation in normal condition (clinical blood test, urinalysis, biochemical blood assay, acid-base properties, properties of electrolytes etc).</td>
<td>To evaluate laboratory results.</td>
</tr>
<tr>
<td>Pathophysiology</td>
<td>Mechanism of dysfunction of organs and systems in pathologic conditions of different genesis (nature and mechanism of biliousness progression, diarrheic syndrome etc).</td>
<td>To give an interpretation to pathologic changes on the base of laboratory investigation in the case of dysfunction of organs and systems of different genesis.</td>
</tr>
<tr>
<td>Propedeutics of internal diseases</td>
<td>Main stages and methods for clinical examination of the patient.</td>
<td>To collect an anamnesis, to carry out clinical examination of the patient’s organs and systems, to reveal clinical signs of pathology. To analyze the obtained data.</td>
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<tr>
<td>Subject</td>
<td>Description</td>
<td>Objectives</td>
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<tr>
<td><strong>Epidemiology</strong></td>
<td>Main stages of epidemiological process (source, mechanism of introduction of infection, transmission routes) in the cases of yersiniosis, incidence rate of the pathology in Ukraine and in the world.</td>
<td>To collect epidemiological anamnesis, to take antiepidemic and preventive measures in the center of infection.</td>
</tr>
<tr>
<td><strong>Immunology and allergology</strong></td>
<td>Basic concept on the subject; role of immune system in infectious process, influence on the duration of isolation infectious organisms from the human body. Immunologic aspects of recurrence development, inveterate bacteria carrying.</td>
<td>To evaluate the results of immunological investigations.</td>
</tr>
<tr>
<td><strong>Dermatology</strong></td>
<td>Pathogenesis, clinical characteristics of exanthemas.</td>
<td>To identify eruption in the patient with pseudotuberculosis, enteric yersiniosis.</td>
</tr>
<tr>
<td><strong>Surgery</strong></td>
<td>Clinical and laboratory signs of perforation of small intestine ulcer, enterorrhagia, appendicitis. Principles of emergency aid.</td>
<td>To diagnose these implications in time, to prescribe proper examination, to deliver immediate medical care.</td>
</tr>
<tr>
<td><strong>Clinical pharmacology.</strong></td>
<td>Pharmacokinetics and pharmacodynamics, unwanted side effects, side effects of chloramphenicol, ciprofloxacin, aminoglycosides, tetracyclines, means of pathogenetic therapy.</td>
<td>To prescribe treatment depending on the age, personal traits of the patient, clinical form, severity of disease; to select the optimum administration and dosage regimen of preparations; to make out prescriptions.</td>
</tr>
<tr>
<td><strong>Reanimation and</strong></td>
<td>Emergency conditions:</td>
<td>To diagnose in time emergency conditions and deliver</td>
</tr>
</tbody>
</table>
**Intensive care**
- Toxic shock syndrome
- Enterorrhagia
- Enterobrosia
- Infection-exhaustion psychosis
- Peritonitis

**紧急援助**
- Toxic shock syndrome
- Enterorrhagia
- Enterobrosia
- Infection-exhaustion psychosis
- Peritonitis

**Following disciplines**

| Family practice | Pathogenesis, epidemiology, dynamics of clinical representations, probable implications of pseudotuberculosis, yersiniosis. Peculiarities of clinical course. Principles of treatment and prophylaxis. | To carry out differential diagnostics of diseases of different genesis with pseudotuberculosis, enteric yersiniosis. To identify pseudotuberculosis, enteric yersiniosis; their implications; to interpret the laboratory data. To hospitalize the patient to infectious disease ward in due time. To fill in the emergency report. To deliver emergency aid if necessary. |

**Intersubject integration**

| Infectious diseases | Characteristics of infectious diseases. Principles of diagnostics, treatment, preventive measures of infectious diseases. Pathogenesis, etiology, dynamics of clinical representations, laboratory diagnostics, probable implications of pseudotuberculosis, enteric yersiniosis. Principles of treatment and prophylaxis. | To carry out differential diagnostics of pseudotuberculosis, enteric yersiniosis with other infectious diseases. To identify pseudotuberculosis, enteric yersiniosis; their implications; to interpret the laboratory data. To prescribe treatment. To deliver emergency aid at the pre-admission stage. |
3.2 Theme contents.

**Etiology**
- Sh.dysenteriae
- Sh.flexneri
- Sh.boydii
- Sh.sonnei

**Epidemiology**
The source of infection is a carrier or shigell patient. The transmission mechanism is fecal-oral: by food, contact, water-borne. Susceptibility is high, postimmunity is nonurable, species-specific and types-specific.

**Pathogenes**
- Partial Sh death
- Sh penetration into colon

**Classification**
- According to duration:
  - acute (up to 2 months)
  - protracted (over 2 months)
- Colitic form
- Gastroenterocolitic form
- Enterocolitic form

**Clinical signs**
- Colicky cramp-like abdominal pain,
- Imperative feelings of defecation,
- Tenesmus, stool with mucus and blood

**Complications**
- Distal colitis syndrome
- Hypovolemic shock
- Rectal prolapse
- Intussusception
- Hemorrhages

**Diagnostics**
- Total blood count (ESR↑, leukocytosis)
- Coprocytogram (mucus, unchanged erythrocytes, leukocytes – 30-40 within eyesight)

**Differential**
- Biochemical
- Specific
- Expression methods
- Serologic methods
- (radio-immuno) say (RIA) IHAT,

**Treatment**
- Antibacterial drugs [nitrofurantoin, fl

**Prevention**
- Non-specific

**Pathogenic**
- Disintoxication therapy
- Enterosorbsents, enzymatic drugs, probiotics
Amoebiasis

**Etiology**

- Entamoeba hystolytica
  - Great vegetative form
  - Cysts

**Epidemiology**

- The source of infection is a carrier; more rare it is amoebiasis patient. The transmission mechanism is fecal-oral. The main way of amoeba transmission is by food, contact, water-borne. Susceptibility is not high. Immunity is unstable.

**Pathogenesis**

- Penetration into stomach
- Cysts penetration into small intestine
- Intestinal amoebiasis
  - Faeces look like raspberry jelly
  - Fever
  - Anemia
  - Asthenia
  - Liver, lungs, brain abscesses
  - Burst
  - Secondary infection of dysbacteriosis

**Classification**

- According to duration:
  - acute (up to 3 months);
  - protracted (up to 6 months);
  - chronic (over 6 months).
- According to the character of course:
  - mild;
  - moderate;
  - severe;
- According to severity:
  - mild;
  - moderate;
  - severe.

**Clinical forms**

- Extra-intestinal amoebiasis
  - Liver, lungs, brain abscesses

**Diagnosis**

- Total clinical
- Parasitoscopy
- Serologic tests
- Additional

- Total blood count
- Coprocystogram analysis (vitriform mucus, erythrocytes, eosinophilis), asthma (Charcot-Leyden) crystals
- Vegetative forms and cysts revealing and identification
- Indirect haemagglutination test (IHAT), ELISA, immunofluorescent antibody test
- Rectoromanoscopy irrigography, US, KT, radiography, abscess punction (if possible)

**Treatment**

- Etiotropic therapy
- Universal drugs (metronidazole (trichopol))
- Surgical treatment
- Antihistaminic drugs, ferments, probiotics, probiotics,

**Prevention**

- Isolation of contagious patients and examinations of contacted persons
- Disinfection
- Measures in the nidus of infection
- Prevention Isolation of contagious patients and examinations of contacted persons
- Disinfection

**Complications**

- Intestinal amoebiasis
  - Pericolitis
  - Intestinal obstruction
  - Chronic intestinal ulcerative colitis
  - Vesicointestinal fistula
  - Dybacteriosis

**Clinical signs**

- Right side colitis
- Faeces look like raspberry jelly
- Anemia
- Asthenia
- Liver, lungs, brain abscesses
- Burst
- Secondary infection of dysbacteriosis

**Etiology**

- Cysts
- Great vegetative form (lumen)
- Intestinal bleeding
- Ameboma
- Pericolitis

**Pathogenesis**

- Penetration into stomach
- Cysts penetration into small intestine
- Intestinal amoebiasis

**Classification**

- According to duration:
  - acute (up to 3 months);
  - protracted (up to 6 months);
  - chronic (over 6 months).
- According to the character of course:
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- Universal drugs (metronidazole (trichopol))
- Surgical treatment
- Antihistaminic drugs, ferments, probiotics, probiotics,

**Prevention**

- Isolation of contagious patients and examinations of contacted persons
- Disinfection
- Measures in the nidus of infection
3.3 Literature recommended:

Main sources:

1. Lectures of Professor.

Additional sources:

3.4. Self-control materials

3.4.1. Questions to be answered

1. How can you characterize the modern state of shigellosis morbidity in Ukraine and in the world?
2. What main serological groups of shigellae do you know? Describe their morphological features.
4. Describe the source of infection; name the mechanism and main ways of getting infection.
5. What are the stages of shigellosis pathologic process?
6. What is the clinical classification of shigellosis?
7. What are the peculiarities of shigellosis clinical course depending on clinical form and agent?
8. What are the possible complications of shigellosis?
10. Methods of shigellosis specific diagnostics. Interpretation of examination findings.
11. What are the main diagnostic signs of shigellosis?

13. What are the principles of shigellosis patients’ therapy?

14. What are the terms and rules of convalescent discharging from the hospital?

15. Shigellosis prevention.

16. What are the terms of dispensary supervision?

17. How can you characterize the modern state of amoebiasis morbidity in Ukraine and in the world?

18. What forms of Entamoeba histolytica existence in human organism do you know? Describe them.

19. Specify the agent’s stability to the action of environmental factors.

20. Describe the source of infection; name the mechanism and the main ways of getting infection.

21. What is the clinical classification of amoebiasis?

22. What are the stages of amoebiasis pathologic process?

23. What are the peculiarities of amebic dysentery and extra-intestinal amoebiasis clinical course?

24. What are the possible complications of amoebiasis?

25. Plan of amoebiasis patient’s examination.

26. What are the methods of amoebiasis specific diagnostics?

27. What are the main diagnostic signs of amoebiasis?

29. What are the principles of amoebiasis patients’ therapy?

30. Give the characteristics of etiotropic drugs.

31. What are the rules of convalescent discharging from the hospital?

32. Prevention of amoebiasis (specific, non-specific).

33. Terms of dispensary supervision.

34. Source and reservoir of infection in the case of pseudotuberculosis, enteric yersiniosis?

35. Transmission mechanism and routes of pseudotuberculosis, enteric yersiniosis.


37. Stages of pathogenesis of pseudotuberculosis, enteric yersiniosis.

38. In which part of the intestine pathologic process in the patient is localized?


40. Reference clinical signs of pseudotuberculosis at the height of disease.

41. Characteristics of dyspeptic syndrome in the case of enteric yersiniosis.

42. Characteristics, origin time and eruption dynamics in the patient with pseudotuberculosis.

43. Pathogenesis and description of Padalka symptom.

44. To describe the Gloves and Socks syndrome.
45. Characteristics of the course of generalized forms of pseudotuberculosis, enteric yersiniosis.

46. Implications typical for pseudotuberculosis, enteric yersiniosis.

47. Extraintestinal implications of pseudotuberculosis, periods of their origin.

48. Haemogramma of the patient with pseudotuberculosis, enteric yersiniosis at the height of disease.

49. Plan of the patient examination with provisional diagnosis of pseudotuberculosis, enteric yersiniosis.

50. Methods for specific diagnostics of yersinioses.


52. Pathogenetic therapy of pseudotuberculosis, enteric yersiniosis.

53. Rules for discharging the patient with yersiniosis from the hospital.

3.4.2. Self-control tests

Variant 1

1. What is the main transmission way of Sonne shigellosis?
A. By contact
B. Air-borne
C. Water-borne
D. Sexual
E. By food

2. Shigellosis of gastroenterocolitis form is mostly caused by Shigellae:
A. Sonnei’s sh.
B. Grigoriev-Shigy’s sh.
C. Flexner’s sh.
D. Large-Sack’s sh.
3. To confirm diagnosis of acute shigellosis it is enough to reveal:
   A. Tenesmus
   B. Frequent liquid stools with mucus and blood
   C. Shigellae in stool
   D. Proctosigmoiditis (at rectoromanoscopy)
   E. Spastic sigmoid colon

4. Coprocytogram at moderate shigellosis reveals:
   A. Liquid stool is enveloped by pieces of mucus, there are undigested muscular fibers, starched corns, increased amount of neutral fat, 30-40 leucocytes within eyesight, moderate amount of erythrocytes
   B. Semi-liquid feces is mixed with mucus, there are undigested muscular fibers, starched corns, increased amount of neutral fat, 5-7 leucocytes within eyesight
   C. Semi-formed stool, mucus is found here and there, there are 10-15 leucocytes within eyesight, cysts are of rounded form up to 10 m in diameter, contain 8 nuclei
   D. Formed stool, mucus is found here and there, there are 5-6 leucocytes and 1-2 erythrocytes within eyesight
   E. Liquid stool is enveloped by mucus, there are undigested muscular fibers, starched corns, increased amount of neutral fat, 10-20 leucocytes within eyesight, small amount of erythrocytes

5. Healing of ulcers at shigellosis is accompanied by:
   A. Rough scars formation with intestinal dysfunction
   B. Mucosa deformation
   C. Superficial scars formation without intestinal dysfunction
   D. Intestinal obstruction development
   E. Intestinal bleeding development

6. Rules of discharging from the hospital for shigellosis patient, which do not belong to the decreed group:
A. Not before 3 days after stool and temperature normalization, presence of 1 negative results of stool bacteriological analysis, carried out in 2 days after antibacterial therapy termination
B. Apparent clinical recovery
C. Presence of 2 negative results of stool bacteriological analysis
D. Presence of 3 negative results of stool bacteriological analysis, carried out in 2 days after antibacterial therapy termination
E. not true.

7. Moderate form of shigellosis is treated by prescribing:
   A. Ferments
   B. Enterosorbents
   C. Nitrofurans, 8-oxyquinoline derivative
   D. “Regidron”, “Gastrolit”, “Oralit” orally
   E. All true

8. Incubation period of shigellosis mostly comprises:
   A. 2-3 days
   B. 6-7 days
   C. 10-12 days
   D. 12-14 days
   E. 16-20 days

9. Choose right answer concerning Shigellae proprieties:
   A. All true
   B. Gram-negative immobile bacilli
   C. Shigellae have O antigens
   D. Shigellae have K antigens
   E. Spores, capsules are not formed

10. Antigen structure of Sigella is:
    A. O-antigen
    B. F-antigen
    C. K-antigen
D. H-antigen
E. No antigens

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**Variant 2**

1. What is the main transmission way of Flexner’s shigellosis?
   
   A. By contact
   
   B. Air-borne
   
   C. Water-borne
   
   D. Sexual
   
   E. By food

2. Which type of Shigellae cause the most severe shigellosis form:
   
   A. Sonnei’s sh.
   
   B. Grigoriev-Shigy’s sh.
   
   C. Flexner’s sh.
   
   D. Large-Sack’s sh.
   
   E. Shtutcer-Shmitc’s sh.

3. Rules of discharging from the hospital for shigellosis patient, which do not belong to the decreed group:
   
   A. Not true
   
   B. Apparent clinical recovery
   
   C. Presence of 3 negative results of stool bacteriological analysis, carried out after antibacterial therapy termination, not before 3 days after stool and temperature normalization
   
   D. Presence of 1 negative result of stool bacteriological analysis, carried out in 2 days after antibacterial therapy termination
   
   E. Not before week after stool and temperature normalization
3. What clinical variant of shigellosis corresponds to such symptoms: colicky cramp-like pain in lower abdomen, tenesmus, frequent scanty stool with mucus and blood streaks
   A. Colitis
   B. Gastroenterocolitis
   C. Gastroenteric
   D. Enteric
   E. Enterocolitis

4. Measures for contact persons in the nidus of shigellosis infection:
   A. Supervision for 10 days
   B. Single bacteriological examination of the decreed contingents with their discharge from work on the term of supervision
   C. Supervision for 5 days
   D. Single bacteriological examination of the decreed contingents without their discharge from work
   E. Supervision isn’t conducted

6. What antibacterial drugs are used for shigellosis treatment, exept?
   A. Nifuroxazid
   B. Doxycyclin
   C. Ampicillin
   D. Intetrix
   E. Furazolidone

7. Serological methods of shigellosis diagnostics all, exept:
   A. Indirect hemagglutination reaction
   B. Direct hemagglutination reaction
   C. Rectoromanoscopy
   D. Passive hemagglutination reaction
   E. Hemagglutination reaction

8. Pain syndrome at shigellosis is accompanied by:
   A. Nervous - muscular intestinal apparatus lesions
B. Central nervous system damage
C. Intestinal atony
D. Intestinal destruction process
E. not true

9. Mucosa of distal colon at acute shigellosis of moderate severity is completely restored morphologically and functionally:
A. 2-3 months
B. A week
C. 6 months and more
D. A month
E. 2-3 weeks

10. Which of shigellosis agents produce exotoxin:
A. Sonnei’s sh.
B. Flexner’s sh.
C. Grigoriev-Shigy’s sh.
D. Large-Sack’s sh.
E. All shigellosis agents

**Keys:**

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**Variant 3**

1. Hemogram of chronic intestinal amoebiasis patients is characterized by:
A. Anemia, eosinophilia, monocytes, lymphocytosis, increase of ESR
B. Normal hemogram
C. Heterophilic leukocytosis, increase of ESR
D. Leukopenia, eosinophilia, anemia
E. Lymphocytosis, monocytosis

2. Coprocystogram at intestinal amoebiasis reveals:
A. Considerable amount of vitriform mucus, erythrocytes, eosinophils, Charcoal-Leyden crystals
B. Grouped erythrocytes are located as columns, there are leucocytes within eyesight
C. Erythrocytes are within all eyesight, small amount of mucus
D. Increased amount of neutral fat, undigested muscular fibres, starched corns.
E. Within the limits of norm

3. Disease onset at amoebiasis is:
   A. Gradual
   B. Acute
   C. Subacute
   D. Acute with abrupt clinical manifestations
   E. Latent

4. Endoscopy of colon at chronic amoebiasis reveals the followings changes:
   A. There are ulcers 10-20 mm in diameter on the unchanged mucosa; these ulcers have edematous, deepened edges, surrounded by hyperemic area and are located more often on folds, the bottom is covered with pus and necrotic mass
   B. Different diameter ulcers, cysts, polyps, amebomas
   C. Diffuse edema, hyperemia of mucosa, intestine spasm, hemorrhages, there are fibrinous stratifications on the ulcers’ surface
   D. Vascular picture impoverishment, single ulcers, «velvety» mucosa, contact and spontaneous hemorrhages
   E. No changes

5. Name the pathogenic human amoebas:
   A. Entamoeba hartmanni
   B. Entamoeba histolytica
   C. Entamoeba coli
   D. Endolimax nana
   E. Jodamoeba butschlii

6. Endoscopy of colon at acute amoebiasis reveals the followings changes:
A. There are ulcers 10-20 mm in diameter on the unchanged mucosa; these ulcers have edematous, deepened edges, surrounded by hyperemic area and are located more often on folds, the bottom is covered with pus and necrotic mass
B. Different diameter ulcers, cysts, polypuses, amebomas
C. Diffuse edema, hyperemia of mucosa, intestine spasm, hemorrhages, there are fibrinous stratifications on the ulcers’ surface
D. Vascular picture impoverishment, single ulcers, «velvety» mucosa, contact and spontaneous hemorrhages
E. No changes
7. Abdominal pain at the onset of uncomplicated amoebiosis localized:
A. Along colon
B. Within entire abdomen
C. In the left iliac region
D. In the right iliac region
E. Both left and right iliac regions
8. What form of amoeba can produce disease after entering organism:
A. Small vegetative form
B. Large vegetative form
C. Cysts containing 2-3 nuclei
D. Cysts containing 4 nuclei
E. Small vegetative form, cysts
9. The source of infection in pseudotuberculosis:
A. man
B. clamps
C. bird
D. rodents
E) mosquitoes.
10. Pseudotuberculosis rash at the hands and feet:
A. urticaria
B. vesicle
C. eritema
D. bulla
E. Roseolous

Keys:

<p>| | | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>3</td>
<td>A</td>
<td>5</td>
<td>B</td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>4</td>
<td>B</td>
<td>6</td>
<td>A</td>
</tr>
<tr>
<td>7</td>
<td>D</td>
<td>9</td>
<td>D</td>
<td>8</td>
<td>D</td>
</tr>
<tr>
<td>10</td>
<td>C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3.4.3. Situational tasks of the second level learning

**Task 1**

Patient K., 27 years, a cook, was admitted to isolation hospital on the second day of illness with complaints of headache, frequent stool (up to 15 times per day), vomiting, nausea, pain in lower abdomen, tenesmus, false urges to defecation. The onset of the disease was acute with chills, increase of body temperature to 39 ºC, repeated vomiting. Acute colicky cramp-like abdominal pain intensified before defecation has appeared in 5-7 hours as well as liquid stool with admixtures of mucus and blood streaks. During hospitalization the patient’s state is heavy, body temperature is 39 ºC, malaise. The patient gets into contact unwillingly. Pulse is 104 beets per minute. Heart sounds are dull. Tongue is dry, greatly coated with grey film. The abdomen is tympanic (swollen), painful in colon region. Sigmoid colon is spastic, painful.

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

**Task 2**

Student A., 22 years old, went to Ethiopia and after her coming back liquid stool and weight losing appeared. Then stool frequency increased to 7 times per
day accompanied by spastic abdominal pain that became intense during defecation. Stool looks like “raspberry jelly”.

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

Task 3

The child of 12 was admitted to the hospital with complaints of repeated vomiting, acute spastic abdominal pain. Liquid stool with small admixtures of mucus were twice. Examination revealed body temperature of 38.4°C, dry tongue, tachycardia, iliac pain at palpation, weakly positive symptoms of peritoneal irritation. There are cases of shigellosis at her class.

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

Task 4

Patient P., 28 years old, a musician, was admitted to the hospital with complaints of spastic pain localized at the lower abdomen, frequent liquid stool with the admixtures of mucus, chills, weakness. He considers himself to be ill for a year when he had frequent liquid stool with the admixtures of mucus and blood for 3 days. He was treated at the isolation hospital from Sonne dysentery. After his discharging from the hospital he didn’t follow a special diet, used alcohols. He had periodical pains at the lower abdomen, diarrhea. He treated himself with tetracycline and phtalasol unsuccessfully. During his illness the patient lost 7 kg, became anxious, didn’t sleep well. His skin is pale, the tongue is coated with white film. Palpation reveals pain of sigmoid intestine.

1. Provisional diagnosis.
2. Examination plan.
3. Treatment plan.
Task 5

Captain of one Indian ship turned for the help to the navigation hospital because one person from his crew had acute abdominal pain. Patient’s examination revealed rapid weight losing, features became sharp, the tongue is dry, puls is weak, rapid. The abdomen is tense, painful at palpation, the liver is enlarged, Orthner’s and Shchetkin’s symptoms are positive. It is known from patient’s epidemiological anamnesis that he had periodic liquid stool with admixtures of mucus.

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

4. Materials for the class of independent work
   4.1. List of study practical tasks to be performed in the practice:
   1. To study the method of shigellosis, amoebiasis patient clinical examination.
   2. To carry out shigellosis, amoebiasis patient’s examination.
   3. To carry out differential diagnostics of shigellosis, amoebiasis.
   4. To work out a plan of shigellosis, amoebiasis patient laboratory examination.
   5. To interpret the results of shigellosis, amoebiasis patient specific examinations.
   6. To recognize complications of shigellosis, amoebiasis.
   7. To work out a plan of shigellosis, amoebiasis patient treatment.
   8. To define medical tactic in the case of emergency states.
   9. To draw up medical documents as diagnosis “Shigellosis”, “Amoebiasis” is concerned.
   10. To master the method for examination of patients with pseudotuberculosis, enteric yersiniosis.
   11. To carry out curation of the patient with pseudotuberculosis, enteric yersiniosis.
   12. To carry out differential diagnostics of pseudotuberculosis, enteric yersiniosis.
13. To prepare a plan for laboratory investigation of the patient with pseudotuberculosis, enteric yersiniosis.
14. To interpret the results of specific examination of the patient.
15. To define implications of pseudotuberculosis, enteric yersiniosis.
16. To prepare a plan for treating the patient with pseudotuberculosis, enteric yersiniosis.
17. To define medical tactics in the case of arising of emergencies.
18. To prepare clinical documentation upon the diagnosed “Yersiniosis”.

4.2 Professional algorithm for diagnostics skills and ability

**formation of shigellosis**

<table>
<thead>
<tr>
<th>No.</th>
<th>Description</th>
<th>To single out complaints which characterize syndromes of:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>To study the method of shigellosis patient clinical examination</td>
<td>- general intoxication;</td>
</tr>
<tr>
<td>2.</td>
<td>To carry out the patient’s examination</td>
<td>- gastro-intestinal lesions;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- other organs lesions.</td>
</tr>
<tr>
<td></td>
<td>I. To know patient’s complaints.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>II To take:</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1. The case history</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2. The life history</td>
<td></td>
</tr>
<tr>
<td></td>
<td>3. The epidemiological history</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>To pay attention to acute onset; terms, sequence of occurrence, dynamics of:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- fever;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- abdominal pain, its localization;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- tenesmus, imperative feeling of defecation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- stool character;</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- other symptoms.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>To find out previous diseases.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>To find out the data of fecal-oral way realization of transmission mechanism, pay attention to patient’s stay in high risk of infection regions, personal contact with shigellosis patient or bacteria carrier, consumption of food products without thermal handling.</td>
</tr>
</tbody>
</table>
### III. To carry out the physical examination of the patient.

1. General examination:
   - general condition of patient;
   - skin; mucosa;

2. Digestive system:
   - tongue inspection;
   - abdominal palpation;
   - stool characteristic;

3. Cardiovascular system:
   - pulse;
   - blood pressure;
   - heart auscultation

4. Respiratory system.

5. Urogenital system

---

To remember: the presence, intensity and dynamics of symptoms and caused by term and severity of disease clinical course and depend on concomitant pathology.

To pay attention to:
- weakness;
- the body temperature,
- pallor, dryness of skin, mucosa;
- skin turgor;

- coated tongue;
- marked spasm, thickening, painfullness of colon, especially sigmoid colon;
- at colitic form faeces gradually loss normal character and have mucus and blood streaks admixtures, sometimes in form of “rectal spitting”
- at gastroenterocolitic form – frequent, bulky, watery stool with admixtures of undigested food, later – with mucus and blood streaks

To pay attention to:
- presence of vomiting;
- moderate tachycardia;
- moderate low blood pressure;
- moderate heart sounds dullness.

Absence of changes at typical shigellosis process.
To prescribe laboratory and additional examination(s), interpret results:

<table>
<thead>
<tr>
<th>1. Total blood count.</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Clinical urine analysis.</td>
</tr>
<tr>
<td>3. Coprocystogram</td>
</tr>
<tr>
<td>5. Bacteriological examination of stool (emetic mass and stomach washing liquid examination is possible at gastroenterocolitic form).</td>
</tr>
</tbody>
</table>

To pay attention to:
- possible dysuria

To pay attention to typical changes: moderate heterophilic leukocytosis with the deviation of the formula to the left, ESR increase.

In the case of heavy severity – proteinuria, erythrina, leukocyturia.

Microscopy reveals mucus, accumulation of leucocytes, erythrocytes, epithelium cells.

Reduction of general protein and albumen level, increase of globulins level are possible in the period of height. Appropriate changes are possible in case of complications rise (hypovolemic shock, ITSh, hemolytic-uremic syndrome).

The result depends on a technique, multiplicity, terms of material taking and inoculation. The later must be done as soon as possible, definitely prior to antimicrobial treatment beginning. Stool is taken in the test-tube sterilized without chemical disinfectants and is quickly delivered to the laboratory. Ploskirev’s, Levine’s, Endo’s mediums are used for inoculation.

Results can be ready in 2-5 hours.

Shigella antigen in the native stool is revealed by anti-shigella agglutinin serums, adsorbed on activated carbon.

It is rather specific. Diagnostic titre at Flexneri shigellosis is 1:400, for other agents...
7. Serological methods
- Conglutination reaction;
- Indirect haemagglutination reaction (IHAR)

7. Allergologic diagnostic:
- Cerukalov’s test;

8. Additional methods:
- rectoromanoscopy

- 1:100.
The minimum diagnostic titre is 1:160.
Blood serum examination is carried out in dynamics on the 5-7th and 10-17th day from the disease onset.

Nowadays it isn’t practically used due to its low specificity
Signs of catarrhal, erosive and ulcerous proctosigmoiditis. It is indicated with differential diagnostic purpose.

---

### How to work with materials for classes self-training as far as amoebiasis diagnostics is concerned

<table>
<thead>
<tr>
<th>№</th>
<th>Task</th>
<th>Sequence of carrying-out</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>To study the method of shigellosis patient clinical examination</td>
<td>I. To know patient’s complaints.</td>
<td>To single out complaints which characterize syndromes of:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- hemocolitis with recurrent process tendency;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- general intoxication;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- other organs lesions.</td>
</tr>
<tr>
<td>2.</td>
<td>To carry out the patient’s examination</td>
<td>II To take: 1. The case history</td>
<td>To pay attention to gradual onset; terms, sequence of occurrence, dynamics of:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- asthenia;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- fever;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- abdominal pain, its localization;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- stool character;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- other symptoms.</td>
</tr>
</tbody>
</table>
2. The life history
3. The epidemiological history

III. To carry out patient’s physical examination.

1. General examination:
   - general condition of patient;
   - skin; faucial mucosa;

2. Digestive system:
   - tongue inspection;
   - abdominal palpation;
   - stool characteristic;

3. Cardiovascular system

4. Respiratory system.

To find out previous diseases.

To find out the data of faecal-oral way realization of transmission mechanism, pay attention to patient’s stay in tropic and subtropics regions, in areas with poor water-supply.

To remember: the presence, intensity and dynamics of symptoms and caused by term and severity of disease clinical course and depend on concomitant pathology.

To pay attention to:
   - weakness;
   - body temperature,
   - growing thin, asthenia,
   - pallor, possible jaundice ;
   - erosions and ulcers with black edges in perianal area, perineum and buttocks in case of skin form;

   To pay attention to:
   - coated tongue;
   - painfulness along colon, especially caecum and the ascending colon, their spasm;
   - hepatomegaly (mainly due to right lobe)
   - liquid stool with a lot of vitriform mucus with blood (“raspberry jelly”);
   - alternating change of diarrhea and constipation (in case of chronic intestinal amoebiasis);

Changes are nonspecific and correspond to the degree of intoxication syndrome intensity and presence of complications

Changes are present in case of extra-intestinal amoebiasis:

To pay attention to:
<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Total blood count.</td>
<td>- cough presence, hemoptysis; - great amount of chocolate color sputum (in case of lesion focus and bronchus combination); - presence of pneumonia signs, dry or exudative pleurisy</td>
</tr>
<tr>
<td>2. Clinical urine analysis.</td>
<td>Changes are present in case of extra-intestinal amoebiasis</td>
</tr>
<tr>
<td>3. Coprocytogram</td>
<td>Neurological symptomatology correspond to localization and size of brain abscess.</td>
</tr>
<tr>
<td>4. Biochemical methods of examination.</td>
<td>As a rule there are no changes in case of acute amoebiasis</td>
</tr>
<tr>
<td>5. Parasitoscopy (stool, pus, taken from ulcers’ surface)</td>
<td>In case of chronic amoebiasis: hypochromic anemia, eosynophilia, monocytopsis, limphocytosis, ESR increase. In case of liver abscess there is heterophilic leukocytosis with the deviation of the formula to the left. ESR is increased.</td>
</tr>
<tr>
<td>6. Parasyitoscopy (additional method)</td>
<td>There are no changes in case of typical process.</td>
</tr>
<tr>
<td>7. Serological methods - Inderect haemagglutination reaction (IHAR)</td>
<td>There is great amount of mucus, erytrocytes, eosinophils, Charcot-Leyden crystals.</td>
</tr>
</tbody>
</table>

Native smears and smears processed by Lyugol solution or Heidenhain’s iron hematoxylin are examined:

- great vegetative form determinaton is of particular importance in case of acute intestinal amoabiasis or exacerbation of chronic one;
- small lumen forms of amoebae and cysts are found in convalescents with chronic amoebiasis in remission as well as in carriers;
- tissue amoebae are revealed in pus from pathological foci as well as in scrapes from skin ulcers in case of inner organs amoebiasis.

NB! It is necessary to take liquid stool
8. Additional methods:
- rectoromanoscopy
- irigography
- US
- pneumonography
- CT (computer-tomography) scan
- abscess punction

containing mucus, and deliver it to the laboratory immediately. Before microscopy it’s necessary to warm up the stage of microscope and isotonic sodium chloride solution to 37-38ºC (vegetative forms stop moving at cooling in 20-30 min., and it is impossible to find them out among stool elements).

NB! There can be 5 types of nonpathogenic amoebae in human intestines.

Amoebae’ separation is during inoculation of artificial mediums (Pavlov’, Beck’ mediums).

It is the most sensitive. Diagnostic titre is 1:128 and more. It is positive from 2-3d week.

Diagnostic titre is 1:80 and more

There are ulcers 10-20 mm in diameter on the unchanged mucosa; these ulcers have edematous, deepened edges, surrounded by hyperemic area and are located more often on folds, the bottom is covered with pus and necrotic mass. There are ulcers, cysts, polyps, amebomae (infiltrations) in case of chronic process.

Uneven filling in of colon’s segments, its spasm.

---

### Professional algorithm for formation skills and experience in diagnostication of pseudotuberculosis and enteric yersiniosis

<table>
<thead>
<tr>
<th>№</th>
<th>Tasks</th>
<th>Sequence of actions</th>
<th>Notices and warnings concerning self-control</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>To master the methods for</td>
<td>I. Inquire into patient</td>
<td>Separation of the complaints which are characteristic for the following</td>
</tr>
</tbody>
</table>
clinical examination of patients with pseudotuberculosis, enteric yersiniosis.

II. To clarify the anamnesis:
1. Anamnesis of disease

To pay attention to acute onset of disease; time, sequence of development, dynamics of
- fever;
- headache;
- eruption;
- damaged oropharyngeal mucosa;
- dysfunction of intestine;
- biliousness;
- arthralgia;
- other symptoms
To determine previous diseases.

To determine the data on the mechanism of fecal-oral transmission mechanism; to pay attention to dietary habits; seasonality of disease.

It should be remembered that: occurrence, intensity, symptom dynamics and severity of course of disease depend on the age of the patient, accompanying pathology.

To pay attention to:
- apathy, adynamia, restraint of the patient;
- body temperature;
- dermahemia;

2. Life history
3. Epidemiological anamnesis

III. To carry out physical examination.
1. General examination:
<table>
<thead>
<tr>
<th>1. General blood test.</th>
<th>To pay attention to characteristic changes: leucocytosis, neutrocytosis, monocytosis, eosinophilia, enlarged erythrocyte sedimentation reaction. Moderate rise in transaminase activity,</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Biochemical blood assay</td>
<td></td>
</tr>
</tbody>
</table>

3. To prescribe laboratory and additional investigations, to interpret the

<table>
<thead>
<tr>
<th>1. Musculoskeletal system.</th>
<th>To pay attention to: arthralgia, absence of symptoms of joint disease;</th>
</tr>
</thead>
<tbody>
<tr>
<td>2. Digestive system:</td>
<td>To pay attention to: biliousness; occurrence, time of development, localization, characteristics of eruption; damaged oropharyngeal mucosa;</td>
</tr>
<tr>
<td>- examination of the tongue;</td>
<td></td>
</tr>
<tr>
<td>- abdomen percussion;</td>
<td></td>
</tr>
<tr>
<td>- abdomen palpation;</td>
<td></td>
</tr>
<tr>
<td>- characteristics of excrements.</td>
<td></td>
</tr>
<tr>
<td>3. Heart-vascular system:</td>
<td>To pay attention to: relative bradycardia, in the case of water deprivation - tachycardia; moderately decreased blood pressure; moderately muffled heart sounds.</td>
</tr>
<tr>
<td>- pulse;</td>
<td></td>
</tr>
<tr>
<td>- blood pressure;</td>
<td></td>
</tr>
<tr>
<td>- heart auscultation.</td>
<td></td>
</tr>
<tr>
<td>5. Nervous system</td>
<td>In the case of severe course of disease – intensity of headache, probable development of meningitis, encephalomenigitis.</td>
</tr>
</tbody>
</table>

To pay attention to: “raspberry tongue”; peculiarities of pain syndrome; Banti's syndrome; Padalka positive syndrome; intensity of diarrheal syndrome, characteristics of excrements; symptoms of peritonitis (their existence indicates perforation of small intestine); relative bradycardia, in the case of water deprivation - tachycardia; moderately decreased blood pressure; moderately muffled heart sounds.
results.


4. Ultrasonic investigation of abdominal cavity organs

5. Bacterial inoculation of blood

6. Stool culture

7. Bacterial inoculation of urine

8. Bacterial inoculation of oropharyngeal lavage

8. Serologic methods:
   - agglutination test,
   - reaction of indirect hemagglutination,

   bilirubinemia due to direct fraction.

   Absence of significant changes in standard course.

   Banti’s syndrome.

   Is prescribed regardless of disease duration during the fever period; it is desirable to fulfill it as soon as possible.

   Is prescribed regardless of disease duration.

   It is desirable to fulfill it in the first days of disease.

   In the first days of disease.

   Are prescribed in paired blood serums at 10 days intervals;

   Diagnostic titer – 1:200.

   Diagnostic titer 1: 100

---

**Shigelosis clinical manifestations depending on its form:**

<table>
<thead>
<tr>
<th></th>
<th>Form of gastrocolitis</th>
<th>Form of colitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amount of stool</td>
<td>bulky</td>
<td>scanty</td>
</tr>
<tr>
<td>Vomiting</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Dehydration signs</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>Epigastrium region</td>
<td>Hypogastrium region</td>
</tr>
<tr>
<td>Gurgling</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>Undigested food</td>
<td>+</td>
<td>–</td>
</tr>
<tr>
<td>debris in stool</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood in stool</td>
<td>-/+</td>
<td>+</td>
</tr>
<tr>
<td>Mucus in stool</td>
<td>-/+</td>
<td>+</td>
</tr>
</tbody>
</table>
### Characteristics of clinical representations of some diseases with exanthema

<table>
<thead>
<tr>
<th>Nosology</th>
<th>Pseudotuberculosis</th>
<th>Yersiniosi s</th>
<th>Typhoid fever</th>
<th>Enterovirus infection</th>
<th>Scarlet fever</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fever</strong></td>
<td>++</td>
<td>++</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td><strong>Intoxication</strong></td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td><strong>Mesenteric adenitis</strong></td>
<td>++</td>
<td>+</td>
<td>++</td>
<td>+/-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Impairment of tonsils</strong></td>
<td>++</td>
<td>-</td>
<td>+/-</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td><strong>Eruption</strong></td>
<td>++</td>
<td>+</td>
<td>+</td>
<td>+/-</td>
<td>+</td>
</tr>
<tr>
<td><strong>Biliousness</strong></td>
<td>++</td>
<td>++</td>
<td>-/+</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td><strong>Tachycardia</strong></td>
<td>+/-</td>
<td>++</td>
<td>-</td>
<td>++</td>
<td>++</td>
</tr>
<tr>
<td><strong>Arthralgia</strong></td>
<td>+</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

4.3. Materials methods of the final phase of occupation

4.3.1. Objectives of the 3rd level

**Task 1**

Patient A., 22 years old, a student, was admitted to isolation hospital on the 3rd day of illness with complaints of weakness, colicky cramp-like abdominal pain, frequent and liquid stool with mucus and blood. The disease onset was marked by the increase of body temperature, headache, tenesmus, frequent stool. He lives at the separate flat with modern conveniences. All family members are healthy. A week ago the patient came back from the village, where similar disease cases were registered. The patient’s state is of moderate severity, body temperature is 37.5 °C. Skin is pale, tongue is moist, coated with white film. Heart sounds are dull. The abdomen is moderately tympanitic (swollen), painful in colon area.
Sigmoid colon is spastic, acute painful. Stool is liquid with mucus and blood streaks (10 times per day).

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

**Task 2**

Patient, 16 years old, male, admitted to department of surgery on the 9th day of disease. Acute attack with elevation of body temperature to 38.5°C, faintness, imperceptible rhinitis. On the second day the papular eruption spread all over the body which lasted for 3 days and was regarded as allergy. He was treated at home. In 5 days periodical abdominal pain developed, sometimes severe (the patient took defense attitude). The patient was hospitalized with diagnosis surgical abdomen.

Physical examination: Body temperature - 38.8°C. Pale, tender abdomen with palpation on the right side in the iliac region, negative peritoneal signs, liver and spleen enlarged. On the 10th day appendectomy was carried out; hyperemia of intestine was observed; in the region of angle the mass of enlarged dense mesenteric lymphonodus was found. Their biopsy was fulfilled. Provisional diagnosis: tuberculous mesenteric adenitis, nonspecific lymphadenitis. Tuberculosis was excluded on basis of Pirquet's reaction and Mantoux test; lymphogranulomatosis was also excluded.

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

**Task 3**

Patient G., 27 years old, complains of appetite loss, fatigability, pain of the lower abdomen, localized mainly at the right iliac region, stool is about 10 times per day, it looks like “raspberry jelly”. Complaints appeared two days ago. Examination revealed body temperature of 36,5°C, tongue coated with white film.
Palpation revealed painfulness along colon, spasm of caecum and ascending segment of colon. From patient’s anamnesis: he came back from Africa two weeks ago.

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

5. Materials of after-work

Proposed topics for essays on the most pressing issues, such as:
«Results generalization of intestinal infections patients examinations from the data of regional isolation hospital»
«Generalization of antiepidemic work experience in the nidus of intestinal infections»
«Prospects for early diagnosis of amoebiasis»
«Clinical and epidemiological characteristics of pseudotuberculosis, enteric yersiniosis»

NEMATODES, CESTOSES, TREMATODOSES

1. Actuality of theme:

Class roundworms (Nematodoses) includes the agents of ascariasis, trichuriasis, trichinosis, enterobiasis (pinworm), filariases, ancylostomiases (hookworms), strongyloidiases, dracunculiasis.
Ancylostoma duodenale and Necator americanus, is estimated to affect approximately one-fourth of the world’s population. The present geographic distribution of hookworm infections lies in the tropical and subtropical zones. Iron deficiency anemia due to hookworms and other nutritional factors looms across most of the developing world.

Infection with the nematode Strongyloides stercoralis is potentially lethal because of its capacity to cause an overwhelming autoinfection, particularly in the immunosuppressed host. Strongloidiasis, although uncommon in comparison with the other major intestinal nematodes, is widely distributed in the tropics.

Ascariasis, or roundworm, infection is the most common helminthic infection of humans with an estimated worldwide prevalence of 1 billion. The causative organism Ascaris lumbricoides, is cosmopolitan in distribution, being most abundant in the tropical countries. Ascaris infection occurs at all stages but is most common in preschool- and young school-age children.

Infection with the nematode Trichuris trichiura is among the most prevalent helminthiasis; approximately 800 million cases occur worldwide, most abundantly in warm, moist regions.

Trichuriasis has a worldwide prevalence; it is most common, however, in poor rural communities and areas in which sanitary facilities are lacking. The intensity of infection is usually light; children in the 5- to 15-years age group have the highest prevalence and probably have heavier worm loads than adults.

Trichinella spiralis is distributed throughout the world apart from Australia and many of the Pacific islands. It is widely spread in nature among a large number of carnivorous animals, humans being an incidental host. Humans usually become infected by eating inadequately processed pork.

Enterobiasis is peroral contagious helminthiasis, anthroponosis. The prevalence of pinworm infection is lowest in nurslings and reaches its maximum in schoolchildren 5-14 years old. Eggs are infective within 6hr of ovideposition and may remain so for 20 days.

Class segmented worms or tapeworms (Cestodoses) includes the agents of
taenia solium (pork tapeworm), taenia saginata (beef tapeworm), echinococcusis (hydatid disease), alveococcosis, hymenolepiasis (dwarf tapeworm), diphyllobothriases.

**Taenia saginata (the beef worm)** occurs throughout the world. Its prevalence is determined by the eating (rare meats) and sanitation habits (human fecally contaminated grazing lands) of people. It has been particularly common in Moslem countries, Ethiopia, and Kenya. It is common, but less frequent, in Central and South America.

**T. solium** occurs most commonly in Eastern Europe, Central and South America, Spain, Portugal, and parts of Africa, China, and India. Cysticercosis is most common in Mexico and in certain parts of Africa and South America.

Diphyllobothriasis occurs in areas where ingestion of raw fish is common, such as Finland, Sweden, Japan, the Baltic countries, and among Canadian and Alaskan Eskimos.

**Hymenolepis nana (the dwarf tapeworm)** is the only tapeworm in which the life cycle can be maintained in nature by humans acting as both the definitive and intermediate host. In this situation, humans spread the disease to other humans by fecal contamination of the environment. This infection is found in all parts of the world, particularly in Africa, South America, and Eastern Europe. It is a particularly common problem among children in institutions.

**Echinococcosis** is seen in most sheep - and cattle-raising areas of the world, including Australia, New Zealand, Argentina, Uruguay, Chile, parts of Africa, Eastern Europe, and the Middle East. It is particularly common in Lebanon and Greece.

Class flukes (Trematodes) includes the agent of fascioliasis, fascipsidosis, clonorchiasis, paragonimiasis, schistosomiases, opisthorchiasis.

**Opisthorchiasis.** Human infections with O.felineus and 0. viverrini are clinically similar to clonorchiasis. These two parasites are common liver flukes of cats and dogs that can occasionally be transmitted to humans. Infection with felineus is endemic in Southeast Asia and eastern Europe, whereas 0. viverrini
infection is commonly found in Thailand.

Fascioliasis is peroral biohelminthiasis. Infection with the liver fluke F. hepatica is a cosmopolitan zoonosis throughout the sheep-raising areas of the world. Human infections have been reported, particularly from South America, Europe, Africa, China, and Australia.

2. Study purpose of practical studies:

2.1. Students have to know:

- Etiology of nematodoses;
- Geographical widespread of nematodoses;
- Epidemiology of nematodoses;
- Life cycles of nematodoses;
- Pathogenesis of nematodoses;
- Clinical manifestations of nematodoses;
- Laboratory diagnostics of nematodoses;
- Principles of the treatment and prophylaxis;
- Etiology of cestodoses and trematodoses;
- Geographical widespread of cestodoses and trematodoses;
- Epidemiology of cestodoses and trematodoses;
- Life cycles of cestodoses and trematodoses;
- Pathogenesis of cestodoses and trematodoses;
- Clinical manifestations of cestodoses and trematodoses;
- Laboratory diagnostics of cestodoses and trematodoses;
- Principles of the treatment and prophylaxis;

2.2. Students have to be able:

- to ask history of the disease with estimation of the epidemiological data;
• to provide examination of the patient and reveal the main symptoms and syndromes of cestodoses and trematodoses;
• to perform differential diagnostics;
• to compose the plan of the laboratory and additional examination of the patient;
• to interpretate the results of the laboratory investigation;
• to analyze the results of the specific methods of the diagnostics;
• to compose individual plan of the treatment with account of the epidemiological data, stage of the disease, presence of the complications, severity of state, allergological history;
• to compose the plan antiepidemic and prophylaxis measures in the focus of the infection;
• to give recommendations relatively to regime, diet, examination in the period of convalescence.
• to ask history of the disease with estimation of the epidemiological data;
• to examinate the patient and reveal the main symptoms and syndromes of nematodoses;
• to perform differential diagnostics;
• to compose the plan of the laboratory and additional examination of the patient;
• to interpretate the results of the laboratory investigation;
• to analyze the results of the specific methods of the diagnostics of nematodoses;
• to compose individual plan of the treatment with account of the epidemiological data, stage of the disease, presence of the complications, severity of state, allergological anamnesis;
• to compose the plan epidemic and prophylactic measures in the focus of the infection;
to give recommendations relatively to regime, diet, examination in the period of convalescence.

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

3.1 Basis knowledge, skills, which are necessary for study of topic

(Interdisciplinary integration)

<table>
<thead>
<tr>
<th>Discipline</th>
<th>To know</th>
<th>To be able</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous discipline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biology</td>
<td>Classification, morphology, life cycle of nematodoses</td>
<td>To interpretate of the results of specific methods of the diagnostics of nematodoses</td>
</tr>
<tr>
<td>Internal diseases</td>
<td>Methods and roles of the objective examination of the patients.</td>
<td>To ask history cases, provide objective examination, determine presence of the pathological symptoms, estimate data of the clinical examination</td>
</tr>
<tr>
<td>Pharmacology</td>
<td>Drugs which are used for treatment of nematodoses</td>
<td>Select effective drug, prescribe adequate dose</td>
</tr>
<tr>
<td>Therapy</td>
<td>The main clinical manifestations of the disease with syndrome of prolonged fever, damage of the respiratory tract</td>
<td>To estimate data of the clinical examination, timely mark diagnosis and prescribe the laboratory investigation</td>
</tr>
<tr>
<td>Surgery</td>
<td>Clinical manifestations of the appendicitis, peritonitis, cancer of the liver, intestinal obturation</td>
<td>To determine complications, prescribe the laboratory investigations and treatment</td>
</tr>
<tr>
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</tr>
<tr>
<td>Epidemiology</td>
<td>The main links of the epidemic process in nematodoses (the source of infection, the mechanisms of the transmission, routes of the transmission), geographical distribution in the world</td>
<td>To ask epidemiological history, prescribe antiepidemic measures in the focus of the infection, to fill up the card of the epidemiological examination</td>
</tr>
<tr>
<td>Family medicine</td>
<td>Epidemiology, pathogenesis, the features of the clinical course of the different phases of the development of the important nematodoses, possible complications, principles of the treatment and prophylaxis</td>
<td>Perform differential diagnostics of nematodoses with diseases of the other origin in dependence on phase of the disease. To distinguish nematodoses, their complications, interpreted the results of the laboratory investigations (blood count, methods of the specific diagnostics, render mergency)</td>
</tr>
<tr>
<td>Intradiscipline integration</td>
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</tr>
<tr>
<td>Infectious disease</td>
<td>The features of the infectious diseases. The principles of the diagnostics, treatment and prophylaxis. Epidemiology, pathogenesis, clinical manifestations of the different phases of the pathogenesis of nematodoses, possible complications. The criterion of the diagnosis, principles of the treatment (etiotropic, pathogenetic, symptomatic therapy), prophylaxis</td>
<td>To perform differential diagnostics of nematodoses with the other infectious and noninfectious diseases, distinguish nematodoses and their complications, interpreted results of the laboratory investigations (general clinical investigations and specific diagnostics), prescribe treatment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Discipline</th>
<th>To know</th>
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</thead>
<tbody>
<tr>
<td>Previous discipline</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biology</td>
<td>Classification, morphology, life cycle of trematodes and cestodoses</td>
<td>To interprite of the results of specific methods of the diagnostics of trematodoses and cestodoses</td>
</tr>
<tr>
<td>Internal diseases</td>
<td>Methods and roles of the objective examination of the patients.</td>
<td>To ask history, provide objective examination, determine presence</td>
</tr>
<tr>
<td><strong>Pharmacology</strong></td>
<td>Drugs which are used for treatment of cestodoses and trematodoses</td>
<td>Select effective drug, prescribe adequate dose</td>
</tr>
<tr>
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</tr>
<tr>
<td><strong>Therapy</strong></td>
<td>The main clinical manifestations of the disease with syndrome of prolonged fever, damage of the respiratory tract</td>
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<td>To determine complications, prescribe the laboratory investigations and treatment</td>
</tr>
<tr>
<td><strong>Epidemiology</strong></td>
<td>The main links of the epidemic process in cestodoses and trematodoses (the source of infection, the mechanisms of the transmission, routes of the transmission), geographical distribution in the world</td>
<td>To collect epidemiological anamnasis, prescribe antiepidemic measures in the focus of the infection, to fill up the card of the epidemiological examination</td>
</tr>
<tr>
<td><strong>Family medicine</strong></td>
<td>Epidemiology, pathogenesis, the features of the clinical course of the different phases of the development of the important cestodoses and trematodoses,</td>
<td>Perform differential diagnostics of cestodoses and trematodoses with diseases</td>
</tr>
</tbody>
</table>

**Next discipline**
possible complications, principles of the treatment and prophylaxis of the other origin in dependence on phase of the disease. To distinguish cestodoses and trematodoses, their complications, interpret the results of the laboratory investigations (blood count, methods of the specific diagnostics, render emergency)

<table>
<thead>
<tr>
<th>Intradiscipline integration</th>
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<tbody>
<tr>
<td><strong>Infectious disease</strong></td>
</tr>
</tbody>
</table>
### 3.2 Contents of the class.

**ASCARIDOSIS**

**Etiology**

- Ascaris lumbricoides

**Epidemiology**

- Anthroponosis. Peroral helmints.

The mechanism of the transmission is fecal-oral

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Early (migratory) phase</th>
<th>Late (intestinal) phase</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clinics</td>
<td></td>
<td>damage of the gastrointestinal tract</td>
</tr>
<tr>
<td>↓</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>General toxic syndrome</td>
<td>Allergic syndrome</td>
<td>damage of the respiratory tract</td>
</tr>
<tr>
<td>– increase the temperature</td>
<td>– itch</td>
<td>– bronchitis</td>
</tr>
<tr>
<td>– arthralgia</td>
<td>– skin rash</td>
<td>– pneumonia</td>
</tr>
<tr>
<td>– mialgia</td>
<td></td>
<td>– rhinopharingitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>– Leffler’s syndrome</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Complications</td>
</tr>
<tr>
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<td>↓</td>
</tr>
<tr>
<td>– acute appendicitis</td>
<td>– perforation of the intestine, peritonitis</td>
<td></td>
</tr>
<tr>
<td>– mechanic jaundice</td>
<td>– asphyxia</td>
<td></td>
</tr>
<tr>
<td>– purulent cholecystitis</td>
<td></td>
<td></td>
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<tr>
<td>– abscess of liver</td>
<td></td>
<td></td>
</tr>
<tr>
<td>– pancreatitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>– obstruction of the intestine (till perforation)</td>
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</tr>
</tbody>
</table>

Diagnostics
- leukocytosis
- eosinophilia (30-40%)

Specific diagnostics

↓

- larvae in the sputum  ovoskopy

Serological methods

↓

- RIHA, immunofermentive method

Treatment

- Albendasolum
- Mebendazole

Antihistaminic remedies

Prophylaxis

- roles of the personal hygiene
- sanitary-hygienic measures

TRICHINELLOSIS

Etiology  
Trichinella spiralis

Zoonosis. Peroral biohelminthiasis.

Epidemiology  
The sources of infection

↓  ↓

Natural foci  Anthropurgic foci

(beers, wolfs, foxes)  (pigs, dogs, cats)

The factor of the transmission is insufficient thermal processing of the meat

Immunity is no prolonged. It is possible repeated infection

Pathogenesis  
Larvae are freed from the cyst walls in the stomach and in the small intestine by acid-pensis digestion → blood → skeletal muscles
A cyst wall develops around the larva and may eventually calcify. Larvae may remain viable for several years.

The leading factor of pathogenesis – sensitization → allergic vasculitis

Clinical manifestations
– enteritis, nausea, abdominal pain
– periorbital edema and edema of the face
– mialgia
– fever
– eosinophilia (50-90%)
– skin rash

Complications
– myocarditis
– pneumonia
– meningoencephalitis
– hepatitis
– nephritis
– systemic vasculitis
– thrombophlebitis
– thrombocytopenia

Diagnostics
– trichinelloskopy (investigation of the meat)
– immunological methods
– skin allergic test

Treatment
Etiotropic
– mebendazole
– albendazolum
Pathogenetic
– desensibilization
– desintoxication therapy

Prophylaxis
– investigation of the meat of presence for Trichinella spiralis

ANCYLOSTOMIDOSES (ANCYLOSTOMOSIS AND NECATOROSIS)

Etiology
Ancylostoma duodenale / Necator americanus

Epidemiology
Anthroponosis. Percutaneous and peroral helminthiases

Main route of transmission - percutaneous

The susceptibility is general
<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Early (migratory) phase</th>
<th>Late (intestinal) phase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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<td>↓</td>
</tr>
<tr>
<td></td>
<td>- allergic reaction</td>
<td>Fixation of ancylostoma to mucous membrane of the small intestine (hypoalbuminemia)</td>
</tr>
<tr>
<td></td>
<td>- perivascular infiltration of the lungs</td>
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<tr>
<td>Clinical manifestations</td>
<td>- itch, urticar rash, increase the temperature, cough with asthmatic component (sputum with blood)</td>
<td>- nausea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- vomiting</td>
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<tr>
<td></td>
<td></td>
<td>- pain in the epigastrium</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- diarrhea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- anemia is leading symptom (in 3-6 month)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- pain in the muscles</td>
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<tr>
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<td>- dry skin</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- edema of the low extremeties</td>
</tr>
<tr>
<td>Compilations</td>
<td>- infection of the skin in the area of the entrance gate → sepsis, tetanus, erythypilas, carbuncule</td>
<td>- hypochromic anemia</td>
</tr>
<tr>
<td></td>
<td>- Quinke’s edema</td>
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<tr>
<td></td>
<td>- laryngospasm</td>
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</tr>
<tr>
<td>Diagnostics</td>
<td><strong>General methods</strong></td>
<td><strong>Biochemical methods</strong></td>
</tr>
<tr>
<td></td>
<td>- leukocytosis</td>
<td>- decrease general protein</td>
</tr>
<tr>
<td></td>
<td>- eosinophilia</td>
<td>- general of serum Fe</td>
</tr>
<tr>
<td></td>
<td>- increase the ESR</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- hypochromic anemia</td>
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<tr>
<td></td>
<td><strong>Specific methods</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td>- finding of the eggs of helminths in the feci</td>
<td></td>
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</tbody>
</table>

**Treatment**

<table>
<thead>
<tr>
<th>Etiotropic therapy</th>
<th>Pathogenetic therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>- albendazole</td>
<td>- Treatment of anemia</td>
</tr>
<tr>
<td>- levamizole</td>
<td>- Treatment of hypoalbuminemia</td>
</tr>
<tr>
<td>- mebendazole</td>
<td>- Desensibilization</td>
</tr>
<tr>
<td>- thiabendazole</td>
<td>-</td>
</tr>
</tbody>
</table>
Prophylaxis

- dehelmintization of the patients
- sanitary-hygienic measures
- devastation of the soil

**STRONGYLOIDOSIS**

<table>
<thead>
<tr>
<th>Etiology</th>
<th>Strongyloides stercoralis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epidemiology</td>
<td>Anthroponosis.</td>
</tr>
<tr>
<td></td>
<td>Percutaneous and peroral geohelminthiasis</td>
</tr>
<tr>
<td></td>
<td>The factors of the transmission are soil, vegetables, fruits</td>
</tr>
<tr>
<td></td>
<td>The contingents of risk is miners, earthworkers</td>
</tr>
<tr>
<td>Pathogenesis</td>
<td>In infection through the skin larvae → into the tissue → blood and lymphatic vessels → heart → lungs → oral cavity → intestine</td>
</tr>
<tr>
<td></td>
<td>Migratory phase</td>
</tr>
<tr>
<td></td>
<td>Intestinal phase</td>
</tr>
<tr>
<td>Clinical manifestations</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- allergic and general toxic reactions</td>
</tr>
<tr>
<td></td>
<td>- skin itch</td>
</tr>
<tr>
<td></td>
<td>- rash</td>
</tr>
<tr>
<td></td>
<td>- Leffler’s syndrome</td>
</tr>
<tr>
<td></td>
<td>- damage of mucous membrane</td>
</tr>
<tr>
<td></td>
<td>- disorder of the absorption of the fats and</td>
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<td></td>
<td>- hypoproteinemia</td>
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<td>- ulcerous damage of the intestine</td>
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<td>- perforative peritonitis</td>
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<td>- necrotic pancreatitis</td>
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<td></td>
<td>- intestinal hemorrhages</td>
</tr>
<tr>
<td></td>
<td>- myocarditis</td>
</tr>
<tr>
<td></td>
<td>- meningoencephalitis</td>
</tr>
<tr>
<td></td>
<td>- chaxexia</td>
</tr>
</tbody>
</table>
Diagnostics

General clinical methods
- eosinophilia (up to 40%) is marked in early phase
- anemia, accelerated ESR are marked in late phase

Biochemical methods
- dysproteinemia
- increase level of bilirubinum

Specific methods
- finding of the larvae in sputum, duodenal secretum, feci

Treatment

Migratory phase
- pathogenetic therapy (antiallergic remedies)
- albendazole, thiabendazole

Intestinal phase
- detection and treatment of the patient
- general sanitary measures
- in endemic areas it is necessary to use only boiling water
- defense of the open parts of the body from larvae
- devastation of the soil (10% NaCl, mineral fertilizations)

ENTEROBIOSIS

Etiology
Enterobius vermicularis
Anthroponosis.
Peroral contagious helminthiasis.

Epidemiology
The mechanism of the transmission is fecal-oral

Pathogenesis
The infection is realized in oral cavity
The maturation of the invasive eggs with larvae is finished in 4-6 hours on the skin of the human.
Larvae in the distal section of the small intestine → rectum (female) → eggs on the perianal area and perineum
The mechanic lesion of the mucous membrane → inflammatory changes, hemorrhages
Clinical manifestations
- itch
- pyodermia
- headache
- disorder of sleep
- irritation
- dizziness

Compilations
- appendicitis
- perforation with peritonitis

Diagnostics
- perianal scrape

Treatment

Etiotropic
- mebendazole
  - treatment is effective only in observance of the rules of the personal hygiene

Prophylaxis
- dehelmintization of the patients
- sanitary-hygienic measures
- in the foci chemi prophylaxis is performed

TRICHOCEPHALOSIS

Etiology Trichocephalus trichiuris

Epidemiology Anthroponosis. Peroral biochelminthiasis

The mechanism of transmission is fecal-oral

The factors of transmission are vegetables, fruits, water

The infection of the human is realized in use

with the food only matured eggs

Pathogenesis

The eggs with invasive larvae → penetrations into the mucous membrane of the small intestine → the large intestine

↓

Traumatization of the wall of Penetration into the mucous the large intestine → and submucous layers → admixture of the bacterial flora constant irritation of the
→ inflammatory reaction → interoreceptors → disorders of
formation of the infiltrates (typhlitis)
→ disorders of the function of the stomach,
duodenum and central nervous system

Clinical manifestations↓↓
- diarrhea
- hemocolitis
- tenesmus
- pain in the right hypogastrium
- hypochromic anemia
- weakness
- dizziness
- decrease of arterial pressure
- tachycardia

Complications
- rectal prolapse
- appendicitis
- hypochromic anemia

Diagnostics
- eosinophilia
- hypochromic anemia
- finding of the eggs of helminths in feci

Treatment
- albendazolum
- mebendazolum

Prophylaxis
- detection and treatment of the patient
- sanitary-hygienic measures
- washing of vegetables and fruits

**DIPHILLOBOTHRIASIS**

**Etiology** (Diphyllobothrium latum)

**Epidemiology**

Zoonosis. Peroral biohelminthiasis

The source of the infection and definitive host is human.

Intermediate host is a copepod (crustacean)

The ancillary host is a fresh water fish
Infection of the human is realized only in the use into the food of the thermic insufficiency cooking fish

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Mechanic influence</th>
<th>Toxicoellergic influence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Attachment to the intestinal wall → attachment by bot to mucous membrane → athrophy and necrotization of the mucous membrane</td>
<td>Sensibilization by products of the metabolism of helminth → endogenic hypo- and avitaminosis B₁₂ and folic acid</td>
<td></td>
</tr>
</tbody>
</table>

↓ ↓

| Clinical manifestations | – weakness | – anemia |
| – dizziness | – pallor |
| – signs of the damage of the gastrointestinal tract | – glossitis |
| – it is possible dynamic or occlusive obstruction ileus | – loss of tongue papillae |
| – achilia |

| Complications | – anemia |
| – obstruction of the intestine (till perforation) |
| – finding of the helminth’s eggs in the feci |

| Diagnostics | – hyperchromic anemia |
| – revealation of the eggs and proglotids of D.latum |

| Treatment | Etiotropic therapy |
| – fenasal |
| – praziquantel |
| – vermox |
| | Pathogenetic therapy |
| – treatment of anemia |

| Prophylaxis | – treatment of the patients |
| – fish tapeworm invasion is prevented by through cooking of freshwater fish |

**THE BEEF TAPEWORM**

**Etiology**

*Taenairhynchus saginatus*

**Epidemiology**

Anthropo- and Peroral helmintiasis.
The source of the invasion and definitive host is human. The intermediate host is cattle.

The factor of the transmission is insufficient thermal processing of the meat.

Pathogenesis

cysticercus → small intestine → larvae fixate to mucous membrane

↓

In 2 months 2 месяца → adult worm → discharge of proglottids filled by eggs

Mechanic action

- lesion of the mucous membrane in the site of the fixation of parazite
- irritation of the interoreceptors
- disorders of the absorption
- intoxication
- sensibilization

Clinical manifestations

- abdominal pains (especially right hypogastrium area)
- nauses
- meteorism
- disorders of appetite
- disorders of stool
- hypoacidic gastritis
- it is typical the active exit of proglottids act of the defication (especially at the night)

General toxic symptoms

- weakness
- fatigue
- disorders of the sleep
- irritability
- headache
- dizziness

Complications

- acute appendicitis
- acute pancreatitis
- acute cholecystitis
- occlusive ileus

Diagnostics

General clinical methods

- moderately eosinophilia
- moderately anemia

Specific methods

- revelation of the eggs and proglottids of helminth in the feci

Treatment
PRAZIQUANTEL

- Prophylaxis
  - dehelminthization
  - devastation of the soil

- veterinary-sanitary control
- the maintenance of the roles of the terminal processing of the meat

PORK TAPEWORM AND CYSTICERCOSIS

Etiology

Taenia solium

Epidemiology

Anthroponosis.

Peroral biohelminthosis

The source of the invasion and definitive host is human. The intermediate hosts are pigs, dogs, cats, sheep.

The factors of transmission is use into the food meat of the pig with cystecercs.

Pathogenesis

Taenia solium arised in use of the meat with larvae of the parasite. The adult helminth parasites in the organism of human. The human is the definitive host.

Cysticercosis arises in the penetration of the eggs of helminth into the intestine. The human is an intermediate host.

\[
\begin{array}{l}
\text{Taenia solium} \\
\text{In infection by cystecercs in 2-3 months} \\
\rightarrow \text{adult helminth} \\
\rightarrow \text{small intestine} \\
\downarrow \\
\text{Mechanic influence} \\
\rightarrow \text{the lesion of the mucous membrane in the site of the fixation}
\end{array}
\]

\[
\begin{array}{l}
\text{Cystecercosis} \\
\text{In infection by eggs of T. soluem in 2-2,5 month} \\
\rightarrow \text{cystecercs} \\
\rightarrow \text{brain, myocardium, skeletal musculature, eyes} \\
\downarrow \\
\rightarrow \text{compression of the tissues} \\
\rightarrow \text{inflammatory process around parasites} \\
\rightarrow \text{toxic and allergic action}
\end{array}
\]
of parasite
- irritation of interoreceptors
- disorder of the process of absorption
- intoxication
- sensibilization

\[ \downarrow \]

Clinical manifestations
- moderate marked dyspeptic manifestations and general toxic syndrome

\[ \downarrow \]

- the clinical manifestations is dependence on localization (brain, eyes, subcutaneous, cellulose)

Diagnostics

General clinic methods
- moderate eosinophilia

*Taenia soleum*
- finding of the helminth’s eggs in the feci
- ovoscopy

*Cystecercosis*
In dependence on localization
(ophtalmoscopy, roentgenogram, computed tomography)

Serological methods
- ELISA

Treatment

*Taenia soleum*
- praziquantel

*Cystecercosis*
- surgical, praziquantel

Prophylaxis

Relevation and treatment
- general sanitary measures
- veterinary control

**HYMENOLEPIASIS**

Etiology
- Hymenolepis nana

Epidemiology
- Peroral contagenous helminthiasis.

The human is the main sourse of the invasion.

The human is the definitive and intermediate host
The ancillary hosts are rats
The mechanism of transmission is fecal-oral
The factors of the transmission are vegetables, fruits, flies, cockroach

<table>
<thead>
<tr>
<th>Pathogenesis</th>
<th>Mechanic lesion</th>
<th>The part stays in the intestine (autoinvasion)</th>
</tr>
</thead>
<tbody>
<tr>
<td>eggs → stomach → penetration into the mucous membranes of the upper sections of the small intestine → larvae</td>
<td>- edema, hyperemia of the all layers of the intestine wall</td>
<td>- the products of the helminth’s metabolism cause</td>
</tr>
<tr>
<td>(cystecercoided) → eggs → environment</td>
<td>- dystrophy, necrosis of the epithelium</td>
<td>- intoxication and allergization</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- damage of the fermentative system</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- dysbacteriosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- disorder of the functions of the liver</td>
</tr>
</tbody>
</table>

Clinical manifestations of the gastrointestinal tract:
- nausea
- vomiting
- decreased appetite
- salivation
- abdominal pain
- diarrhea
- moderate hepatomegaly

Clinical manifestations of the nervous system:
- weakness
- fatigue
- headache
- dizziness
- irritation
- seizures
- epileptic attacks

Sensibilization:
- urticaric rash
- vasomotoric rhinitis
- asthmatic bronchitis
- Quinke’s edema

Complications:
- dysbacteriosis

Diagnostics:
- moderate eosinophilia
- identification of the double membrane eggs in the stool

Treatment:
- praziquantel
- niclosamide
Prophylaxis
- paramomycin
- dehelmintization
- sanitary-hygienic measures
- struggle with rodents

ECHINOCOCCOSIS

Etiology
Echinococcus granulosus

Epidemiology
Zoonosis. Peroral biohelmintisis. There are 2 types of the foci:

1. The natural foci (wild animals: beers, elks, wolfs, foxes)
2. Synantropic foci (sheeps, goats, dogs) The definitive host is dog.

Pathogenesis
Eggs of helminths → fixation on the mucous membrane of duodenum → oncosphere → V.portae → liver (or brain, heart, kidney) → formation of the hydatid cyst in the site of the penetration

↓
Toxico-allergic action of the metabolic products of Echinococcus

↓
The local damage
- compression of the tissues
- the disorders of trophics
- necroses, granulation
- replacement of the perished by connective tissue

Clinical manifestations
- weakness, fatigue
- headache
- increase of the temperature
- dyspeptic manifestations
- skin rash, itch

Location of hydatid cysts:
Liver
(usually in the right lobe)
- jaundice (cholestasis)
- pain in the right part of the chest
- pain in the right subribal area
- on palpation – immovable compact formation
Lungs
- dyspnea
- pain in the chests
- cough

Brain
- clinics of the tumor

Complications

The rupture of hydatid cyst → anaphylactic shock

The cyst may rupture:

1. into the biliary tract → cholangitis intermittent ductal obstruction
2. through the capsule of the liver → into the peritoneal cavity → dissemination of scolices → formation of daughter cysts

Complications
- an acute inflammatory reactions (peritonitis, meningitis, pleurisy)
- the suppurating of the cysts → abscess
- in prolonged invasion → amyloidosis of the parenchymatous organs
  echinococcosis of the liver
  There are 3 stages
  - mechanic jaundice
  - cirrosis of the liver
  - portal hypertension
  echinococcosis of the lungs → massive hemorrhage
  echinococcosis of the brain → epileptic attacks, loss of the vision, pareses, paralyses
  echinococcosis of the ovaries → rupture of the salpinx → hemorrhage

Diagnostics
- leukocytosis
- anemia
- accelerated ESR
- eosinophilia
- roentgenograms
- USD
- computed tomography
- serological reactions (complement fixation, hemagglutination and other)
- a skin test (Casoni)

Treatment
- the leading method of the treatment is surgical

Prophylaxis
- roles of the personal hygiene
- helminthological investigation and dehelminthization of the dogs
OPISTHORCHIASIS

Etiology
Opisthorchis felineus

Epidemiology
The natural-focal zoonosis. Biohelmintiasis

The definitive hosts are human and animals (cats, dogs, pigs)

The intermediate hosts are fresh water mollusk from the family Bithynia; fishes from the family (metacercariae)

The factors of the transmission is fish with larvae

Pathogenesis
Metacercariae → bile ducts → gall bladder → intrahepatic bile ducts and ducts of the pancreas

↓

Early acute phase
Toxic action of the products of the destruction of larvae → toxicoallergic reactions → lesion of the walls of bile ducts and ducts of pancreas

↓

Late chronic phase
- allergic action of helminths
- toxic action
- mechanic action

Clinic
- diarrhea
- allergic rash
- pain in the right hypogastrium (in the patient which came to endemic regions; asymptomatic course in the inhabitants of the endemic regions
- pain in the area of the liver and gall bladder
- dyspeptic manifestations
- hepatomegaly
- icteric sclerae

Complications
- abscessis of the liver
- cholangitis
- rupture of parazitive cyst
- peritonitis
- primary cancer of the liver

Diagnostics
- eosinophilia
- leukocytosis
- ovoscopy
FASCIOLOPSIASIS

Etiology  Fasciola hepatica

Epidemiology  Zoonosis with natural foci. Peroral biochelminthiasis.

The source of infection and definitive hosts are human, cattle,
sheep, horses, rodents.

The intermediate host is snail mollusks

The factors of the transmission are water flowers, vegetables

Pathogenesis  Penetration of larvae of F. Hepatica into intestine → liver and
bile ducts, pancreas, brain, eyes

↓  ↓

Acute migratory phase  Late (chronic) phase
- toxicoallergic action of the helminth’s products
- there are eosinophilic infiltration and necroses of the hepatocytes in the liver
- pain in the right hypogastrium area
- enlargemenent of the liver
- diarrhea

Clinical manifestation

Acute phase  Subacute phase  Chronic phase

General toxic and allergic manifestations
- increase of the temperature
- skin rash

Treatment
- Praziquantel
- Pathogenetic therapy

Prophylaxis
- The guarding of the water reservoirs
- Sanitary control of the processing of the fish products

- biochemical methods
- USD, cholecystography
- Serological methods in chronic stage
- arthralgia
- hepatomegaly, jaundice
- loss of weight
- disorders of the activity of the gastrointestinal tract
- admixter of the secondary flora

↓

Complications
- anemia
- cachexia
- purulent meningitis
- cholecystitis
- flegmone of the gall bladder
- abscess of the liver
- mechanic jaundice
- acute pancreatitis
- fibrosis of the liver
- myocarditis

Diagnostics
- eosinophylia
- anemia
- leukocytosis

- biochemical methods
  - the increase of the activity of ALT and AST
  - dysproteinemia

- specific diagnostics
  - serological methods (reaction complement fixation)
  - finding of the helminth’s eggs in the feci

Treatment
- chloxylum
- praziquantel
- antibiotics (in complications)

Prophylaxis
- don’t use into the food water plants in the foci of fasciolopsiasis
- boiling of the water
- garding of water reservoirs from the pollution
- sanation of the infected animals
3.3 Literature:

3.4. Materials for the self control

3.4.1. Control questions for self control:

1. What helminthiasis are treated to the class of the round warns (Nematodoses)?
2. What is the mechanism and factors of the infection in ascaridosis?
3. What are the phases of the pathogenesis in ascaridosis?
4. What are the clinical manifestations of the migratory phase in ascaridosis?
5. What are the complications of ascaridosis?
6. What is the blood count of the early (migratory) phase in ascaridosis?
7. What are the principles of the therapy of ascaridosis?
8. What are the principle ways of the infection in ancylostomidosis?
9. What etiotropic and pathogenesis therapy should be administered in ancylostomidosis?
10. What is the mechanism of the invasion in trichinelllosis?
11. What are the cardinal signs of trichinelllosis?
12. What methods are used for diagnostics of trichinelllosis?
13. What are the factors of the transmission of the infection in trichocephalosis?
14. What are the ways of the infection in enterobiosis?
15. What are the criterions of the diagnosis in enterobiosis?
16. What are the ways of the transmission in strongyloidosis?
17. What are the contingents of the risk for infection in strongyloidosis?
18. What are the main clinical symptoms in strongyloidosis?
19. What are the complications of strongyloidosis?
20. What laboratory methods are used for diagnostics of strongyloidosis?
21. What is the definitive and intermediate host in diphillobothriasis?
22. What is the life cycle Taenia soleum (pork worm)?
23. What are the clinical manifestations of Taenia soleum?
24. What are the clinical manifestations in cystecercosis?
25. What is the life cycle of Hymenolepis nana?
26. What is epidemiology of hymenolepiaisis?
27. What are the leading clinical syndromes in hymenolepiasis?
28. What is the etiology of echinoccosis?
29. What are the leading factors of the pathogenesis of echinoccosis?
30. What is the possible localization of cyst in echinoccosis?
31. What are the clinical manifestations in dependence on localization of cyst in echinoccosis?
32. What methods are used for diagnostics of echinoccosis?
33. What is the principal method of the treatment in echinoccosis?
34. What are the features of the life cycle trematodoses?
35. What is the epidemiology of opisthorchiasis?
36. What are the clinical manifestations of the late (chronic) stage of opisthorchiasis?
37. What are the complications of opisthorchiasis?
38. What methods are used for diagnostics of opisthorchiasis?
39. What treatment should be administered in trematodoses?
40. What methods are used for prophylaxis of trematodoses?

3.4.2. Tests for self control      a=2

1. Where do the eggs of Ascaris lumbricoides develop into invasive stage?
   A. in intestines of human,
   B. in perianal folds during 4-6 hours,
   C. in the ground during 2 weeks by temperature 25°C,
   D. in lungs,
   E. in liver of human.

2. The source of Enterobiosis invasion is infected:
   A. cattle,
   B. pig,
   C. human,
   D. cat,
   E. fish.
3. What is the main method of diagnostics of Enterobiosis?
A. indirect hemagglutination test,
B. immune-enzyme analysis,
C. coprogram,
D. investigation the scrape from the perianal skin,
E. coproculture.

4. For what helminthiasis percutaneous way of transmission is typical?:
A. strongyloidiasis,
B. necatoriasis,
C. ankylostomiasis,
D. B and C are correct,
E. everything is right.

5. At serious ascariasis following surgical complications are typical, except:
A. asphyxia,
B. suppurative cholangitis,
C. rectal prolapse,
D. intestinal obstruction,
E. abscesses of the liver.

6. Which method of treatment of Enterobiosis is the most correct:
A. anthelmintic drugs combined with strict hygiene regime,
B. anthelmintic drugs combined with daily night spasmolytic enemas,
C. 3-4 courses of treatment with metronidazole,
D. surgical treatment,
E. daily night enemas with ammonia soda to prevent perianal itching.

7. Indicate which type of worms belongs to contagious?:
A. trichuriasis,
B. teniasis,
C. enterobiasis,
D. opisthorchiasis,
E. ascariasis.
8. The patient 30 years old has weakness, fever up to 39 °C. On R-graphy eosinophilic lung infiltrates migratory nature were revealed. What disease the patient has?
A. pulmonary tuberculosis,
B. lobular pneumonia,
C. ascariasis, migration phase,
D. polisihmentarna pneumonia,
E. lung cancer.

9. The patient complains of weakness, poor appetite, intermittent abdominal pain. In ovsokopy study of feces roundworm's eggs were found. What treatment should be prescribed to patient for deworming?
A. hygienic treatment,
B. levamisole,
C. ceftriaxone,
D. norfloxacin,
E. fenasal.

10. The patient with ancylostomiasis has manifestation of iron deficiency anemia (decreased absolute number of red blood cells, decreased hemoglobin and color index). Indicate the cause of this clinical manifestation in ancylostomiasis?
A. bleeding,
B. hookworm are true hematophah,
C. toxic-allergic action of ankilostom metabolites,
D. perforation of vessels,
E. violation vascular permeability.

11. In the specific diagnostics of trichinellosis the main is to detect:
A. ovum in feces,
B. helminthiasis in feces,
C. larva in biopsy material,
D. ovum in scraping from perianal folds,
E. eosinophilia in blood.
12. The symptoms of which helminthic invasion are: acute onset, fever, muscular pains, conjunctivitis, allergic rash?
   A. echinococcosis,
   B. ascaridiasis,
   C. diphyllobothriasis,
   D. opisthorchiasis,
   E. trichinellosis.

13. The patient A. has headache, deltoid muscles pains, appearance of skin rash. The medical examination has detected painfulness of muscles of left shoulder joint. The eosinophilia in the blood is up to 40%. The rontgenography of left shoulder joint has shown calcifications (size 3mm x 1,2mm) with effect of perifocal inflammation in soft tissues of deltoid muscle. What disease is characterized by these clinical signs?
   A. nonspecific polyarthritis,
   B. trichinellosis,
   C. leptospirosis,
   D. toxicallergic dermatitis,
   E. extrapulmonary tuberculosis.

14. Trans-dermal route of transmission is typical?
   A. trichocephalosis,
   B. teniasis,
   C. strongylosis,
   D. opisthorchiasis,
   E. ascariosis.

15. Leading syndrome in trichinellosis?
   A. immunosuppressive,
   B. toxic-allergic,
   C. adynamic – neurotic,
   D. dyspepsial,
   E. all not true.
16. The development of mature forms of Trichinella are:
A. on the microvilli in the small intestine,
B. in the striated muscle,
C. in the submucosal layer of the small intestine,
D. in the large intestine,
E. in the stomach.

17. The development of the larval stage of Trichinella are:
A. on the microvilli in the small intestine,
B. in the intermuscular septa of the striated muscle,
C. in the submucosal layer of the small intestine,
D. in the large intestine,
E. in the stomach.

18. Transmissible mechanism of transmission is typical:
A. dirophyllariosis,
B. teniasis,
C. strongylosis,
D. opisthorchiasis,
E. ascariasis.

19. By biohelminths include all, exept:
A. difilobotrios,
B. strongylosis,
C. teniasis,
D. teniarinhosis,
E. trichinellosis.

20. Dogs are the source of contamination:
A. teniasis,
B. opisthorchiasis,
C. strongylosis,
D. toxocarosis,
E. ascariasis.
21. What is the agent of pork worm disease?
A. Taenia soleum,
B. Hymenolepis nana,
C. Taeniarhynchus saginatus,
D. Trichinella spiralis,
E. Dyphylobotrium latum

22. What are the principal clinical syndromes in pork worm disease?
A. respiratory syndrome,
B. cholestatic syndrome,
C. dyspeptic syndrome,
D. meningeal syndrome,
E. everything is right

23. How is the source of invasion in case of cystecercosis?
A. cattle,
B. dog,
C. pig,
D. human,
E. fish

24. What methods are used for diagnostics of cystecercosis?
A. General blood analysis,
B. biochemistry methods,
C. computed tomography,
D. investigation of faeces,
E. nothing is right

25. The invasion of the human with Dyphillobotirum latum is possible to get after eating:
A. fresh water fishes,
B. sea fishes,
C. pork,
D. beef,
26. What is the duration of the life of the adult forms in echinoccosis?
A. It is continued from 6 months till 1 year,
B. It is continued 1 week,
C. It is continued 10 weeks,
D. It is continued more than 10 years,
E. It is continued 7 days,

27. What is epidemiology of echinoccosis?
A. It is transmissive helminthiasis,
B. It is hepatic helminthiasis,
C. It is biohelminthiasis,
D. It is contagious helminthiasis,
E. It is especially dangerous disease

28. What is the possible localization of hydatid cyst?
A. liver,
B. lungs,
C. brain,
D. everything is right,
E. nothing is right

29. What is treatment should be administered in pork tapeworm?
A. vermitin (phenasalum; Niclosamidum),
B. Filicis maris rhizome,
C. mebendazol,
D. decaris (levamysol),
E. chloxil

30. How is the definitive host in case of Taeniarhynhosis?
A. cattle,
B. dog,
C. pig,
D. human,
E. fish

31. What is epidemiology of hymenolepiasis?
A. It is peroral helminthiasis,
B. It is percutaneous helminthiasis,
C. It is contagious helminthiasis,
D. It is transmissive helminthiasis,
E. It is especially dangerous disease

32. What is epidemiology of fascioliasis?
A. It is anthroponosis,
B. It is zoonosis,
C. The definitive host is human and cattle,
D. The intermediate hosts are the pigs,
E. The intermediate hosts are mollusks,

33. What is epidemiology of fascioliasis?
A. It is anthroponosis,
B. It is zoonosis,
C. The definitive host is human and cattle,
D. The intermediate hosts are the pigs,
E. The intermediate hosts are mollusks.

To fulfill the table
The dynamics of the clinical symptoms of Nematodosis

<table>
<thead>
<tr>
<th>The stage of the disease</th>
<th>Acute (migratory) phase</th>
<th>Chronic phase</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Ascaridosis</td>
<td>Ancylostomidos</td>
</tr>
<tr>
<td>General toxic</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Symptoms</td>
<td>Eyes</td>
<td>Brain</td>
</tr>
<tr>
<td>----------------------------------</td>
<td>------</td>
<td>-------</td>
</tr>
<tr>
<td>Allergic</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Increase the temperature</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Arthralgia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Mialgia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Skin rash</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td><em>Lesion of the respiratory tract</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pain in the chest</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Cough with sputum</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Leffler’s syndrome</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Asthmatic bronchitis</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td><em>Lesion of the gastrointestinal tract</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abdominal pain</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Diarrhea</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Leukocytosis</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Marked eosinophilia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Moderate eosinophilia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Anemia</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Hypoproteinemia</td>
<td>+</td>
<td></td>
</tr>
</tbody>
</table>

**To fulfill the table**

The clinical manifestations of cystecercosis in dependence on localization of cystecercs
<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Cerebral hemispheres</th>
<th>IV Ventricle</th>
<th>Basis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Headache</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Dizziness</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Nausea</td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td>+</td>
<td>+</td>
<td></td>
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<tr>
<td>Epileptic attacks</td>
<td>+</td>
<td></td>
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<tr>
<td>Disorders of psychics (dimension, hallucinations)</td>
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<tr>
<td>Dispnoe</td>
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<tr>
<td>The harsh of the state in impairment</td>
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<td>+</td>
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<tr>
<td>the change of the body position</td>
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<tr>
<td>Disorder of vestibular conduction</td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Paresis, paralyses</td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Decrease of the hearing</td>
<td>+</td>
<td></td>
<td></td>
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<tr>
<td>Progressive impairment of the vision</td>
<td>+</td>
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</tr>
</tbody>
</table>

### 3.4.3. Situational tasks (a=2)

**Task 1**

The patient of 42 years old complains on acute headache, tenderness of the muscles of the extremities. The general weakness, fever, edemas around the eyes developed a week ago. The physician diagnosed influenza and prescribed Amixin IC. An improvement didn’t come. His wife also fell ill. She complaints of muscle pain, bad condition. They had eaten fried pork, bought 12 days ago. The temperature is 38,3°C. the face is edematous. The muscles of extremities are painful. The abdomen is soft. Stool is 2 times per day grueled. In the blood count:
Нв-133 г/л, Л-15,0, В-1 %, Е-40 %, У-1 %, В-7 %, С-3 %, Л-8 %, М-6 %, ESR-25 мм /г.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

**Task 2**

The woman of 36 years old is a worker of the fish plant. She came to the polyclinic with complaints of heartburn, unstable stool, weakness. The itching rash periodically developed on the body. On examination: her skin is pale, single elements of urticaric rash are marked. The stomach is soft, slightly painful around the umbilicus. On examination of the blood: hyperchromatic anemia, eosinophilia to 9 % are marked.

1. What is the preliminary diagnosis?
2. What methods are used for diagnostics of the disease?
3. What therapy would you prescribe?

**4. Materials of individual work before the practical class**

**4.1. The list of the study practical skills:**

- make up methods of the examination of the patients with cestodoses or trematodoses
- to perform curation of the patient with cestodoses or trematodoses
- to perform differential diagnostics cestodoses or trematodoses
- to compose the plan of the laboratory investigation
- to interpret the results of the specific investigation
- to determine complications cestodoses or trematodoses
- to compose the plan of the treatment of patient with cestodoses or trematodoses
- make up methods of the examination of the patients with nematodoses
- to perform examination of the patient with nematodoses
• to provide differential diagnostics nematodoses
• to compose the plan of the laboratory investigation
• to interpret the results of the specific investigation
• to determine complications nematodoses
• to compose the plan of the treatment of patient with nematodoses

4.2. Professional algorithm for formation practical skills of the diagnostics of the nematodoses (ascaridosis, trichinellosis, trichicephalosis, enterobiosis (pinworm), ancylostomidoses (hookworms), strongyloidioses).

<table>
<thead>
<tr>
<th>№</th>
<th>Task</th>
<th>Sequence of actions</th>
<th>Notices and warnings concerning self-control</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Possess of the methods of clinical examination of the patient with nematodoses</td>
<td>I. To elucidate complaint of the patient.</td>
<td>To determine complaints of the patient, which are typical for syndromes:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2. To take the history (anamnesis)</td>
<td>- general intoxication</td>
</tr>
<tr>
<td></td>
<td></td>
<td>I. Case history</td>
<td>- allergic manifestations</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- disorders of gastrointestinal tract.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>To pay attention on appearance and dynamics of:</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- fever;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- irritation;</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>- headache;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- disorder of the sleep;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- disorder of the stool;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- rash;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>- pain in the epigastrium area</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>To establish the diseases in the last, anamnesis of vaccination.</td>
</tr>
<tr>
<td>3.</td>
<td>Use into food dirty fruits and vegetables, insufficiency thermal processing pork, beef or fresh water fish, water plants; presence in the endemic areas (opisthorchiasis)</td>
<td></td>
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<tr>
<td>---</td>
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</tr>
<tr>
<td>II. Life history</td>
<td>Remember: presence, dynamics of the symptoms depend from the period of the disease, severity of the course, age of the patient, concomitant pathology</td>
<td></td>
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</tr>
<tr>
<td>1. total blood count.</td>
<td>Pay attention on:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2. urinalyses.</td>
<td>- temperature;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3. investigation of feci</td>
<td>- edema of the face;</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4. biochemical methods</td>
<td>Pay attention on anemia, leukocytosis, eosinophylia.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5. ultrasound investigation</td>
<td>Finding of the eggs or proglottids in feci</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. cholecystography</td>
<td>Dysproteinemia, changes of the biochemical tests (increase of total bilirubin and it’s fractions, ALT, AST and other)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| 7. fibro-esphago-gastro- | }
**Professional algorithm for formation practical skills of the diagnostics cestodoses (diphyllobothriasis, hymenolepiasis, echinoccosis, pork warm disease and cystecercosis, beef worm disease), and trematodoses (fascioliasis, opisthorchiasis).**

<table>
<thead>
<tr>
<th>№</th>
<th>Task</th>
<th>To know</th>
<th>To be able to</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Possess of the methods of clinical examination of the patient with cestodoses and / or trematodoses</td>
<td>1. To elucidate complaints of the patient.</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>- disorders of gastrointestinal tract.</td>
</tr>
<tr>
<td>2</td>
<td>Possess of the methods of clinical examination of the patient with cestodoses and / or trematodoses</td>
<td>2. To take the history (anamnesis)</td>
<td>To pay attention on appearance and dynamics of:</td>
</tr>
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<td></td>
<td></td>
<td>I. Case history</td>
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<td></td>
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<td>- disorder of sleep</td>
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<td></td>
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<td></td>
<td>- disorder of stool</td>
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<tr>
<td></td>
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<td>- rash</td>
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<tr>
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<td>To establish the diseases in the last, anamnesis of vaccination.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Use into food dirty fruits and vegetables, insufficiency thermal processing pork, beef or fresh water fish, water plants; contact with dogs; presence in the endemic areas (opisthorchiasis)</td>
</tr>
</tbody>
</table>
Prescribe laboratory and instrumental investigations, interpret the results of the investigations

<table>
<thead>
<tr>
<th>II. Life history</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. blood count.</td>
</tr>
<tr>
<td>2. urinanalysis</td>
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</tbody>
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Remember: presence, dynamics of the symptoms depend from the period of the disease, severity of the course, age of the patient, concomitant pathology

Pay attention on:
- temperature;

Pay attention on anemia, leukocytosis, eosinophylia.

Finding of the eggs or proglottids in feci

Dysproteiemia, changes of the biochemical test (increase of total bilirubin and it’s fractures, ALT, AST and other)


ВИТЯГ
з протоколу № 5
засідання кафедри інфекційних хвороб
Запорізького державного медичного університету
від 25 грудня 2013р.

ПОРЯДОК ДЕННИЙ

Про затвердження навчально-методичних матеріалів кафедри

СЛУХАЛИ:

Проф. Рябоконь О.В. доповіла про навчально-методичний посібник підготовлений співробітниками кафедри для самостійної підготовки англомовних студентів 5 курсу: «Introduction to infectology. Infections diseases with fecal-oral transmission» (Manual for practical training and independent work of students for the 5th year of the Medical Faculty on Infectious Diseases to the module №1. (автори: Рябоконь О. В., Оніщенко Т.Є., Фурик О.О., Машко О.П. ), обґрунтувала доцільність його використання у навчальному процесі.

УХВАЛИЛИ:

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Зав.каф. інфекційних хвороб
професор
О.В.Рябоконь

Секретар
Ас., к.мед.н.
О.П.Машко
ВИТЯГ з протоколу № 5
засідання циклової методичної комісії
з терапевтичних дисциплін
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професор С.Я. Доценко