MINISTRY OF PUBLIC HEALTH SERVICE
ZAPOROZHYAN STATE MEDICAL UNIVERSITY

Department of infectious diseases

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INFECTION DISEASES OF WOUND INFECTION AND MULTIPLE MECHANISM TRANSMISSION

Module 5

Manual for practical training and independent work

For students of the 5th year of medical faculty

Specialty *222*«General Medicine»

Master's level of training

2020

UDC 616.94(075.8)
R98

Ratified on meeting of the Central methodical committee

of Zaporizhzhia State Medical University

(protocol N 3from 27.02.2020)

and it is recommended for the use in educational process for foreign students.

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DISEASES OF WOUND INFECTION AND MULTIPLE MECHANISM TRANSMISSION (Module 5): Manual for practical training and independent work for students of the 5th year of medical faculty Specialty *222«*General Medicine» Master's level of training / Ryabokon E.V., Onishchenko T.E., Furyk E.A., Zadiraka D.A., Kalashnik K.V. –Zaporizhzhia: [ZSMU], 2020. – 151c.

According to the curriculum for the course "Infectious Diseases" (2020), Master's level of training, field of knowledge 22 "Health care", Specialty 222 "General Medicine" Designed for students of the 5th year of medical faculty of ZSMU.

UDC 616.94 (075.8)

CONTENTS

INTRODUCTION

Infection diseases of wound infection and multiple mechanism of transmission are the most common in the structure of all infectious diseases. There are a number of infectious diseases with multiple transmission mechanism. Infection in these diseases depends on different factors: type of employment, conditions and place of residence, diet, and other factors. In some infectious diseases, the mechanism of transmission determines the clinical form, course and outcome of the disease. Such diseases are described in this manual.

Manual contains materials of theoretical and practical character necessary to prepare students for the successful development of infectious diseases with multiple mechanisms of transmission. Manual includes a brief synopsis of the theoretical material, questions for self, homework, tests, tasks, algorithms of diagnostics of infectious diseases with multiple transmission mechanism. This manual presents modern data on the etiology, epidemiology, pathogenesis, clinical manifestations, diagnosis and treatment of infection diseases. The manual provides students with the opportunity to study independently develop knowledge and get some practical skills.

LEPTOSPIROSIS

1.URGENCY OF THE ISSUE:

Epidemiologia

Leptospirosis is a geographically widespread zoonotic disease caused by pathogenic spirochetes of the genus Leptospira. A variety of wild and domestic mammals, particularly rodents, cattle, pigs, sheep, horses and goats are the natural hosts. Humans are infected incidentally following exposure to animal urine, contaminated water or soil, or infected animal parts, and, rarely, through ingestion of infected material. Exposure can result from contact with contaminated floodwater (particularly in urban rodent-infested slums), occupational exposure (farmers, abattoir and tannery workers, soldiers, sewerage plant workers, pet traders and laboratory workers), and increasingly through exposure during recreational activities such as triathlons. Leptospirosis is an under-reported disease, and its true global incidence is unknown, though it is thought to be approximately 10 times more common in the tropics than in temperate regions.1 Modeling conducted by the WHO estimated in 2013 that there are 873 000 cases worldwide annually, with 48 600 deaths.

In most settings members of the rodent family are the most important reservoirs for maintaining transmission. Infection usually occurs during infancy, and infected animals may shed the organism in their urine intermittently or continuously throughout life, resulting in contamination of the environment, particularly water. Most animal infection is asymptomatic, but clinical disease also occurs and may be fatal.

Leptospirosis in humans is characterized by an acute febrile illness followed by mild self-limiting sequelae or an even more severe, and often fatal, multiorgan involvement. The disease was first described by Larrey in 1812 of fever jaune among Napoleon ’s troops at the siege of Cairo. It was initially believed to be related to the plague but not as contagious. Throughout the remainder of the 19th century, the illness was known in Europe as bilious typhoid.

Pathogenesis and Pathology

Leptospires are highly motile, obligate aerobic spirochetal bacteria, about 0.25 * 6–25 μm in size. They can survive for days or weeks in warm, damp, slightly alkaline conditions, especially in still or slowly moving fresh water in the temperate summer, and in damp soil and water in the tropics, particularly in the rainy season.

Leptospires belong to the order Spirochaetales and the family Leptospiraceae. Traditionally, the organisms are classified based on antigenic differences in the lipopolysaccharide envelopes that surround the cell wall.On the basis of DNA–DNA hybridization studies the genus Leptospira is recognized as containing 21 species. Nine of these are regarded as pathogenic (Leptospira interrogans, L. kirschneri, L. noguchii, L. alexanderi, L. weilii, L. alstonii, L. borgpetersenii, L. santarosai and L. kmetyi), 5 are of intermediate or unclear pathogenicity (L. inadai, L. fainei, L. broomii, L. licerasiae and L. wolffii), and the remaining seven are nonpathogenic free-living saprophytic species that do not infect animal hosts (L. biflexa, L. meyeri, L. wolbachii, L. vanthielii, L. terpstrae, L. yanagawae and L. idonii). An older parallel classification system based on serology identifies more than 200 pathogenic and 60 non-pathogenic saprophytic serovars of Leptospira. Some serovars are strongly associated with a particular animal host, such as serovar Hardjo and cattle, and as some serovars are found in more than one species of Leptospira, by convention isolates are identified by both species and serovar. Leptospirosis is a systemic infection with leptospires gaining access to the circulation through abraded skin, or via intact mucous membranes, including the oral cavity and conjunctivae. Transplacental infection can also occur, resulting in fetal death or neonatal infection. Leptospires make their way into the bloodstream without producing any focal lesion at the inoculation site, leading to a bacteremia of as high as 106/mL of blood (similar to that seen in the spirochetemia of relapsing fever), and subsequent hematogenous spread to target organs including the liver, lung and kidney. Histopathological findings include evidence of a systemic vasculitis with endothelial cell injury, with damaged endothelial cells showing varying degrees of swelling, denudation and necrosis. Leptospires are seen in large- and medium-sized blood vessels and in the capillaries and interstitial spaces of various organs. The major affected organs are:

- the kidneys, with a diffuse acute tubular necrosis and interstitial nephritis;
- the lungs, usually congested, with focal or massive hemorrhage occurring in both the alveolar septa and intra-alveolar spaces; and
- the liver, which shows cholestasis associated with mild degenerative changes in hepatocytes and leptospiral infiltration of Disse's space and invasion of the perijunctional region between hepatocytes.

Other systems may also be affected, with myocarditis, meningoencephalitis and uveitis all occurring in severe disease. The pathological consequences of infection are probably mediated by a combination of a direct toxic effect of the leptospires and the resulting immune response. Pathogenic leptospires adhere directly to host cells and to the extracellular matrix, and pathogen-associated molecular patterns (PAMPs) including Leptospira outer membrane proteins (OMPs) and lipopolysaccharides activate the innate immune response through Toll-like receptor (TLR)2-dependent and TLR4-dependent pathways. In severe leptospirosis levels of pro-inflammatory cytokines are very high, and there is evidence of vigorous inflammasome activation leading to tissue damage, particularly in the kidneys and lungs. Thrombocytopenia and abnormalities of the coagulation system are also commonly seen. During recovery, leptospires continue to be excreted in the urine for some days. Recent whole genome sequencing of both pathogenic and nonpathogenic Leptospira species and serovars have identified a series of genes possibly related to adhesion, invasion and the hematological changes that characterize leptospirosis, allowing in-depth studies of virulence and pathogenesis.**Signs and symptoms.**

A good clinical history is often the key to accurate diagnosis in leptospirosis. Important features include a plausible exposure history and a clinical picture consistent with the disease.

Expert consensus is that leptospirosis occurs as two recognizable clinical syndromes. A third syndrome of asymptomatic infection is more controversial. Anicteric leptospirosis is a self-limited disease similar to a mild flulike illness. Icteric leptospirosis is a severe illness characterized by multiorgan involvement or even failure.

Subsequent sequelae depend on the serovar involved and the health, nutritional status, and age of the patient, as well as the rapidity of definitive and supportive treatment.

An acute illness follows any infection with any serovar of leptospirosis. Most of the following symptoms develop in varying degrees: high temperature (38- 40 °C), rigors, sudden headache, nausea and vomiting, anorexia, diarrhea, cough, pharyngitis, nonpruritic skin rash, and muscle pain. Muscle pains are typically localized to the calf and lumbar areas. This phase of illness lasts 5-7 days and either regresses to a relatively asymptomatic period or progresses to a more severe illness. In anicteric leptospirosis, the acute illness is followed by 1-3 days without fever and then progresses to 4-30 days of the immune (delayed) phase of the illness.

The physical examination findings differ depending on the severity of disease and the time from onset of symptoms. Patients may appear mildly ill or toxic. Early in the disease, temperatures as high as 40 °C and tachycardia are common. Hypotension, oliguria, and abnormal chest auscultation at presentation may portend severe illness. When fever is severe and prolonged, hypotension and shock due to volume depletion may also occur. The fever typically subsides within 7 days.

Early in the disease, the skin is warm and flushed. Additional skin findings include a transient petechial eruption that can involve the palate. Later in severe disease, jaundice and purpura can develop. The classic ocular finding of conjunctival suffusion occurs early irrespective of disease severity. Conjunctival suffusion is characterized by redness of the conjunctiva that resembles conjunctivitis but that does not involve inflammatory exudates.

Uveitis is a common feature following acute leptospirosis; however, patients who receive antibiotics during the acute phase of illness may develop only mild uveitis.

Muscle tenderness can occur with the myositis of early infection. This can be particularly prominent in the paraspinal and calf muscles but can involve any muscle. Neurologic examination can reveal signs of meningitis, including neck stiffness and rigidity and photophobia. Early in the disease, the stiffness on neck examination can be confused as muscular in origin; however, this symptom may actually represent early meningismus.

Lung examination results may be normal in early or mild illness. In severe illness, signs of consolidation due to alveolar hemorrhage may be found. In patients with cardiac-related pulmonary edema, rales and wheezes can be heard.

The incidence of pulmonary involvement has increased over the past few years, affecting up to 70% patients. Alveolar hemorrhage that manifests as dyspnea and hemoptysis is the main pulmonary manifestation. This clinical manifestation may be severe and can occur in the absence of typical presentations of Weil disease.

Pulmonary involvement has emerged as a serious cause of mortality, becoming the main cause of leptospirosis-associated death in some countries.

Myocarditis may occur in severe disease. All of the physical findings of biventricular heart failure can be found, including elevated jugular venous pulsations; a new S3 gallop; and dysrhythmias, including atrial fibrillation, heart blocks of varying severity, and ventricular ectopy.

Abdominal examination may reveal liver enlargement and tenderness due to hepatitis. Acalculosus cholecystitis, which may be suggested by a positive Murphy sign, is a finding of profound systemic illness. Pancreatitis has also been described in severe cases.

Heme-positive stool and even gross blood can be found on rectal examination in patients with D1C and bleeding.

In severe disease, delirium may develop either as a consequence of shock or independent of it. Delirium may be an early finding in severe disease. Late in disease and into convalescence, prolonged mental symptoms may persist, including depression, anxiety, irritability, psychosis, and even dementia.**Diagnostic**

Leptospirosis may be difficult to distinguish from other infectious causes of fever, and a high index of suspicion is required based on the local epidemiology. Clinical and laboratory features may be nonspecific, and specific laboratory tests either take several weeks or are unavailable. Depending on the setting, the differential diagnosis includes malaria, dengue, scrub typhus and other rickettsial illnesses, ehrlichiosis and acute viral infections such as influenza. Conjunctival suffusion is rare in these conditions and is

therefore one of the few diagnostically useful clinical signs. Hantavirus infection can mimic severe leptospirosis, causing hepatorenal syndrome and pulmonary hemorrhage.

Routine blood tests are usually nonspecific, with hyponatremia, mild to moderately increased transaminases, mildly raised white blood cell count, and thrombocytopenia all common. An elevated creatine kinase may be suggestive of leptospirosis, occurring in around 50% of patients. Urinalysis often shows proteinuria, pyuria and granular casts, and occasionally microscopic hematuria.

There is no adequate gold standard test for the diagnosis of leptospirosis, with the microagglutination test (MAT) and bacterial culture both imperfect in terms of sensitivity, even when combined. The MAT is the most commonly used diagnostic test, and when applied to paired acute and convalescent samples is considered the reference standard; but it is complex to perform, serovar-dependent and restricted to reference centers. Recently developed serology-based rapid diagnostic tests perform variably in the field, particularly in endemic areas. PCR-based molecular tests show considerable promise for rapid, accurate diagnosis but are not yet widely available. Pathogenic Leptospira spp. can be grown in vitro from clinical specimens including blood, urine and cerebrospinal fluid (CSF), though special media are required. Growth is usually observed in 1–2 weeks, but may take up to 3 months. A method of growing leptospires on solid agar (LVW media) has been developed recently, which facilitates more rapid growth, isolation of single colonies, and simplified antimicrobial sensitivity testing.

In summary, timely diagnosis of leptospirosis remains problematic, and the emphasis should be on early empirical treatment of clinically suspected cases.

Complications

Complications include meningitis, extreme fatigue, respiratory distress, and renal interstitial tubular necrosis, which results in renal failure and often liver failure (the severe form of this disease is known as Weil’s disease, though it is sometimes named Weil Syndrome). Cardiovascular problems are also possible. Approximately 5-50% of severe leptospirosis cases are fatal; however, such cases only constitute about 10% of all registered incidents.**Treatment**

Most cases of leptospirosis are self-limiting, and whether antimicrobials have a beneficial effect in mild disease has been the subject of controversy. However, the evidence increasingly supports the antimicrobial treatment of all patients suspected of having leptospirosis, both to reduce the duration of illness and to prevent progression to severe disease.

For mild leptospirosis, doxycycline (200 mg initially then 100 mg twice a day for 7 days) and azithromycin (2 g then 1 g once a day for 2 further days) have been shown to be equally effective, though the latter was better tolerated. Both have the advantage of being effective against scrub typhus and other rickettsial diseases and therefore useful as empirical treatment in areas where both leptospirosis and rickettsioses are common causes of undifferentiated fever. For children under 8 years old and pregnant women azithromycin (or amoxicillin) is preferable and doxycycline should not be used.

For suspected severe leptospirosis 7 days' treatment with parenteral penicillin (1.5 million units four times a day), ceftriaxone (1 g once a day), cefotaxime (1 g four times a day) or doxycycline (200 mg initially then 100 mg twice a day) have all been shown to be equally effective.

In severe cases, supportive care with renal replacement therapy (hemofiltration, hemodiafiltration or hemodialysis), ventilatory support and transfusion of blood products may also be required. Given the vasculitic nature of the disease adjunctive therapy with corticosteroids has been suggested, but there is currently insufficient evidence to recommend this routinely.

Prevention

Prevention measures are based on local knowledge of the epidemiology of the disease, and include avoidance of potential sources of infection such as animal farm water runoff and stagnant water, control of rodent populations, animal vaccination and antimicrobial prophylaxis with doxycycline 200 mg weekly for individuals at high risk of exposure. No human vaccine is currently available.

Viral Hemorrhagic Fevers

Introduction

The term viral hemorrhagic fever (VHF) is used to describe a group of viral infections in which a subset of patients develop a clinical syndrome of severe febrile illness with hemorrhagic signs. VHFs are of public health importance due to the high case fatality rate of some VHFs and the possibility of human-to-human transmission. VHFs are difficult to diagnose and treat. The history of VHFs is characterized by some notable outbreaks. In particular, the 2014–15 Ebola virus out-break in West Africa, which resulted in loss of 1000s of lives and severely damaged the healthcare systems and economic development of the affected ountreis. This catastrophic outbreak has refocussed attention on the need for global leadership and coordination of out-break preparedness and response. This chapter will aim to provide a clinically relevant overview of the most significant viruses (in terms of mortality and the risk of human-to-human transmission). It will discuss general measures for clinical management and public health containment that should be useful for known viruses, and adaptable to future novel VHF pathogens.

Virology

All known VHFs are caused by single-stranded enveloped RNA viruses, including arenaviruses, bunyaviruses, filoviruses and Flaviviruses. There is no precise definition with which to classify VHFs, and dengue fever and henipavirus infections (Nipah and Hendra virus) are sometimes categorized as VHFs, although bleeding is rare in these infections.

Arenaviruses are classified into two categories: Old World and New World, based on geographic location in Africa and the Americas, respectively. Lujo and Lassa virus are the two Old World arenaviruses known to cause human disease. Of the New World arenaviruses, Junin virus, Machupo virus, Guanarito virus and Sabia virus are the etiologic agents of Argentine HF, Bolivian HF, Venezuelan HF and Brazilian HF, respectively.

In the Bunyaviridae family, Crimean–Congo hemorrhagic fever virus (CCHF), hantaviruses – particularly Andes virus, Hantaan virus and Sin Nombre virus – and Rift Valley fever virus cause VHFs in humans. Diseases caused by hantaviruses generally manifest as either hemorrhagic fever with renal syndrome (HFRS) or hantavirus pulmonary syndrome (HPS). SFTS virus (genus Phlebovirus, family Bunyaviridae) is one

of the most recently discovered pathogens. It causes severe fever with thrombocytopenia syndrome (SFTS) in East Asia.

The Ebolavirus genus is comprised of four distinct species responsible for human disease (Zaire ebolavirus, Sudan ebolavirus, Bundibugyo ebolavirus and Tai Forest ebolavirus), each having different mortality rates. Marburg virus is a single species (Marburg marburgvirus).

Flaviviridae that cause HF include yellow fever virus and dengue virus, both of which will not be discussed in depth here. Other Flaviviruses can cause hemorrhagic fever, including Kyasanur Forest disease virus, Omsk hemorrhagic fever virus and Alkhumra virus, but transmission of these viruses is geographically restricted and they are not discussed further. **Epidemiology**

With the exception of dengue fever, humans are not the natural host for VHFs. Because the viruses rely on animal reservoirs for maintenance and transmission, index infections are geographically restricted to areas where the reservoir and vector reside. Consequently VHFs occur primarily in rural areas where there is substantial contact with rodents, ticks, bush meat, bats or mosquitoes. However, the 2014–15 Ebola outbreak in West Africa demonstrated how easily a VHF can spread into urban areas. Additionally, with increasing international travel, and in particular the return of international healthcare workers to their home countries, cases may present anywhere in the world. These maps are useful for targeting surveillance and preparedness activities but their accuracy is dependent on the current state of knowledge and the availability of data. As such the maps are guides alone and cannot be assumed to be definitive.

Some VHFs are endemic (such as HFRS in China) or have predictable seasonal epidemiology (such as CCHF in Turkey). However, VHF epidemiology is notable for epidemics, ranging from small, wellcontained outbreaks to complex humanitarian emergencies with thousands of fatalities. For various reasons, the overall incidence of VHFs is likely under-reported. The few seroprevalence studies that have been undertaken demonstrate a hidden burden of subclinical or mild infections or a broader geographical area of transmission. In addition, many isolated VHF cases likely go undiagnosed or unreported to public health authorities, particularly when they occur in an unusual region

or present as mild, self-limiting disease. Furthermore, during epidemics, healthcare systems may become overwhelmed and case confirmation and reporting may be inadequate.

For viruses that demonstrate human-to-human transmission, infection can occur through exposure of mucosal membranes or skin breaks to infectious body fluids via mucosal membranes or skin breaks. Sexual transmission occurs for Ebola. Notably, semen (along with other immune privileged fluids such as aqueous humour and CSF) can continue to harbour Ebolavirus following resolution of viraemia and sympoms. Sexual transmission probably also occuse for CCHF, LAssd, Marbury and Junin. There are reports of suspected airborne transmission of Lassa fever during an epidemic in a Nigerian hospital in 1970, and this is not thought to be a likely route of transmission. Viral persistence on surfaces changes with arterial and environmental conditions the risk of transmission via fomites consequently is unclear and varies.1 Nosocomial transmissions of CCHF and SFTS possibly associated with aerosol-inducing procedures have been reported. The risk of nosocomial infection and disease amplification in healthcare settings is especially important for Filoviridae, as exemplified by the high number of healthcare workers (HCWs) infected during the 2014–15 Ebola epidemic.

Pathogenesis

Following viral exposure, most initial viral replication occurs mostly in local tissues, monocytes, macrophages and/or dendritic cells, before migration to the lymphoreticular system. The viruses causing VHFs have broad tissue tropism and subsequent dissemination occurs to the lymphatic system, blood (monocytes and macrophages) and a variety of solid organ targets, depending on the virus. The liver and spleen are commonly affected. High viral loads are common whilst the immune response is often poor, and may be worsened by viral-induced immunosuppression. High viral loads are predictive of mortality, for some VHFs, such as Ebola and CCHF.

Knowledge of the pathophysiology of VHF in humans is incomplete due to the geographic isolation of most outbreaks, reliance on historical data and the difficulty in safely collecting and managing biologic samples. While the pathogenesis differs between

viruses, and the host–virus relationship is complex, there are common characteristics that contribute to the manifestations of shock and multiple organ failure.

1. Excessive microvascular permeability occurs due to direct endothelial cell injury by the virus or via mediators such as proinflammatory cytokines/chemokines.
2. Thrombocytopenia is common, as is inhibition of platelet function, leading to impaired platelet aggregation.
3. To a varying extent for each of the VHFs, coagulopathy may be due to alterations to the fibrinolytic system, or consumption or activation of coagulation factors. Together, these deficits may contribute to disseminated intravascular coagulopathy.

Volume depletion from vomiting and diarrhea and ‘third spacing’ due to vascular leakage frequently results in hypovoleamic shock in EVD patients. A substantial proportion of EVD patients in the 2014–15 outbreak had evidence of acute kidney injury on admission., although the mechanism for this may be partially independent of pre-renal failure caused by hypovolaemia.**Diagnosis and Clinical Features**

The range of severity of VHFs, and the wide differential diagnosis for many of the symptoms, makes diagnosis difcult. Vigilance for clusters of cases (particularly if the cluster involves an HCW or exposure to a known VHF risk factor) may lead to early identification of a potential outbreak. The diagnosis of a VHF is based on three components: (a) a history of exposure, (b) clinical suspicion and (c) laboratory findings.

- A thorough case history must include the following information for the duration of the incubation period:
- All recent travel, particularly to endemic areas.
- Membership of an occupational risk group (e.g. HCW, veterinarian, abattoir worker).
- Contact with known or suspected cases of VHF (dead or alive), or severely ill febrile persons.
- Contact with deceased persons, who may have been infected with a VHF (usually during funeral practices).
- Contact with body fluids from a possible case of VHF or cadaver (e.g. laboratory workers).

- Contact with items contaminated by body fluids (bed sheets, medical instruments).
- Being breast-fed by a woman with a possible VHF.
- Sexual contact with a possible VHF case (including during convalescence).
- Contact with potential vectors.
- Contact with animals, animal droppings or animal products (cooking or consumption of bush meat, contact with bat drop-pings, etc.).
- Any entry into environments where a reservoir may have been present (e.g. caves housing bat populations or houses with rodent infestations).

The clinical manifestations of VHFs are heterogeneous. Importantly, although hemorrhage is eponymous, it is usually present in a minority of cases. After an incubation period of variable duration, there is usually a prodrome of a generalized febrile illness. Symptoms may include malaise, prostration, myalgia, headache and pharyngitis. At this early stage, the infection may resemble many other endemic diseases, and there is a broad differential diagnosis. Clinical experience from the 2014–15 West African Ebola epidemic has provided useful experience of the range of clinical symptomatology and also the differential diagnosis. It is important to note that not all patients are febrile on contact with the heatlhcare system.

As the disease progresses, specific organ involvement becomes more evident and pathognomonic features narrow the differential diagnosis. Gastrointestinal symptoms (commonly nausea, vomiting and diarrhea) can be severe and lead to significant volume depletion. Conjunctival injection, petechial rash, or hemorrhage is common. The likelihood of progression to life-threatening disease varies between viruses. When present, hemorrhagic symptoms include petechial rash, purpura, haematocchezia, melena, uterine or vaginal bleeding, oozing from intravenous sites, or epistaxis. These may be complicated by coagulopathy, notably, disseminated intravascular coagulopathy (DIC). Endothelium disruption manifests as pulmonary edema and peripheral edema. Neurologic disturbances include encephalopathy, seizures and coma. Hypovolemia and electrolyte disturbances may be common, and renal failure is predominant in some diseases (e.g. HFRS). Death may be due to septic shock or hypovolemic shock. It is suspected that the

sudden death of some patients may be due to cardiac arrhythmias caused by electrolyte imbalances.

For those who survive, weakness, fatigue and prostration are common in the post-infectious phase. Inflammatory complications have been reported and include uveitis, arthropathy, epidydimoorchitis, hepatitis, pericarditis, meningitis, and transverse myelitis. There are a number of follow-up activities for the West African Ebola survivors which should further define these complications and, therefore, help manage them. For Ebola, localised disease, such as uveitis and meningitis, may arise dring convalescense as a result of persistence of the virus. Sensironeural deafness is a not uncommon complication for Lassa fever, and may be permanent. Stigmatization of persons returning to their communities may be severe and contribute to the psychological complications already experienced by this cohort.**Investigation findings**

Great care must be taken in collecting, handling and transporting specimens. Potentially infectious specimens must be labeled as hazardous. Additional testing of patient samples should be minimized. Laboratories should be notified in advance that samples from a patient with suspected VHF will be transported to them. The samples must be packaged, labeled and transported according to national and international (i.e. IATA) guidance on the transport of infectious substances. Testing should be undertaken only by reference laboratories with adequate bio-containment facilities. Filoviruses, CCHF and most arenaviruses require BSL-4 handling. During an outbreak field laboratories capable of handling these dangerous pathogens can be established.

Laboratory diagnosis is usually based on blood or serum samples. Test methods include: RT-PCR, virus-specific IgM or IgG testing, detection of viral antigens using ELISA, virus isolation, visualizing using electron microscopy, or demonstrating a fourfold rise in antibody titre. Point-of-care rapid diagnostic tests for some VHFs are likely to be available in the near future.21 Diagnosis may be possible from other body uids, or from post-mortem tissue samples under some circumstances.

As safely acquiring and testing laboratory samples safely is difficult during an epidemic, the characteristic laboratory results for all viruses have not been fully

elucidated. Hemoglobin may fall with hemorrhage, although hematocrit may rise due to hemoconcentration. Peripheral white blood cell counts are often low early in disease, but may increase later. Thrombocytopenia is common for some conditions and platelet function is often impaired, even in the presence of low-normal platelet counts. Coagulation studies may demonstrate DIC.

Liver function is often abnormal. Aspartate aminotransferase (AST) is usually raised and virtually all VHFs distinguish themselves from viral hepatitis by the disproportionately high AST levels compared with alanine aminotransferase (ALT). Patients are rarely jaundiced (except in yellow fever) and the bilirubin is usually normal. Electrolyte disturbances commonly include sodium and potassium abnormalities. Renal failure is especially common in hantavirus infection in the Eurasia region (HFRS) and Ebola.**Treatment**

For many of the VHFs, virus- or host-directed specific treatments are not available. However good supportive care likely improves outcomes and should be universally implemented. The extent of clinical intervention available also differs between high- and low-income countries. Here, we focus on clinical management in low-income countries.

Healthcare workers must wear suitable personal protective equipment. Treatment of a patient as a suspected case of VHF should not be delayed to rule out other diagnoses, or while waiting for laboratory confirmation. A general approach to the clinical management of a suspected case (with presumed human-to-human transmission risk) is shown in Table 1.1. Due to an evolving evidence base, management of the patient beyond the provision of symptom-based interventions, Fluid replacement and electrolyte management must often be made on a case-by-case basis.

Table 1.1

Contain suspected and confirmed cases	Notify alert, cooperative patients of the requirement and process of isolation Immediately isolate the suspected case Implement appropriate infection control measures

	Urgently notify the appropriate public health authorities, such as the district health officer If safe to do so, take blood samples and notify receiving laboratory of clinical suspicion Perform malaria rapid diagnostic testing of all patients in endemic regions
Provide supportive management to all patients	Treat for other co-morbidities Treat empirically for suspected coinfections with broad spectrum antibiotics Institute regular observation of vital signs (pulse, respiratory rate, blood pressure) Manage fever with paracetamol Provide supplemental oxygen for hypoxia Provide oral rehydration solution in all patients and iv fluid resuscitation for dehydration or hypovolemia Monitor and correct electrolyte imbalances Monitor and correct hypoglycemia .Provide simple analgesia and opioid analgesia where required .Provide psychological support for anxiety .Provide environmental management for confusion and use sedation if required .Use antiemetics for gastrointestinal symptoms .Manage shock according to local practice or WHO guidelines for septic shock .Administer blood, platelets or FFP if required, and available .Manage seizures
Consider further treatment options following confirmed	1. Consider anti-viral if available. 2. Consider other clinical trial agents 3. Consider convalescent blood/plasma

diagnosis, ideally in the setting of a clinical trial	
Provide additional intensive care interventions when they can be done safely	Consider ICU admission, when able. If indicated: 1. Provide mechanical ventilation 2. Administer vasopressors 3. Consider dialysis

Pregnant women with a VHF have a high incidence of miscarriage, intrauterine fetal death and pregnancy-related hemorrhage. Pregnant women may present in an atypical manner, including with afebrile illness. Mortality is high in this population for both the mother and the child.

For patients responding poorly to supportive care, there are several additional treatment options to consider. The antiviral drug ribavirin is indicated for the treatment of Lassa fever, 25 and HFRS due to hantavirus infection.26 While several small studies have been undertaken, there remains no strong evidence for the use of ribavirin in treating CCHF or SFTS. However, when available, it is often used for treatment of patients. It should not be used in treating filovirus infections. When used, the drug should be given early in the clinical course and in intravenous form. Anemia is a common complication of ribavirin treatment, and regular hemoglobin monitoring should be performed. Use of immune plasma is a beneficial treatment for Argentine Hemorrhagic Fever, but there is insufficient evidence to support its routine use for other VHFs.

The following treatment strategies have insufficient clinical trial data in humans and should be used only as part of a clinical trial. Intravenous immunoglobulin has been used in human trials for CCHF, with no mortality benefit shown. Interferon’s have been used in animal model studies of arenavirus and filovirus infection but data on use in humans is not available to date. During the ongoing Ebola epidemic in West Africa, the WHO prioritized use of different experimental vaccines and therapeutics (including monoclonal antibodies, polymerase inhibitors, small interfering RNAs, and convalescent plasma), and

vaccines. While these efforts are ongoing at the time of writing, a coordinated approach to clinical research to improve the evidence base is a necessary component of the response to any VHF outbreak. For the future preparedness should continue around how clinical research can be better integrated rapidly into public health responses.

Prevention

Prevention of VHF index cases relies largely on avoidance of the reservoir species or vector, although this is difficult for viruses where the transmission cycle is incompletely understood. Vaccines exist for Argentine HF and HFRS in some countries in Asia, although these are not widely available. An experimental Vesicular Stomatitis Virus-Ebola Virus Vaccine (VSV-EBOV) has demonstrated excellent efficacy and adequate safety in an interim analysis of a ring vaccination cluster-randomised study.

Prevention of secondary cases relies on a rapid, coordinated public health response, consisting of four elements – case detection, case isolation, contact tracing and safe burial. Screening for suspected VHF should be undertaken on entry to the healthcare system. Patients with suspected or confirmed VHF should be isolated, ideally in a dedicated isolation room with separate amenities, although during outbreaks use of shared facilities may be necessary. Healthcare workers designated to treating these patients require personal protective equipment, and training for its correct use. Equipment should be assigned to these patients exclusively and non-consumables decontaminated after use. Handling of deceased persons should be undertaken by trained staff and restricted to that necessary for safe and dignified disposal of the body. As the virus concentration is likely to be high at the time of death, the importance of infection control when handling a deceased patient cannot be understated. Healthcare workers who have experienced unprotected exposure to body fluids should immediately stop clinical tasks, decontaminate the area and notify the designated team physician for evaluation (as post-exposure prophylaxis and management differs between viruses). For Ebola, viable virus may persist in immune privileged body fluids (semen, aqueous humor) for a number of months following acute infection. Clinicians undertaking invasive procedures on people surviving Ebola should manage infection control according to the most current evidence.

Broader public health management relies on early notification of potential cases and activation of early warning systems to generate prompt intervention. Subsequent contact tracing of direct contacts (in viruses with human-to-human transmission) assists in controlling onward transmission.

Community perceptions of the meaning of the disease and mechanism of spread influence how an outbreak can be controlled. Cultural practices regarding social greetings and contact, care of unwell family members and burial practices can facilitate person-to-person transmission. At a time of extraordinary personal and social upheaval, community fears can be further exacerbated by an influx of foreign medical staff. Consequently, local leadership is a critical asset in control.

2. TASKS OF THE TRAINING COURSE (INDICATING EXPECTED LEVEL OF LEARNING):

2.1 The student should know:

- Rules of hospitalization of patients with leptospirosis and epidemic hemorrhagic fever;
- Causation of leptospirosis and epidemic hemorrhagic fever; disease-evoking power of germs;
- Epidemiology of leptospirosis and epidemic hemorrhagic fever;
- The main chains of pathogenesis;
- Role of allergic and lymphocytic processes at leptospirosis;
- clinical signs of leptospirosis and epidemic hemorrhagic fever in standard progress;
- clinical and epidemiological aspects of diseases progress;
- pathogenesis, period of development and clinical signs of complications of leptospirosis and epidemic hemorrhagic;
- specific and non-specific laboratory diagnostics of leptospirosis and epidemic hemorrhagic fever;
- treatment methods of leptospirosis and epidemic hemorrhagic fever;
- rules of prophylactics;

- rules of patients' behavior in case of emergencies at leptospirosis and epidemic hemorrhagic fever;
- seroprognosis;
- rules of discharge for patients recovered from leptospirosis and epidemic hemorrhagic fever from inpatient hospital;
- rules of periodic health examination of recovered patients.

2.2. The student should be able to: a-3

- Follow the main rules of behavior by sickbed at leptospirosis and epidemic hemorrhagic fever;
- Make up medical history estimating epidemiological data;
- Examine the patient and find out the main symptoms and syndromes of leptospirosis and epidemic hemorrhagic fever, justify the clinical diagnosis, and solve the issue of necessary inpatient treatment
- Perform differential diagnostics of leptospirosis and epidemic hemorrhagic fever;
- Based on clinical examination define possible complications of leptospirosis and epidemic hemorrhagic fever;
- Fill in medical documentation based on previously stated diagnosis " leptospirosis" and " epidemic hemorrhagic fever" (emergency call to regional epidemiological department);
- Make up a plan of patient's laboratory and instrumental examination;
- Analyze the results of laboratory examination;
- Analyze the results of specific methods of diagnostics based on material and period of the disease;
- Make up an individual treatment plan taking into account epidemiological data, stage of disease, available complications, severity of the condition, allergic anamnesis, comorbidity, provide rescue emergency care at pre-hospital stage;

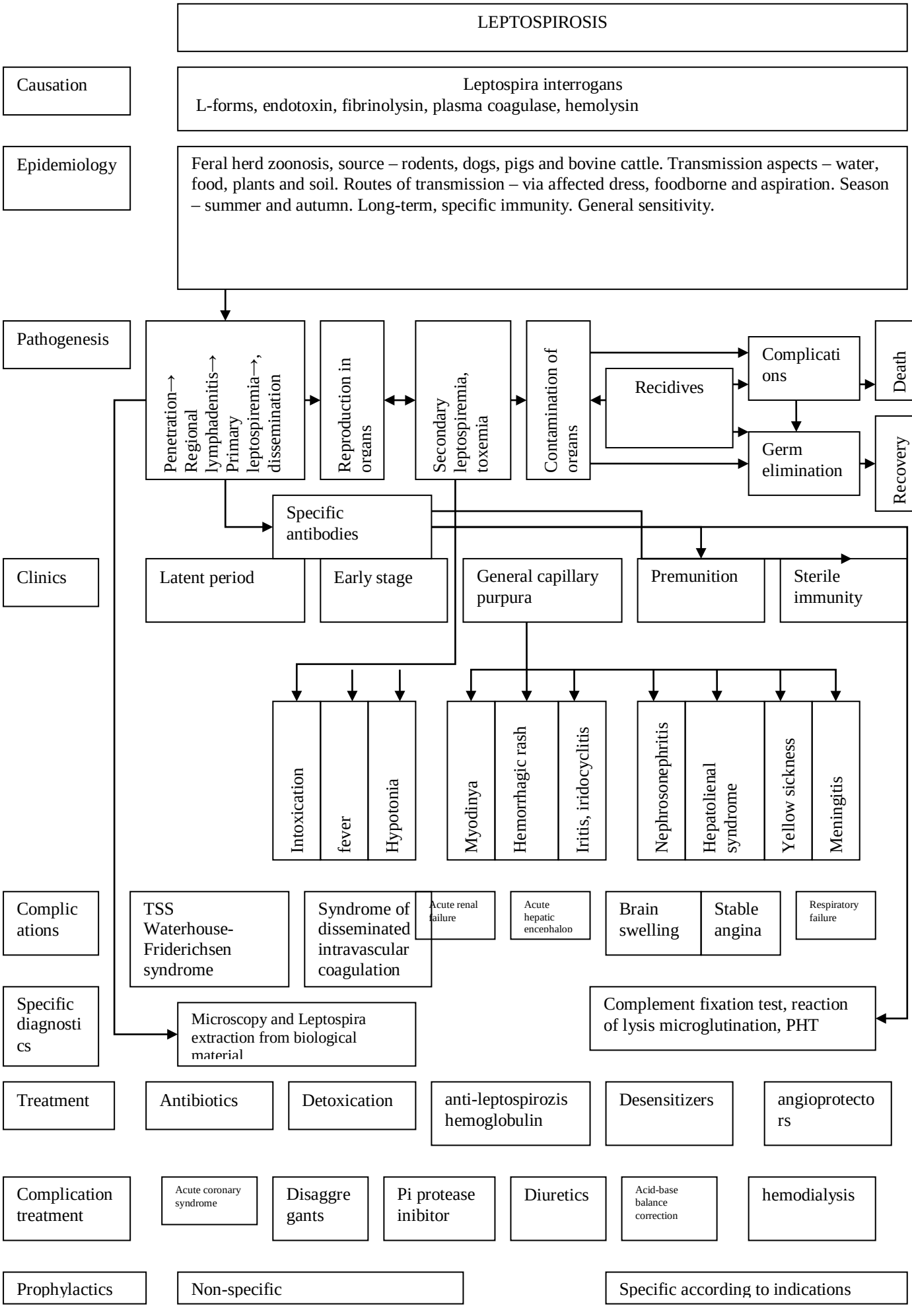
- Make up antiepidemic and preventive measures plan for the centre of leptospirosis and epidemic hemorrhagic fever;
- Provide recommendations related to mode of treatment, diet, examination and medical supervision during recovery period.

3. Information to be obtained during pre-classroom independent work.

3.1.Basic knowledge and skills necessary for subject learning (interdisciplinary integration)

Discipline	To know:	To be able to:
Microbiology	Specific features of Leptospira, Hantavirus (4 types of epidemic hemorrhagic fever germs); methods of specific diagnostics of leptospirosis and epidemic hemorrhagic fever	Estimate results of specific diagnostics methods of leptospirosis and epidemic hemorrhagic fever
Physiology	Aspects of physiological standards of human organs and systems; aspects of laboratory examination in standard condition (general blood and urine analysis, biochemical blood analysis, features of negative resistance converters, electrolytes etc).	Estimate data of laboratory examination
Pathologic physiology	Mechanism of disorders in functioning of organs and systems at pathological conditions of various genesis	Interpret pathological modifications based on results of laboratory examination at disorders of organs and systems functions of various genesis
Epidemiology	Epidemiological process (source, mechanism of infection, routes of transmission) at leptospirosis and epidemic hemorrhagic fever, prevalence of such pathologies in Ukraine and worldwide	Make up an epidemiological history, perform antiepidemic and preventive measures in the centre of infection
Immunology and allergology	Key terms of the discipline, role of immunity system in infectious process, influence on the term of	Analyze data of immunological examinations

	germ elimination from human organism. Immunological aspects of complications	
Neurology	Pathogenesis and clinical signs of toxic encephalopathy, meningism, meningitis, epidemic hemorrhagic fever and eclampsia	Perform clinical examination of the patient with affected nervous system
Dermatology	Pathogenesis and clinical characteristics of exanthema	Define rash of the patient with leptospirosis and epidemic hemorrhagic fever
Surgery	Clinical and laboratory signs of gastrointestinal hemorrhage, methods of rescue emergency care	Make due diagnosis of such complications, prescribe proper examination and provide rescue emergency care
Nephrology, urology	Clinical and laboratory signs of epidemic hemorrhagic fever with uremic coma, kidney rupture and renal capsule tear	Make due diagnosis of such complications, prescribe proper examination and provide rescue emergency care
Propedeutics of medical diseases	Main stages and methods of patient clinical examination	Make up medical history, perform clinical examination of the patient by different organs and systems, and define clinical symptoms of pathology. Analyze received data
Clinical pharmacology	Pharmakokinetics and pharmakodynamics, adverse effects of penicillin, tetracyclines and cephalosporins of II generation and means of pathogenic therapy	Prescribe treatment with regard to age, individual symptoms of the patient, chose an optimum mode of drug intake and dosage, provide prescriptions
Reanimation and intensive care	Emergencies: - TSS, DIC syndrome; - Acute renal failure; - Febrile psychosis	Make due diagnosis of and provide rescue care in emergencies


LEPTOSPIROSIS
Causation
Leptospira interrogans
L-forms, endotoxin, fibrinolysin, plasma coagulase, hemolysin
Epidemiology
Feral herd zoonosis, source – rodents, dogs, pigs and bovine cattle. Transmission aspects – water, food, plants and soil. Routes of transmission – via affected dress, foodborne and aspiration. Season – summer and autumn. Long-term, specific immunity. General sensitivity.
Pathogenesis
Penetration→ Regional lymphadenitis→ Primary leptospiremia→, dissemination
Reproduction in organs
Secondary leptospiremia, toxemia
Contamination of organs
Recidives
Complications
Death
Germ elimination
Recovery
Specific antibodies
Clinics
Latent period
Early stage
General capillary purpura
Premunition
Sterile immunity
Intoxication
fever
Hypotonia
Myodinya
Hemorrhagic rash
Iritis, iridocyclitis
Nephrosonephritis
Hepatolienal syndrome
Yellow sickness
Meningitis
Complications
TSS Waterhouse-Friderichsen syndrome
Syndrome of disseminated intravascular coagulation
Acute renal failure
Acute hepatic encephalop
Brain swelling
Stable angina
Respiratory failure
Specific diagnostics
Microscopy and Leptospira extraction from biological material
Complement fixation test, reaction of lysis microglutination, PHT
Treatment
Antibiotics
Detoxication
anti-leptospirozis hemoglobulin
Desensitizers
angioprotectors
Complication treatment
Acute coronary syndrome
Disaggregants
Pi protease inibitor
Diuretics
Acid-base balance correction
hemodialysis
Prophylactics
Non-specific
Specific according to indications

Epidemic hemorrhagic fever

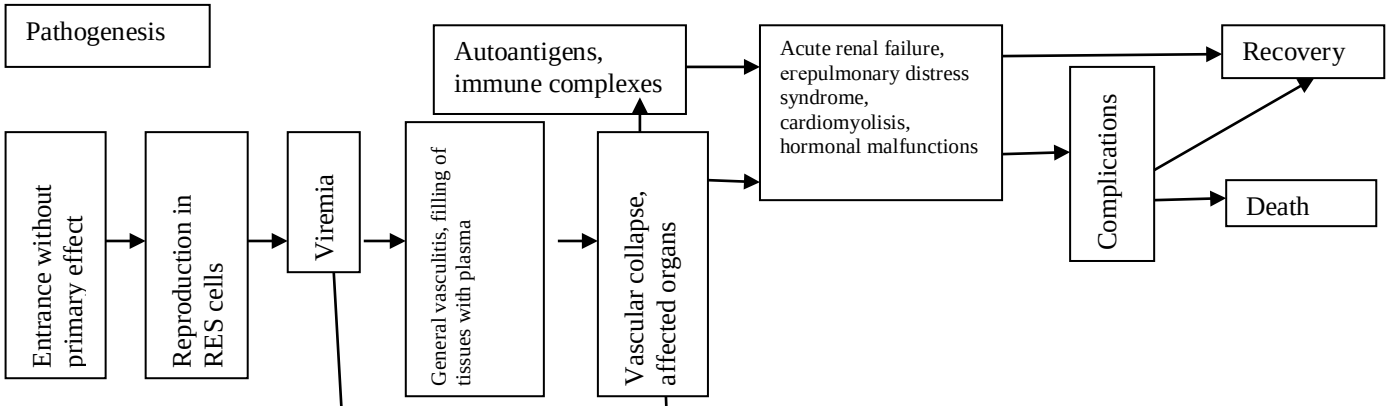
Causation

RNA-containing virus (2 forms – eastern and western)

Epidemiology

Feral herd zoonosis, source and container of infection – mice, rats and other rodents. Routes of transmission – air-borne, contact, foodborne. Season – May - October. Long-term, life-long immunity.

Pathogenesis



Clinics

- Early stage (1-3 days): Fever, intoxication, dermahemia and mucous hyperemia, mucous hemorrhage, meningeal syndrome
- Oligo-/anuretic period (4-11 days): toxicosis, ache in red-footed alcon, vomiting, thrombosis, hemorrhagic rash, hemorrhage, oligo/anuresis
- Polyuretic peiod (12-20 days): asthenia, polyruia
- Recovery period (from 21st -25th day): slow function renewal

Complications

- Uremia
- Renal rupture
- Acute vascular failure
- Pulmonary edema
- Encephalomeningitis
- Eclampsia
- Focal pneumonia

Specific diagnostics

- IIFR, EIA, NT
- Virus cultivation in cell culture

Treatment

- Bed rest
- Deintoxication
- Acid-base balance correction
- Pi protease inhibitors
- Heparin
- Deaggregants
- Prednisolone
- Antihistamines
- Hemodialysis

Prophylactics

- Non-specific (deratization)
- Formalin and recombinant

3.4. Materials for self-control.

3.4.1. Questions for self-control:

1. The main pathogenic aspects of Liptospira.
2. Source of infection at leptospirosis.
3. When and why does yellow sickness occur at leptospirosis?
4. What clinical syndromes develop at serious form of leptospirosis?
5. Characterize renal disorders at leptospirosis.
6. In what time period can relapse of the disease occur at leptospirosis?
7. Characterize intravascular coagulation at leptospirosis.
8. Name specific methods of leptospirosis diagnostics.
9. What antimicrobial drugs are used in treatment of leptospirosis?
10. List complications of leptospirosis.
11. Source and mechanism of infection at epidemic hemorrhagic fever.
12. Classification of epidemic hemorrhagic fever.
13. The main chains of pathogenesis of epidemic hemorrhagic fever.
14. The main clinical syndromes of epidemic hemorrhagic fever.
15. Fever curve at epidemic hemorrhagic fever.
16. Complications of epidemic hemorrhagic fever.
17. Criteria of epidemic hemorrhagic fever diagnosis.
18. Characteristics of drugs used for treatment of patients with epidemic hemorrhagic fever.
19. Rules of discharge for patients recovered from epidemic hemorrhagic fever.
20. Specific signs of renal disorders at epidemic hemorrhagic fever.

3.4.2. Tests for self-control *a=2*

Choose correct answers

1. Name mechanism of infection transmission at leptospirosis:

A. fecal - oral;

B. airborne;

C. percutaneous;

D. arthropod-borne;

E. multiple.

2. Pathogenic aspects of Leptospira include:

A. endotoxin;

B. plasm coagulase;

C. hematoxin;

D. exotoxin;

E. fibronolysin;

3. Key syndrome of epidemic hemorrhagic fever pathogenesis is the development of:

A. distal colitis;

D. endocarditis;

C. peritonitis;

D. vascular endothelium affect;

E. encephalomeningitis.

4. A patient was admitted to the hospital on the 7th day of the disease with complaints of high temperature, headache, pain in the muscles, especially in calf muscles. Dermal integuments and scleras are icteric. There is hemorrhagic rash on the skin. Urine is bloody. The patient was fishing two weeks ago. What is the most likely diagnosis?

A Leptospirosis

B Yersiniosis

C Salmonellosis

D Brucellosis

E Trichinellosis

5. Leptospirosis is a disease:

A. anthroponotic

B. zoonotic

C. anthropozoonotic

D. sapronotic

6. The source of infection in leptospirosis is

A. gamblers

B. Wolves

C. the camels

D. person

E. birds

7. For seasonal leptospirosis:

A. spring

B. winter

C. Summer-Autumn

D. Summer

8. The most common way of transferring leptospira is:

A. Water

B. transmissible

C. alimentary

D. Parenteral G.

E. AD airborne

9. The infection with leptospirosis occurs

A. when swimming in water.

B. with a tick bite.

C. with the use of pickled mushrooms.

D. in contact with a sick person.

E. with a mosquito bite.

10. Typical for leptospirosis:

A. lesions of the gastrointestinal tract

B. catarrhal syndrome

C. Bulbar syndrome

D. Kidney damage

E. sardonic smile

11. With leptospirosis is typical:

A. manifestations of hemorrhagic syndrome

B. chair in the form of "marsh mud"

C. severe damage to the lymphatic system

D. syndrome of "hood and gloves"

E. opisthotonus

12. For leptospirosis is typical:

A. Incubation period from 3 to 30 days

B. incubation period from 2 to 24 hours

C. incubation period from 15 to 45 days

D. Incubation period up to 6 months

E. incubation period up to 12 months

13. When leptospirosis is typical:

A. subacute onset of the disease

B. gradual onset of the disease

C. acute onset of the disease.

14. Characteristic symptom of leptospirosis:

A. Arthralgia, diarrhea, acute adrenal insufficiency

B. fever, myalgia, hepatic-renal insufficiency

C. abdominal pain, vomiting, diarrhea

D. ptosis, miosis, anisocoria, strobism.

E. tenesmus, stool with an admixture of blood.

15. When leptospirosis is typical:

A. Dry mouth

B. pain in the calf muscles

C. violation of swallowing

D. hallucinations

E. symptom of Rosenberg.

16. For the initial period of leptospirosis is typical:

A. Chills, fever

B. symptom of Chiari - Avcina

C. cramping abdominal pain

D. frequent loose stools.

E. convulsive syndrome.

17. With leptospirosis is characteristic:

A. roseous rash

B. symptom of Padalki

C. VOOP

D. orcoepididymitis

E. exophthalmos.

18. Leptospirosis should be differentiated with:

A. shigellosis

B. Viral hepatitis

C. botulism

D. tetanus

E. Tularemia

19. A typical symptom for leptospirosis is:

A. Hypertension.

B. myalgia.

C. neuralgia.

D. catarrhal respiratory syndrome.

E. Diarrhea

20. The characteristic complications of leptospirosis are:

A. ARF

B. neuritis of the auditory nerve

C. peritonitis

D. cholecystitis

E. colitis.

21. For hemograms in leptospirosis is typical:

A. Thrombocytosis

B. eosinophilia

C. neutrophilic leukocytosis with a shift of the formula to the left

D. Lymphopenia

E. relative lymphocytosis

22. Diagnosis of leptospirosis is confirmed:

A. detection of leptospira in the blood with the help of darkfield microscopy

B. The reaction of Vidal

C. Breakout of Byurne

D. detection of leptospira in feces.

E. isolation of the pathogen from food

23. For bacteriological diagnosis of leptospirosis use:

A. phlegm

B. blood

C. feces

D. sperm

E. nasopharyngeal mucus

24. For the diagnosis of leptospirosis use:

A. Sample with anthraxin

B. skin - allergic test Byrne.

C. by the reaction of Vidal

D. smear and a thick drop of blood.

E. RALL with reference staffs

25. For specific therapy of leptospirosis apply:

A. antileptospiroznaya vaccine

B. anti-leptospirosis gamma-globulin.

C. antileptospirosis bacteriophage.

D. anatoxin

26. The driver of KGF is

A. virus

B. bacterium

C. Protozoa

D. chlamydia

E. Mycoplasma

27. Crimean hemorrhagic fever

A. is called bunyavirus

B. is caused by flavivirus

C. is caused by a filovirus

D. is caused by arenavirus

28. Krymskaya hemorrhagic fever

A. is a natural focal disease

B. transmitted by transmission

C. can be transmitted from a sick person

D. is transmitted by alimentary route

29. The main source of CHF

A. Hare

B. domesticated birds

C. foxes

D. mosquitoes

30. An increase in the incidence of CHF is observed in

A. February-March

B. March-April

C. May-July

D. October-December

31. Characteristic for Crimean hemorrhagic fever

A. Zheltuha

B. enlarged spleen

C. a petechial rash

D. stiffness of the muscles of the occiput

E. Muscle Pain

32. The most typical symptoms of the prehemorrhagic period of CHF

A. Injection of vessels of sclera and conjunctiva

B. puffiness of the face

C. high fever

D. impaired vision

33. Cause of death in Crimean hemorrhagic fever

A. acute renal failure

B. acute respiratory failure

C. a broken kidney

D. Hemorrhage and hemorrhage.

34. In the general analysis of blood in CHF,

A. persistent leukopenia

B. Thrombocytosis

C. leukocytosis

D. Thrombocytopenia

E. eosinophilia

35. For specific diagnosis, CHF is used

A. PCR with detection of the DNA of the CSL virus

B. ELISA with detection of specific antibodies to CHL virus

C. Bacteriological method

D. Blood smear microscopy

36. For etiotropic treatment, CHF is used

A. Acyclovir

B. interferon

C. ribavirin

D. There is no etiotropic treatment.

37. Hemorrhagic fever with renal syndrome

A. is called hantavirus

B. is caused by flavivirus

C. is caused by a filovirus

D. is caused by arenavirus

38. Hemorrhagic fever with renal syndrome is characterized

A. fever

B. liver damage

C. the defeat of the kidneys

D. hemorrhagic syndrome

39. One of the dangerous specific complications of HFRS is

A. acute liver failure

B. purulent meningitis

C. acute renal failure

D. perforation of ulcers of the intestine

40. The following hemorrhagic fevers may be transmitted from person to person

A. Dengue

B. Ebola

C. Crimean hemorrhagic fever

D. Marburg

E. Lossa

41. The carriers of hemorrhagic fevers may be

A. mosquitoes

B. Lice

C. fleas

D. mites

42. Periods of hemorrhagic fevers

A. prehemorrhagic

B. hemorrhagic

C. incubation

D. latent

43. Etiotropic therapy with ribavirin is prescribed if the causative agent of hemorrhagic fever is

A. Bunyavirus
B. flavivirus
C. filovirus
D. arenavirus

44. For the pathogenetic therapy of hemorrhagic fevers,

A. fused plasma
B. Thrombose concentrate
C. Specific horse serum
D. erythrocytic mass

Right answers:

1. C;	9. A	17. C	25. B	33. D	41. A, D
2. A, B, C, E;	10. D	18. B	26. A	34. A, D	42. A, B, C
3. D	11. A	19. B	27. A	35. A, B	43. A, D
4. A	12. A	20. A	28. A, B, C	36. C	44. A, B, D
5. B	13. C	21. C	29. A	37. A	
6. A	14. B	22. A	30. C	38. A, C, D	
7. C	15. B	23. B	31. C, D	39. C	
8. A	16. A	24. D	32. A, B, C	40. B, C, D	

Fill in the table: ***a=3***

Dynamics of leptospirosis bacteriological diagnostic methods

Period of disease / Method	1st week	2nd week
Blood bacterioscopy	+	-
Urine bacterioscopy	-	+
Spinal liquid bacterioscopy	+	-
Bacterioscopy of tissue biopsy samples	+	-
Biological method	+	-
Blood inoculation	+	-

3.4.3 Problems for self-control.

Problem 1. **a=2**

A man, 37 years old, is suffering from the disease for 3 days – the disease was acute at early stage, the patient felt frozen, his temperature increased up to 39.6 C, he had "coffee-grounds" vomiting. He took antipyretic drugs, without any effect. Yellow sickness developed today and caused hospitalization of the patient with a suspicion of viral hepatitis. Medical history showed that the patient had rabbits, nutrias and a dog in his household. During examination the patient was conscious, T 39.5 C; swollen face, hyperemic, conjunctival vascular injection, hemorrhage under conjunctiva from both sides. Positive pinch sign, seldom petechias on skin close to collar bones. Hoarse breathing. Muffled and rhythmic heart sounds. Dry tongue. Stomach is not tender, liver coming out from coastal arch for 3 cm, palpatory spleen. Positive Pasternatsky's symptom from both sides, daily urine made approximately 300 ml, urine –of reddish color.

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

4. Materials for auditory individual work.

4.1. List of practical tasks to be done during the practical class:

- Study the method of examination of patient with leptospirosis and epidemic hemorrhagic fever.
- Examine the patient with leptospirosis and epidemic hemorrhagic fever.
- Perform differential diagnostics of leptospirosis and epidemic hemorrhagic fever.
- Make up a plan of laboratory examination
- Study the results of specific examination of patients with leptospirosis and epidemic hemorrhagic fever.
- State the complications of leptospirosis and epidemic hemorrhagic fever.
- Make up a treatment plan for the patient with leptospirosis and epidemic hemorrhagic fever.
- Define medical approach in case of emergencies.
- Arrange medical documentation based on diagnosis of leptospirosis and epidemic hemorrhagic fever.

4.2. Professional algorithm of gaining knowledge and skills about leptospirosis and epidemic hemorrhagic fever.

#	Task	Sequence of actions	Remarks and cautions for self-control
1.	Study the methods of examination of patient with leptospirosis and epidemic hemorrhagic fever Examine the patient	I. Define the complaints of the patient.	Divide complaints attributable to syndromes of: - total toxicosis - organs attack
2.		II. Define the history: 1. Medical history	Pay attention to acute start, period, sequence of symptoms and intensiveness of - fever; - headache; - insomnia; - rash; - other symptoms.
		2. Patient's life history	Define previous diseases.
		3. Epidemic history	Define data related to realization of vector-borne transmission mechanism, pay attention

			to patient's residence in centers of leptospirosis and epidemic hemorrhagic fever.
		II. Perform proper examination.	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
		1. General examination: - general condition of the patient;	Pay attention to: - psychoemotional state of the patient; - body temperature; - skin modifications (color, rash); - availability, location and type of rash on skin and mucous membranes;
		- skin, mucous membranes of oral pharynx;	Pay attention to: - Patient's movements; - Dominating location of pain.
		2. Muscles and skeleton. - patient's manner of walking; - muscles palpation.	Pay attention to: - Sleep pattern; - availability of meningeal signs, impairment of consciousness;
		2. Nervous system: - insomnia; - pathological symptoms;	- pathological reflexes and convulsions; Pay attention to: - cardiac rate, extrasystole; - hypotonia (signification reduction means complications!);
		3. Heart-vascular system: - beat; - arterial tension;	- moderately muffled heart sounds. Pay attention to: - signs of bronchitis in some patients.
		- heart auscultation.	Pay attention to: - reduced diuresis; - urine color;
		4. Respiratory system: - lungs auscultation. 5. Excretory system:	Pay attention to: - Hepatolienal syndrome; - diarrhea.
		6. Digestive system:	
3.	Prescribe laboratory and other testing, estimate their results	1. General blood analysis.	Pay attention to typical changes: leucocytosis with a left shift, thrombocytopenia, erythropenia.
		2. General urine analysis	Oliguria, albuminuria, cylindruria, hypo(iso)sthenuria, micro- and macrohematuria.

		3. USE of abdominal cavity organs	Hepatolienal syndrome
		4. Spinal fluid diagram	Neutrophilous, and further lymphocytic pleocytosis, modified erythrocytes.
		5.Biochemical blood examination	Metabolic acidosis, reduced electrolyte level, increased BUN, creatinine, alanine aminotransferase, ACT, lactoferrin, creatine phospokinase, hypoglycaemia, coagulation failure.
		6. Serological methods: - HVF, - IGHT, - ELISA	Prescribed in paired serums with an interval of 10 days; - from the 2nd week, diagnostic titer 1:100; - from the 2nd week, diagnostic titer 1:80; - from the first days.

Rabies, Tetanus

1.Importance of the theme:

After a long period of investigations, infections of the central nervous system – tetanus and rabies are very relevant today. In the middle of last century 150 cases of disease were reported annually in Ukraine, but rabies prophylaxis resulted in decreasing of morbidity rate and in the last decade of 20th century only few cases of human rabies have been reported. Increased number of cases was noted recently: in 2006 4 persons died of rabies in Ukraine (Odessa, Chernigov, Kirovograd and Donetsk regions).

Some 100 000-108000 persons annually seek for medical aid having been exposed to animals (exposures are categorized as either bite or nonbite), and 60% of them have been exposed to rabies-suspect animals. Recently 2-3 persons are said to die of disease each year in Ukraine, all of them had sought for medical aid at the onset of clinical rabies. The importance of tetanus is explained by its peculiarities: worldwide distribution, high incidence, the absence of long termed immune response after disease, very severe clinical course with high mortality rate (15-20%). Rabies occurs nearly worldwide – in 113 countries. Some 50 000 persons and millions of animals are said to die of the disease each year.

WHO considers rabies as one of the most dangerous diseases of humans and animals leading to great social and economic losses.

Importance of tetanus is explained by its peculiarities: worldwide distribution, high sensitivity, absence of long termed immunity even after disease, especially severe course of disease with high mortality rate (15-20%) In 1974 WHO established new strategy in infectious diseases eradication program. According to this strategy there are 6 diseases we must give top priority to control: diphtheria, measles, whooping cough, poliomyelitis, tuberculosis and tetanus (in 1990 yellow fever and hepatitis B were added to this list). All these diseases can be prevented by immunization. Nevertheless worldwide some 500 000 cases of tetanus are reported annually. Acute injuries are the usual portal of entry. Parenteral drug abuse was reported as a portal of entry too (non-sterile syringes, injections in low sanitary conditions) .

2. The Purpose of the Lesson (with indication of planning mastering level):

2.1. A student must know: *a-2*

- etiology of rabies, factors of virus' pathogenicity;
- etiology of tetanus, factors of virus' pathogenicity;
- epidemiology of rabies;
- epidemiology of tetanus;
- pathogenesis of rabies;
- pathogenesis of tetanus;
- clinical manifestations of rabies;
- clinical manifestations of tetanus;
- pathogenesis, terms and manifestations of complications in tetanus;
- laboratory confirmation of rabies;
- laboratory findings in tetanus;
- treatment of rabies;
- treatment of tetanus;
- prognosis in rabies;
- prognosis in tetanus;
- prevention of rabies in animals and humans;
- post exposure prophylaxis of rabies;
- primary prevention and wound management in tetanus ;
- treatment of emergency conditions that can occur in patients with rabies
- treatment of emergency conditions that can occur in patients with tetanus
- the discharge rules in tetanus;
- out patient follow up of convalescent after tetanus

2.2. A student must be able*: a-3*

- to gather information regarding patient's anamnesis with estimation of

epidemiological data in a person with suspected rabies;

- to gather information regarding patient's anamnesis with estimation of epidemiological data in a person with suspected tetanus;
- to examine patient with suspected rabies and substantiate preliminary diagnosis for early hospitalization;
- to examine patient with suspected tetanus and substantiate preliminary diagnosis for early hospitalization;
- to make differential diagnosis of rabies with other clinically similar disorders- tetanus, botulism, lethargic encephalitis, poliomyelitis, polyneuritis delirium tremens, atropine and strychnine poisoning;
- to make differential diagnosis of tetanus with other clinically similar disorders- paratonsilitis, trichinosis, rabies, hysteria, meningitides, eclampsia;
- to diagnose possible complications of tetanus and emergency conditions on a basis of clinical assessment;
- to compose a plan of laboratory and additional investigations of rabies-suspect animal
- to administer treatment for a patient with tetanus, taking into consideration epidemiology data, stage of disease, grade of severity, presence of complications, allergy and underlying diseases;
- to give first medical aid for a patient with preliminary diagnosis of rabies before hospitalization
- to give first medical aid for a patient with preliminary diagnosis of tetanus before hospitalization
- to compose a plan of rabies postexposure prophylaxis
- to compose a plan of rabies preexposure prophylaxis and preventive measures in a focus of rabies.
- to compose a plan of tetanus prevention after injury
- to give recommendations regarding regimen, diet, investigations and management of a convalescent after tetanus

3. Materials for prior to auditory independent work

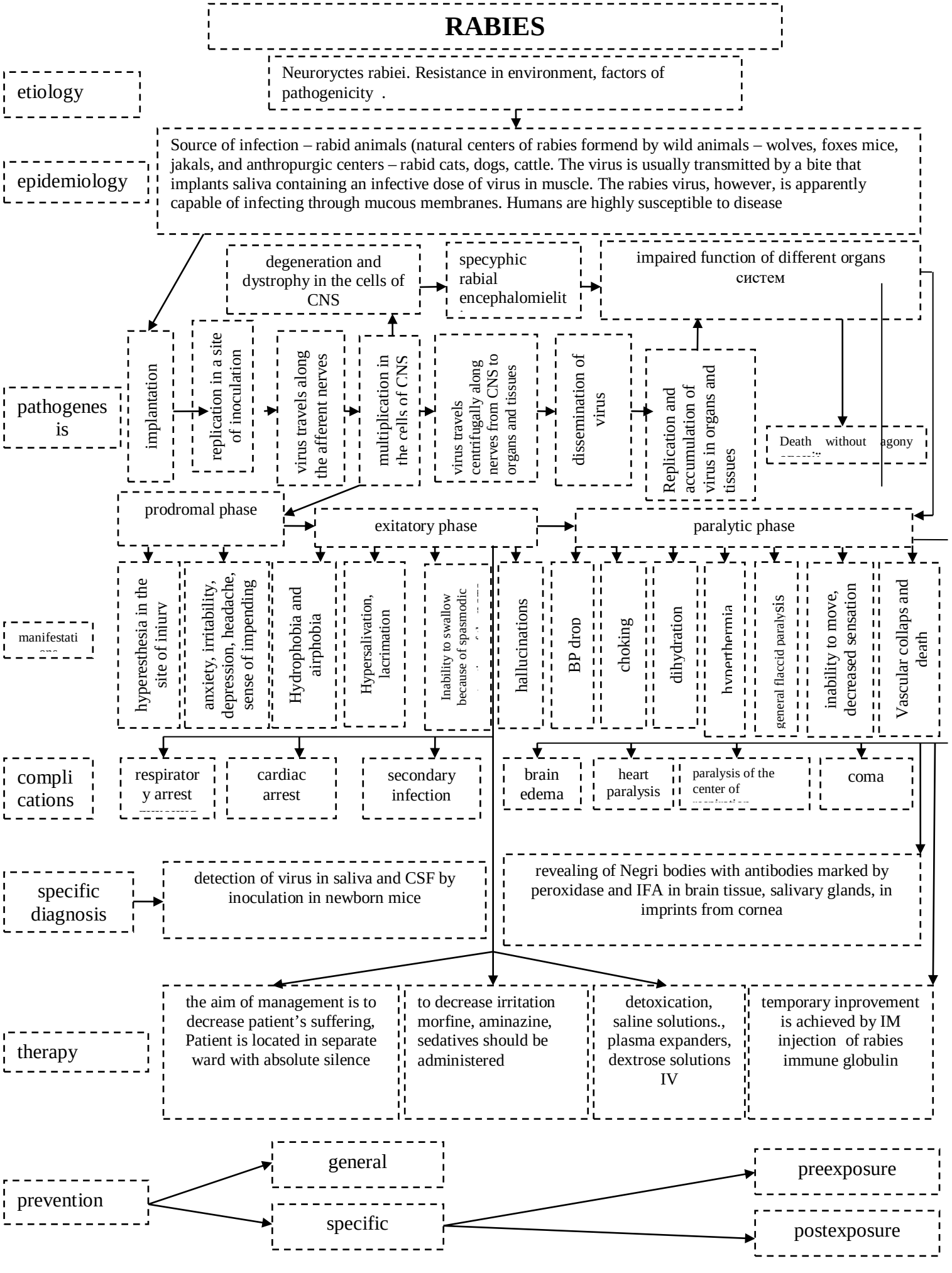
3.1. Basic knowledge, skills and habits necessary for study of a topic (Subjects integration)

Subjects	To Know	To Know How
Microbiology	Etiology (classification, morphologic characteristic of the pathogen- *Clostridium tetani* і *Neuroryctes rabiei*, methods of revealing and identification)	To reveal the organism. To calculate concentration of disinfectants for disinfection in a case of tetanus and rabies.
Histology	Histological changes in tetanus. Peculiarities of inclusion bodies (Negri bodies) in the neurons of the brain	To reveal Negri bodies in histological preparation. To explain clinical signs appearance.
Physiology	Musculoskeletal, cardiovascular, nervous, renal and immune systems function	To explain a variety of clinical signs and laboratory abnormalities
Pathological Physiology	Pathogenesis of tetanus and rabies. Pathogenesis of complications.	To explain the main symptoms and signs appearance, causes of relapse, failure of inadequate therapy
Clinical immunology and allergology	Immunologic changes as a part of pathogenesis and host defenses. Immunologic peculiarities of antitetanus immune response.	To explain confirmative serologic tests. To appraise the results of immunologic investigations (titer of antitoxic antibodies in patients with tetanus and healthy persons). To appraise immunogram of a patient.
Epidemiology	The routs of transmission, main sources of infection in tetanus and rabies. Distribution of these diseases in Ukraine and in the world.	Epidemiological history. To organize preventive measures at a site of rabies (campground, village).
Neurology	Pathogenesis of tetanospasms, fever, risus sardonicus anxiety, excessive salivation	Examination of patients with different central and peripheral nervous system disorders.

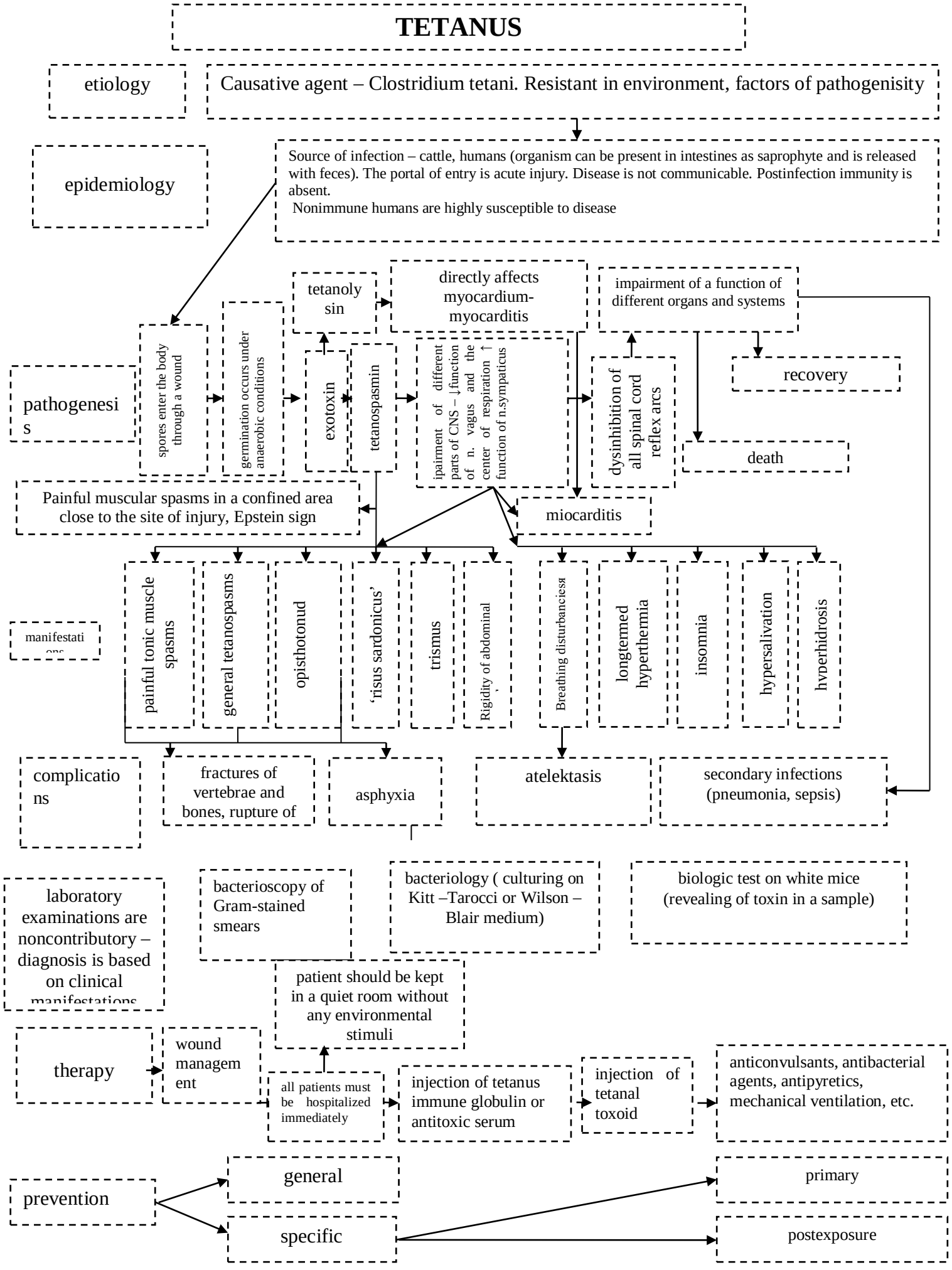
	and other symptoms of tetanus and rabies	Differential diagnosis with hysteria, hypocalcemia, meningoncephalitides, and meningitides if different etiology.
Surgery	Manifestations of injuries, painful muscle spasms and spontaneous rupture of muscles and ligaments, fractures of bones, contractures; principles of intensive care of emergency case.	Differential diagnosis, wound management, primary prevention, diagnosis of complications possible in tetanus, to order investigations and administer suitable therapy.
Propaedeutics of Internal Diseases	History of disease. Patient's examination.	To gather information about patient's history and chief complaints, to distinguish those, most important for diagnosis of tetanus and rabies. To examine the patient, to reveal the main symptoms and signs of disease. To distinguish the set of diagnostic features of tetanus and rabies.
Pharmacology	Regimens of treatment. Supportive care. Pharmacodynamics, side effects of chloramphenicol, anticonvulsants, sedatives, anaesthetic analgesics and other medication recommended for treatment of tetanus and rabies.	To administer treatment of specific infection including ancillary therapy. To write the scheme of treatment in tetanus and rabies.
Intensive care **Emergency conditions:** Acute respiratory failure Cardiac arrest	Emergency conditions: Acute respiratory failure Cardiac arrest Convulsions Acute brain edema	To diagnose emergency condition, to administer intensive care

Convulsions Acute brain edema To diagnose emergency condition, to administer intensive care		
Dentistry	**Lockjaw**	**Differential diagnosis with dental caries, acute arthritis.**
	Future subjects	
General practice medicine	Pathogenesis, epidemiology, manifestations, complications, prevention and therapy of tetanus and rabies.	Differential diagnosis of clinically similar diseases with tetanus and rabies. Early diagnosis and hospitalization of patients. First medical aid in out patient.
Themes integration		
Infectious diseases meningoncephalitides, purulent meningitides, peritonsillar abscess, parotitis	To know peculiarities of manifestations, laboratory diagnosis, treatment	To differentiate tetanus with other infectious diseases with similar symptoms

3.2.1. Structural logical scheme of theme contents.



TETANUS


etiology
Causative agent – Clostridium tetani. Resistant in environment, factors of pathogenisity
epidemiology
Source of infection – cattle, humans (organism can be present in intestines as saprophyte and is released with feces). The portal of entry is acute injury. Disease is not communicable. Postinfection immunity is absent.
Nonimmune humans are highly susceptible to disease
pathogenesis
spores enter the body through a wound
germination occurs under anaerobic conditions
exotoxin
tetanoly sin
tetanospasmin
directly affects myocardium-myocarditis
impairment of a function of different organs and systems
ipairment of different parts of CNS – ↓function of n. vagus and the center of respiration ↑ function of n.sympaticus
dysinhibition of all spinal cord reflex arcs
recovery
death
Painful muscular spasms in a confined area close to the site of injury, Epstein sign
miocarditis
manifestations
painful tonic muscle spasms
general tetanospasms
opisthotonud
'risus sardonicus'
trismus
Rigidity of abdominal
Breathing disturbanciesя
longtermed hyperthermia
insomnia
hypersalivation
hyperhidrosis
complications
fractures of vertebrae and bones, rupture of
asphyxia
atelektasis
secondary infections (pneumonia, sepsis)
laboratory examinations are noncontributory – diagnosis is based on clinical manifestations
bacterioscopy of Gram-stained smears
bacteriology (culturing on Kitt –Tarocci or Wilson – Blair medium)
biologic test on white mice (revealing of toxin in a sample)
patient should be kept in a quiet room without any environmental stimuli
therapy
wound management
all patients must be hospitalized immediately
injection of tetanus immune globulin or antitoxic serum
injection of tetanal toxoid
anticonvulsants, antibacterial agents, antipyretics, mechanical ventilation, etc.
prevention
general
specific
primary
postexposure

3.4. Materials for self control

3.4.1. Questions for self-control:

1. Peculiarities of ”Street rabies virus” and “fixed rabies virus”.
2. Wild and domestic animals that serve as a sources of rabies in humans.
3. The mechanism of the nervous system damaging. The most damaged areas of the nervous system.
4. Clinical manifestations of rabies in animals (dogs).
5. Clinical manifestations of rabies in humans. Two distinct clinical patterns of rabies – furious (agitated) and paralytic (“dumb”). Rare clinical patterns – bulbar and cerebellar.
6. Differential diagnosis of rabies with tetanus, encephalitis, poliomyelitis, infectious polyneuritis, delirium tremens, strychnine and atropine poisoning, and other CNS diseases.
7. Laboratory diagnosis in the mammal responsible for bite and in patients. Samples for post-mortem confirmation of rabies; IFA, histological investigation, neutralization test. Management of the patient with rabies, role of sedatives, parenteral feeding..
8. Pre-exposure and postexposure prophylaxis.
9. Prevention of rabies: passive immunisation - antitoxin therapy with tetanus immune globulin of human origin (TIG), equine antitoxic serum (doses, rules of administration), active immunisation – tissue culture vaccines.
10. Anti- rabies service in out patient clinic.
11. Rabies post-vaccination neuroparalytic complications: meningoencephalitis, myelitis, polyneuritis, allergic reactions and anaphylaxis.
12. Prevention of rabies in domestic animals and cattle. Importance of sanitary propaganda.

13. Characteristic features of Clostridium tetani
14. Distribution of Clostridium tetani in the environment, portals of entry. Conditions for germination.

15. Tetanus-susceptible individuals – incidence rates in different age groups.
16. Injuries that are the most common portals of entry for Clostridium tetani.
17. Toxic and bacterial components of tetanus' pathogenesis.
18. Tetanolysin, tetanospasmin and tetanal toxoid .
19. The mechanism of the nervous system injury. The most damaged areas of the nervous system.
20. Clinical classification of tetanus – localized, generalized, and cephalic forms; fulminant, acute, sub acute, chronic, relapsing.
21. Clinical diagnosis of tetanus.
22. The terms of the disease onset. Early signs of tetanus.
23. Facial expression and body position in tetanus.
24. Reflex muscle contractions in tetanus.
25. The progression of the disease. Complications of tetanus.
26. Tetanus neonatorum.
27. Prognosis in tetanus, depending on age of patient, duration of incubation and wound localization.
28. Differential diagnosis of tetanus with purulent meningitis and encephalitis, rabies, epilepsy, hysteria, variety of metabolic conditions and poisonings (hypocalcemic tetany, strychnin poisoning), differential diagnosis of trismus with tonsillitis, dental caries, peritonsillar abscess, parotitis and other CNS diseases. Differential diagnosis of tetanus with purulent meningitis and encephalitis, rabies, epilepsy, hysteria, variety of metabolic conditions and poisonings (hypocalcemic tetany, strychnin poisoning), differential diagnosis of trismus with tonsillitis, dental caries, peritonsillar abscess, parotitis and other CNS diseases.
29. Laboratory findings in tetanus.
30. Therapy of tetanus: antitoxin therapy with tetanus immune globulin of human origin (TIG), antitoxic serum of equine origin(doses, rules of administration), tetanal toxoid in the treatment of the disease.
31. Supportive care in tetanus.
32. Intensive care in tetanus.

33. Management of patients with tetanus; indications for mechanical ventilation.
34. Rules for discharge of convalescents after tetanus.
35. Preventive measures (primary prevention, wound management).
36. Indications for prophylaxis of tetanus with Td and TIG, a scheme of administration.
37. Prevention of tetanus in neonates.
38. Peculiarities of ”Street rabies virus” and “fixed rabies virus”.
39. Wild and domestic animals that serve as a sources of rabies in humans.
40. The mechanism of the nervous system damaging. The most damaged areas of the nervous system.
41. Clinical manifestations of rabies in animals (dogs).
42. Clinical manifestations of rabies in humans. Two distinct clinical patterns of rabies – furious (agitated) and paralytic (“dumb”). Rare clinical patterns – bulbar and cerebellar.
43. Differential diagnosis of rabies with tetanus, encephalitis, poliomyelitis, infectious polyneuritis, delirium tremens, strychnine and atropine poisoning, and other CNS diseases.
44. Laboratory diagnosis in the mammal responsible for bite and in patients. Samples for post-mortem confirmation of rabies; IFA, histological investigation, neutralization test. Management of the patient with rabies, role of sedatives, parenteral feeding..
45. Pre-exposure and postexposure prophylaxis.
46. Prevention of rabies: passive immunisation - antitoxin therapy with tetanus immune globulin of human origin (TIG), equine antitoxic serum (doses, rules of administration), active immunisation – tissue culture vaccines.
47. Anti- rabies service in out patient clinic.
48. Rabies post-vaccination neuroparalytic complications: meningoencephalitis, myelitis, polyneuritis, allergic reactions and anaphylaxis.
49. Prevention of rabies in domestic animals and cattle. Importance of sanitary propaganda.
50. Characteristic features of Clostridium tetani
51. Distribution of Clostridium tetani in the environment, portals of entry. Conditions for germination.

52. Tetanus-susceptible individuals – incidence rates in different age groups.
53. Injuries that are the most common portals of entry for Clostridium tetani.
54. Toxic and bacterial components of tetanus' pathogenesis.
55. Tetanolysin, tetanospasmin and tetanal toxoid .
56. The mechanism of the nervous system injury. The most damaged areas of the nervous system.
57. Clinical classification of tetanus – localized, generalized, and cephalic forms; fulminant, acute, sub acute, chronic, relapsing.
58. Clinical diagnosis of tetanus.
59. The terms of the disease onset. Early signs of tetanus.
60. Facial expression and body position in tetanus.
61. Reflex muscle contractions in tetanus.
62. The progression of the disease. Complications of tetanus.
63. Tetanus neonatorum.
64. Prognosis in tetanus, depending on age of patient, duration of incubation and wound localization.
65. Differential diagnosis of tetanus with purulent meningitis and encephalitis, rabies, epilepsy, hysteria, variety of metabolic conditions and poisonings (hypocalcemic tetany, strychnin poisoning), differential diagnosis of trismus with tonsillitis, dental caries, peritonsillar abscess, parotitis and other CNS diseases. Differential diagnosis of tetanus with purulent meningitis and encephalitis, rabies, epilepsy, hysteria, variety of metabolic conditions and poisonings (hypocalcemic tetany, strychnin poisoning), differential diagnosis of trismus with tonsillitis, dental caries, peritonsillar abscess, parotitis and other CNS diseases.
66. Laboratory findings in tetanus.
67. Therapy of tetanus: antitoxin therapy with tetanus immune globulin of human origin (TIG), antitoxic serum of equine origin(doses, rules of administration), tetanal toxoid in the treatment of the disease.
68. Supportive care in tetanus.
69. Intensive care in tetanus.

70. Management of patients with tetanus; indications for mechanical ventilation.
71. Rules for discharge of convalescents after tetanus.
72. Preventive measures (primary prevention, wound management).
73. Indications for prophylaxis of tetanus with Td and TIG, a scheme of administration.
74. Prevention of tetanus in neonates.

3.4.2. Tests for self control

Choose correct answers **α=2**

1. What group of infections tetanus belongs to?

A. Antroponosis
B. Vector-born infections
C. Slow infections
D. Endogenious infections
E. Antropozoonotic diseases

2. Characteristic clinical sign(s) of rabies:

A. Air phobia
B. Nephritis, hypothermia
C. Hepatitis, itching
D. Hypotonia, miocarditis
E. Hyperthermia

3. Characteristic clinical sign(s) of tetanus:

A. Opisthotonus
B. Photophobia, hypothermia
C. Acousticophobia, itching, bulbar palsy
D. Trismus, seizures
E. Hypotonia, hypothermia, vomiting

4. Methods of specific prophylaxis of tetanus:

A. Administration of antivirals
B. Immunization of infants

C. Immunization of adults from groups of risk.

D. Injection of specific immune globulin

E. Isolation of patient and injection of specific immune globulin

5. Clinical phases of rabies:

A. Prodromal

B. Prodromal, full-blown disease, convalescense

C. Prodromal, full-blown disease, chronisation

D. Exitatory

E. Paralytic

6. Rabies virus impairs:

A. Musculoskeletal system

B. Urinary tract

C. Nervous system

D. Liver and kidneys

E. Skin and mucous membranes

7. Methods of diagnosis of rabies in animals :

A. Bacteriological

B. Virological

C. Histological

D. Diagnosis is based on epidemiologic data only

E. Diagnosis is based on clinical data only

8. What is the main reservour and the source of infection in rabies in nature?

A. Foxes

B. Dogs and cats

C. Sick person

D. Wolves

E. Subjects contaminated with saliva of infected animals.

9. The way in which tetanospasmin is transported into CNS:

A. Through perineuronal lymphatic spaces

B. Through the blood stream, lymphatic system, and neuronal transport

C. Through blood-brain barrier

D. Along sensitive nerves

E. Through the blood stream

10. In tetanus:

A. Organism is sensitive to antibiotics

B. Diagnosis is confirmed by blood culture

C. Tetanospasmin is transported from the site of inoculation through the blood stream

D. The main cause of death is cardiac arrest

E. Tetanus immune globulin is used for prevention

Models of correct answers:

1. E 6. C

2. A, E 7. C

3. A, D 8. A, D

4. B, C 9. A, E

5. A, D, E 10. A

Fill the table: **α=3**

Differential diagnosis of rabies and tetanus (A.Chornovil

Disease **Symptoms and signs**	**Rabies**	**Tetanus**	**Lisophobia**	**Viral encephalitis**	**Botulism**
Increased temperature	+	+	–	+	–
Tonic muscle spasms	–	+	–	+–	–
Insomnia	+	+–	+–	+–	–
Hypersalivation	+	–	+–	–	–
Dryness of mouth	–	–	+–	–	+
Airphobia, hydrophobia, Acousticophobia	+	–	+–	–	–
Vision disorders (diplopic, blurred vision)	–	–	–	–	+
Alteration of consciousness	+	–	+–	+–	–
Flaccid asymmetrical paralysis	–	–	–	+	–
Good prognosis	–	+–	+	+–	+–
Local skin injury	+	+	–	–	–
Attacks of excitement	+	–	+	+–	–

3.4.3. Tasks for self control

Task 1 **α=2**

1. Patient, 43 years old, complains of pain and woodiness of low jaw movement, contraction of abdominal muscles. Then spasm of muscles of mastication followed resulting in inability to open the mouth and deglutitive problem. During last month he liked to run barefooted.

1. Preliminary diagnosis

2. Laboratory investigations

3. Treatment

Task 2 **α=2**

Woman, 26 years old, has been bitten by a street dog. Large lacerated wounds are localized in tie area of face.

1. What kind of first medical aid is recommended immediately?

Task 3 **α=3**

Man, 37 years old, has sought for medical aid. He‘s informed that 4 hours ago he has injured his foot. Patient couldn’t inform about immunization against tetanus.

1.Order investigations and administer treatment for this patient.

2. What postexposure prophylaxis should be administrered?

4. Materials for auditory independent work

4.1.1.List of training practical tasks, that should be carried out at the practice lesson :

- To master methods of examination of a patient with rabies.
- To examine a patient with rabies.
- To make differential diagnosis
- To compose a plan of laboratory investigations.
- To make conclusions based on the results of specific diagnostic procedures in a patient with rabies
- To compose a plan of treatment of a patient with rabies
- To define a mode of treatment of emergency conditions that may occur.

- To fill medical forms and documents on the fact of established diagnoses “Rabies”

4.1.2. List of training practical tasks, that should be carried out at the practice lesson :

- To master methods of examination of a patient with rabies.
- To examine a patient with rabies.
- To make differential diagnosis
- To compose a plan of laboratory investigations.
- To make conclusions based on the results of specific diagnostic procedures in a patient with rabies
- To compose a plan of treatment of a patient with rabies
- To define a mode of treatment of emergency conditions that may occur.
- To fill medical forms and documents on the fact of established diagnosis “Rabies”

4.1.3. List of training practical tasks, that should be carried out at the practice lesson :

- To master methods of examination of a patient with tetanus.
- To examine a patient with tetanus.
- To make differential diagnosis
- To compose a plan of laboratory investigations.
- To make conclusions based on the results of specific diagnostic procedures in a patient with tetanus.
- To compose a plan of treatment of a patient with tetanus.
- To define a mode of treatment of emergency conditions that may occur.
- To fill medical forms and documents on the fact of established diagnosis “Tetanus.”

4.2.1. Professional algorithm of skills formation in diagnosis of rabies

№	Task	Sequence of execution	Remarks and recommendations regarding self- control
1.	To master the methods of examination of patient with rabies To examine patient with suspected rabies	I. To evaluate patient's complaints	To separate complaints that characterize syndromes of: – local manifestations (abnormal unpleasant sensations in the site of inoculation – burning, local or radiating pain, pruritus, tingling) – impairment of CNS (early signs of psychics disorder: headache, anxiety, depression, sense of impending death, irritability)
		II. To find out anamnesis: 1. Anamnesis morbi	To pay attention at: – local inflammatory changes referred to the site of inoculation though a wound has been alreadyhealed; – fever; – loss of appetite – sleep abnormalities (with frightening dreams) – Other symptoms (visual and smell hallucinations are possible when face is the area of inoculation)
		2. Anamnesis vitae	To find out diseases and information about immunization patient had in the past.
		3. Epidemiologic anamnesis	To ascertain whether a patient visited regions with increased risk of rabies contraction (anthropurgic or natural center), the history of bite or deposition of saliva on impaired skin by a known or suspected rabid animal.

		III. To examine a patient 1. General examination 2.Gastrointestinal system 3.Cardio-vascular system 4. Respiratory system: 5. Nervous system	Remember: duration of disease is 4-8 days Pay attention at: –weakness, depression that are changed periodically by attack of excitation; – increased temperature from low-grade fever up to 40–41 °C; Pay attention at: – hypersalivation; – vomiting; – abdominal distention and constipation Pay attention at: – tachycardia; – arythmia; – hollowness of heart sounds Pay attention at: – tachypnea; – breathing disorders (cyclic respiration, choking) Pay attention at: – apathy and depression of prodromal phase are changed by increasing anxiety and, later, by unconsciousness – increased reflex excitability that is changed by prominent suppression of motor and sensory function; – hydrophobia, an increased sensitivity to air movement, bright light and loud noise; – convulsions; – visual and smell hallucinations
2.	To order laboratory investigations and additional diagnostic procedures, to explain their results	1. Complete blood analysis 2. Urinalysis 3. Histological methods: а) in dead animals б) in patients post mortem 4)biological test 5) Serological tests: a) in dead animals	In both animals and humans possible postmortem sources for virus isolation are brain, spinal cord, salivary glands, lacrimal glands, muscle tissue, lungs, kidneys, pancreas, adrenal glands. Brain tissue examination is used for Negri bodies presence. Mouse inoculation is recommended in diagnosis of rabies in animals. A fluorescent rabies antibody test (FRA)

		б) in patients post mortem	of animal's brain tissue is most sensitive and accurate means of examining for rabies that is generally available and reliability (99,95%) is such that a negative report weighs heavily in the decision not to treat a bitten person.

4.2.2.Professional algorithm of skills formation in diagnosis of tetanus

№	Task	Sequence of execution	Remarks and recommendations regarding self- control
1.	To master the methods of examination of patient with tetanus To examine patient with suspected tetanus	I. To evaluate patient's complaints	To separate complaints that characterize syndromes of: – local manifestations (painful muscular spasms in a confined area close to site of injury) General manifestations (difficulties in swallowing, fever, sweating, painful tonic muscle contractions, urinary retention, constipation) – impairment of CNS (insomnia, headache, anxiety, irritability)
		II. To find out anamnesis: 1. Anamnesis morbi 2. Anamnesis vitae 3. Epidemiologic anamnesis	To pay attention at: -abrupt onset – local inflammatory changes referred to the site of injury though it has been healed; – fever; – painful spasms of the muscles of mastication, and involvement of other muscle groups (back, abdomen, thorax) - other signs To find out diseases and information about immunization patient had in the past. Spores of C. tetani enter the body by inoculation through a wound. The most frequently reported injuries are puncture wounds and lacerations. Chronic lesions – decubitus ulcers, gangrene, burns, frostbite, dental and other abscesses are reported less frequently. Parenteral drug abuse is reported in some patients.

		III. To examine a patient 1. General examination 2. Muscular-skeletal system 3.Gastrointestinal system 4.Cardio-vascular system 5. Respiratory system: 6. Nervous system	Remember: incubation period may vary from 3 to 54 days (the majority of cases begin from 6 to 14 days following an acute injury). Injuries to the lower extremities are likely to be associated with longer incubation period. Tetanus may take the next clinical forms: 1)generalized, typical (descended); 2) atypical – bulbar, neonatal tetanus, fulminant, ginecological, thoracoabdominal, ascended; 3) local (cephalic). Examination should be performed very carefully, because every stimuli may lead to tetanospasms. Pay attention at: – generalized muscle rigidity; – risus sardonicus; – hyperthermia; – diaphoresis Pay attention at: – intensive muscle pain resulted from their persistent tonic contractions; – persistent and sustained tonic spasms of the back muscles - opisthotonus, abduction at the shoulders, flexion of the elbows and wrists, and extension of the legs. Pay attention at: – spasm of the pharyngeal muscles and trismus may result in swallowing disorders that may lead starvation and dehydration; – constipation Pay attention at: – tachicardia; – hypertensia – frequency breathing; – cramps of the respiratory muscles, diaphragm, also laryngospasm, which can lead to a difficult superficiales and frequent breathing, which is the reason for insufficient ventilation of the lungs; – congestive pneumonia; Attention for a: – totally preserved consciousness of the patient; – reflex convulsions, which can be painful and dangerous, and amplified by touch, other irritants; – deep tendon reflexes strengthened; – progressive hypoxia, which can cause irreversible changes in the central nervous system and lead to death.
2.	To order laboratory investigations and additional diagnostic	1. Complete blood analysis 2. Urinalysis 3. Bacteriological method	The absence of typical changes (except the cases with secondary bacterial flora complications) Specific diagnostic investigations are rarely used: it is based on revealing of organism and its

	procedures, to explain their results (diagnosis of tetanus is based on clinical manifestations, laboratory examinations are noncontributory)	3.Biological test	toxin in aspirates of affected areas. Anaerobic cultures of wound specimens are usually negative. Biological test is based on neutralization of toxin in mice and remains the standard reference method for assay of antitoxin in the serum.

5. Materials for after auditory independent work

- Peculiarities of rabies clinical course.
- Modern methods of laboratory confirmation of rabies.
- Present days problems of treatment of rabies.
- Modern explanation of pathogenesis of rabies.
- Peculiarities of clinical course of tetanus.
- Modern methods of laboratory confirmation of tetanus.
- Modern methods of treatment of tetanus.
- Modern explanation of pathogenesis of tetanus.

Erisipelas, Fellinosis

1.Actuality of theme:

The external covers of man protect the inner organs from negative factors of environment. Skin and mucous membranes is reliable barrier on the way to agent's penetration. However, frequent injuring of skin cause the declining of protective possibilities. Some agents can damage the skin, causing various diseases. Such illnesses are erysipelas and felinities.

Erysipelas is not a dangerous infections, it can't cause to death. However, it has a relapsive course and strikes the lymphatic capillaries of skin coursing elephantiasis as a result. Women suffer with erysipelas more frequent by than men.

Cats are living everywhere with men, they are the potential source of infection.The invasion of Bartonella hensella into the skin occurs themselves from bites, and scratches inflicted by cats. Local damages of skin are combined with the defeat of regional lymph nodes. This disease is called fellinosis or benign limphoreticulosis or cat scratch diseases.

Erysipelas and fellinosis are observed everywhere.

2. Lessons purpose (point of mastering level which is planned):

To study the etiology, epidemiology, clinic, laboratory diagnostics, differential diagnosis, treatment and prophylaxis of erysipelas and fellinosis.

2.1. ***A student must know:*** ***α-2***

- etiology of erysipelas, pathogenic factors of the agent;
- ways of streptococcus penetration into the skin;
- pathogenesis erysipelas;
- classification of erysipelas;
- clinical displays of different forms of erysipelas;
- complication of erysipelas;
- principles of clinical diagnostics and differential diagnosis;
- principles of treatment of erysipelas;

- principles of primary prophylaxis of erysipelas and relapse prophylaxis;
- dispensary of patients with the relapsive erysipelas over the health centre;
- etiology, source of infection and ways of invasion of cat-scratch diseases;
- pathogenesis and clinical displays of fellinosis;
- classification of clinical forms of fellinosis;
- criteria of diagnosis and specific methods of it's confirmation;
- treatment and prophylaxis of fellinosis.

2.2. A student must be able: ***α-3***

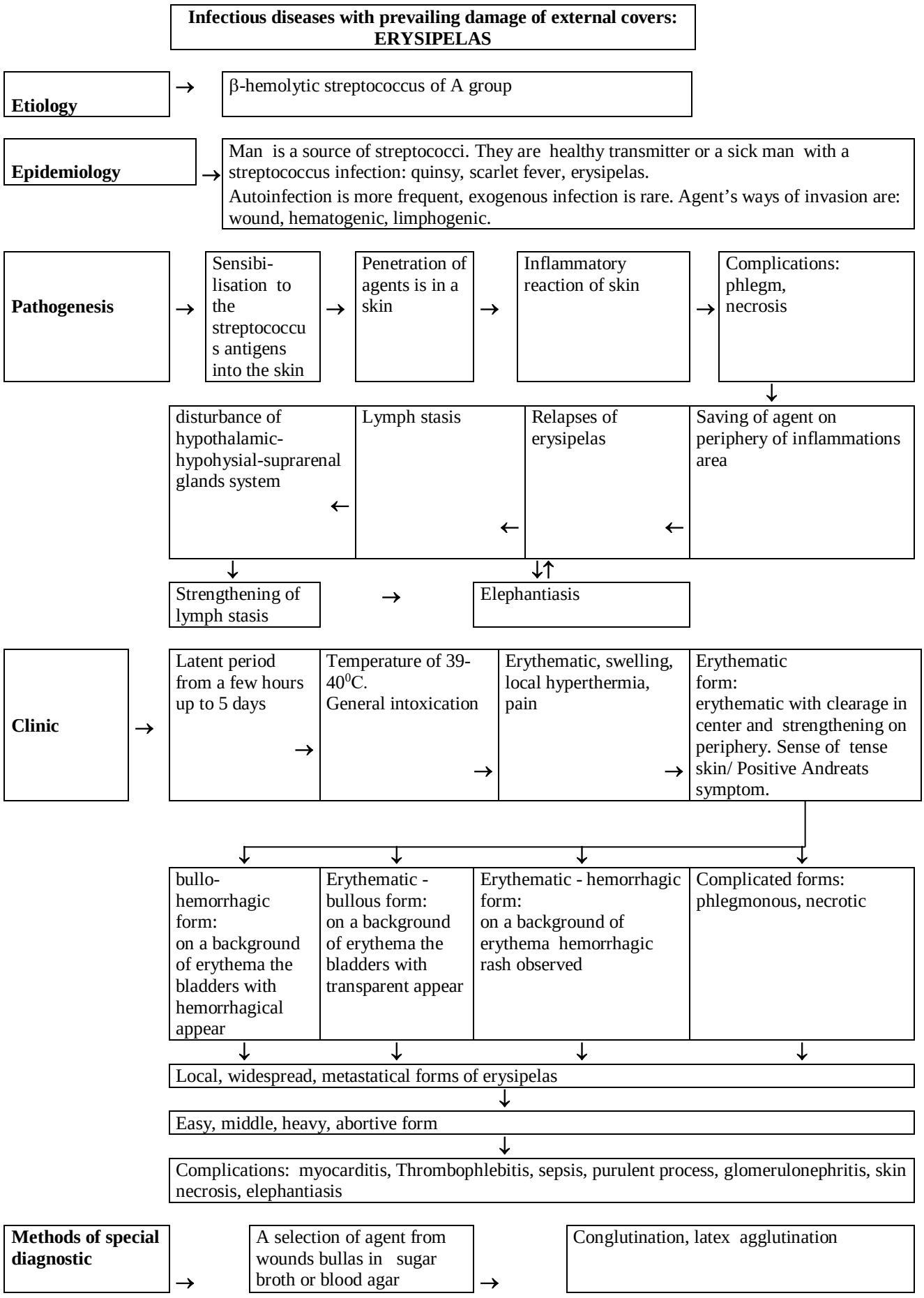
- To follow work rules bedside a patient with erisipelas and fellinos;
- to take history of illness and appraising the information of epidemiologic history;
- to examine a patient and find out basic symptoms and syndromes of erysipelas and cat-scratch diseases;
- to differential to erysipelas and cat-scratch diseases;
- to recognize different clinical forms of erysipelas and cat-scratch diseases;
- to fill in a medical document: an urgent report about infectious patient to a district epidemiology branch by fact of previous diagnosis "Erysipelas", "Fellinosis";
- to work out a plan of laboratory and instrumental examine of patient;
- to interpret the results of laboratory examination;
- to analyze the results of specific methods of diagnostics;
- to work out an individual plan of treatment taking into consideration: weight, the sex, clinical form of disease, allergic history, concomitant pathology;
- to work out a plan of clinical observation and treatment against the relapse of erysipelas;
- to give recommendations for to the measures of the unspecific prophylaxis of erysipelas and fellinosis;

3. Materials of preauditory independent work.

3.1.Basic knowledge, abilities, skills, necessary for a study theme (intersubjects integration.

Discipline	To know	To able
Previous disciplines		
Microbiology	Properties of β-Streptococcus. Methods of agent clean culture selection out of material from patient and its authentication. Characteristics of Bartonella hensella - agent of erysipelas.	To inoculate the material from patient on streptococcus. To carry out skins allergic tests. To Interpretive result PCR of the detection Bartonella hensella nucleic sequences/
Physiology	Physiology norm of parameters of men organs and systems of. The norm indexes of laboratory investigations (general blood, urine, biochemistry of blood analysis).	To estimate information of laboratory investigations.
Pathophysiology	Mechanisms of inflammation, damage of organ functions and systems organs	To interpret pathological changes of organ function by laboratory examination while erysipelas and felinosis.
Immunology and allergology	Basic object concepts, role of system immunity in infectious process. Immunological aspects of microorganisms persistence.	To estimate information of immunological investigations.
Epidemiology	Epidemic process (source, mechanism of infection invasion transmitting ways), spreading of the pathology among population.	To take epidemiologic history. To carry out prophylactic measures in the focus of infection
Neurology	The clinical signs of central and peripheral nervous systems damaging	To fulfil the clinical examination of patient with the damage of the nervous system.
Dermatology	Pathogenesis, clinical description of exanthema.	To recognize the characteristic changes of skin while erysipeloid and felinosis.
Surgery	The clinical and laboratorial signs of purulented lymph node, phlegm, abscess and its treatment.	To diagnose complicated forms illness and to prescribe adequate surgical treatment.
Internal diseases	The methods of patient clinical examination.	To take present history, fulfill clinical and laboratory investigation. To analyze the findings.
Clinical pharmacology	Pharmacokinetic and pharmacodinamic, side's effects for an antibiotic (penicillin, cephalosporin).	To prescribe an adequate treatment, depending on agent's sensitivity, individual features of patient, to choose the optimum regime of reception and dose of the drag, write recipes.
Reanimation and intensive therapy	Urgent states and complications of diseases: • toxico-infective shock • sepsis	To diagnose complications and provide urgent medical aids while • toxico-infective shock • sepsis

3.2. Structurally logical chart of maintenance of theme.*



↓ ↓

It is not reliable, can be positive for healthy people as well

↓

A diagnose is set on the basis of clinical symptoms characteristic

Treatment →	Etiotropic: Benzilpenicillin of 100-200 thousand /day; Ceftriaxon of 2,0 gr/day	Pathogenic: Nimesulid, diasolin prednisolon, UVR- treatment , angioprotective drugs (etamsilat sodium, venorhuton, ascorhutin)	Treatment of complications, including surgical treatment of abscesses, Philemon's, necrosis

Prophylaxis →	Prevention skin injery of skin, super cooling, hygiene and cleanness of skin	Timely treatment of streptococcus nature diseases	Prophylaxis of relapses: Bicillin-5 - 1.5 million in/m every third week during 2 years.

Infections diseases with the prevailing damage of external covers: Felinosis (cat crutch diseases)

↓

Etiology → Bartonella hensella (Gram-negative stick-form bacterium, immobile, does not have spores and capsules, persistent in an external environment, intracellular parasite. Afipia felis (Grame -negative stick, mobile with a plait).

↓

Epidemiology → Source of Bartonella hensella is a lady-cat, especially Siamese breed, rarely then dogs. Animals are not ill, but the transmitters of infection.
Ways of infecting: animal bite, scratches by claws, salivation.

Pathogenesis → Penetration of an agent through a skin → Local inflammatory reaction → Lymp henna delivery an agent in regional lymphatic nodes

↓ ↓

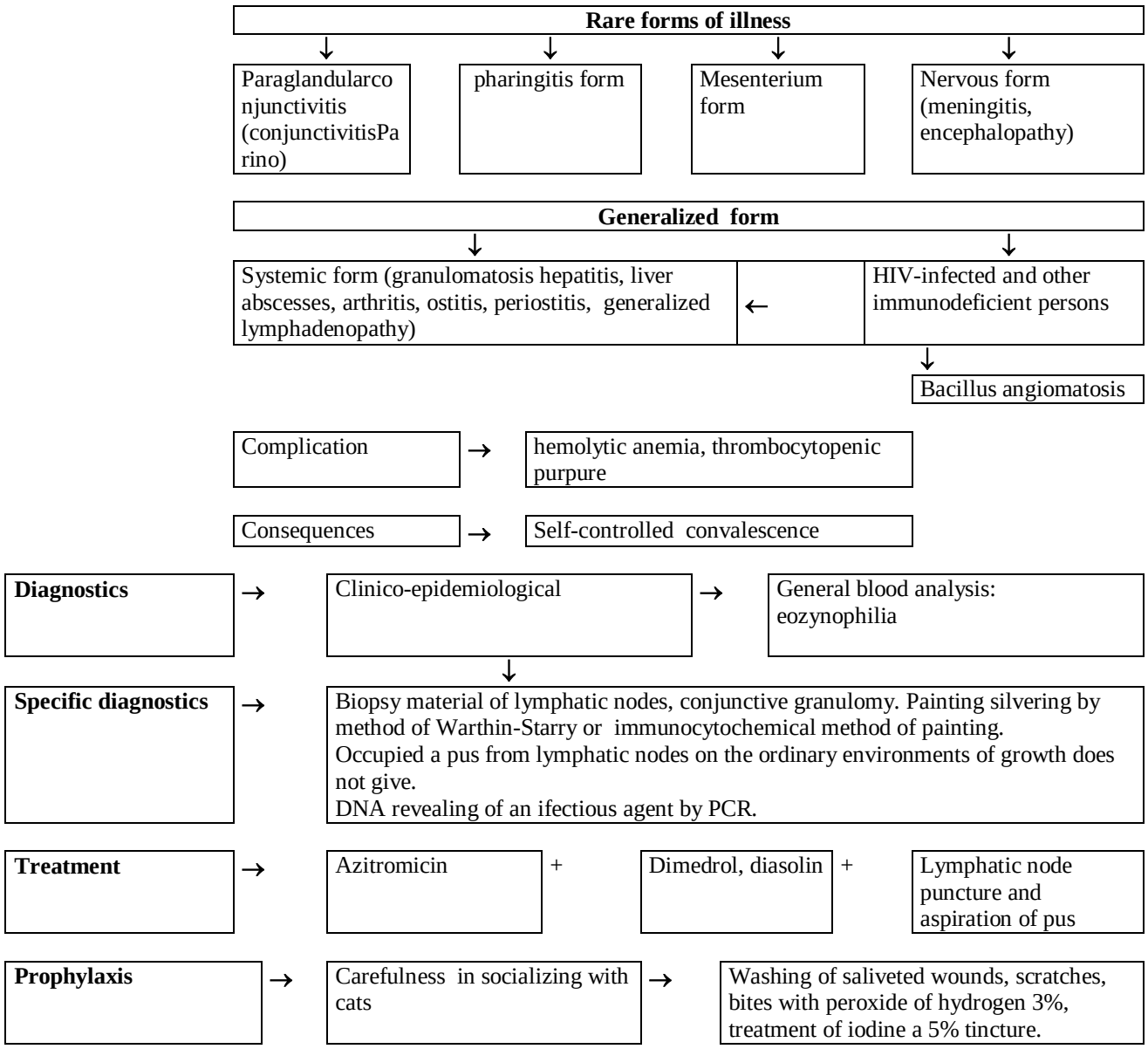
General forms: damage of liver, spleen, remote lymphatic nodes

Pathohystological change in lymphatic nodes
1. Reticulohystiocytic hyperplasia;
2. Granulomatosis;
3. Mikroabscessingduvannya;
4. Sclerosis of lymph nodes.

Clinic → Latent period from 3 to 20 days, average from 1 to 2 weeks → On the place of scratches which could heal over already, there are papules, pustule, scuttle → Regional lymphadenitis in 10-12 days after primary affect appearance

↓

lymphatic node increase during 2-3 weeks to 3-5 s-m, with hyper mated skin above, painful ← Suppuration of lymphatic node, with festering excretions ← Healing with formation of scars, lasts up to 24 months



3.4. Materials Self-control

3.4.1. Question for self-control **(α=2)**

1. Determination of concept “erysipelas”.
2. What group of microorganisms are the exciters of erysipelas and felinossi. Name these exciters.
3. Ways of agent penetration into a skin.
4. Pathogenesis of inflammatory process is derma while erysipelas.
5. Classification of erysipelas clinical forms.
6. Complication of erysipelas.

7. Differential signs of erysipelas and phlegm and other diseases.
8. Etiotropic therapy of erysipelas: medications, doses, duration of course.
9. Prophylaxis of relapses.
10. Clinical observation on reconverting persons and patients with recurrent erysipelas.
11. Source and pathogenesis of felinosis.
12. Classification of felinosis clinical forms.
13. Felinosis clinical symptoms and diagnoses.

3.4.2. Tests are for self-control

To choose right answers: **α=2**

1. What is the causative agent of erysipelas?

A. DNA-viruses

B. staphylococci

C. RNA-viruses

D. streptococci

E. pneumococci

2. In case of frequently recurrent erysipelas one prescribes:

A. penicillin in the quantity of 300,000 units for 5-7 days

B. penicillin in the quantity of 500,000 units for 3-5 days

C. sulfanilamide's in the dose of 1.0 g 6 times for 7 days

D. lincomicini hydrochlorides 30 %-2.0 ml in/m 3 times a day during 7 day

E. ampicillin in the dose of 1.0 g 6 times a day for 2 weeks

3. Erysipelas belongs to the following group of infections:

A. respiratory

B. intestinal

C. blood infections

D. infections of external integuments

E. zoogamous infection

4. A full-scaled picture of erysipelas includes:

A. ulcer with seropurulent discharge

B. carbuncle surrounded by jelly-like edema

C. erythematic, edema, pain

D. blood-filled pustules

E. dark-red papules

5. Patients suffering from erysipelas have the following changes in the blood

A. neutrophilic leukocytes and accelerated erythrocyte sedimentation rate (ESR)

B. leucopenia with lymphomonocytosis

C. leukocytes with lymphocytes

D. leucopenia and slowed-down ESR

E. leukocytosis with lymphomonocytic blood reaction

6. For primary erysipelas one prescribes:

A. tetracycline in the dose of 3.0 g 4 times a day

B. bycillin-3 in the dose of 1,000,000 units once a week

C. penicillin in the quantity of 1,000,000 units every 4 hours ad muscularly

D. chloramphenicol in the dose of 0.5 g 4 times a day

E. penicillin in the quantity of 200,000 units 6 times a day for 3 days

7. The agent of felinosis is:

A. Streptococcus pneumonias;

B. Spirocheta minus;

C. Bartonella hensela;

D. Clostridium perfringens;

E. Chlamydia pneumonias.

8. Felinosis latent period lasts:

A. to a few clock to the week;

B. 1-2 weeks;

C. 10-14 days;

D. more than 40 days;

E. 2-5 days.

9. Felinosis infection source is:

A. man;

B. cat;

C. wild animals;

D. birds;

E. rodents.

10. Felinosis is belonging to the group of infections:

A. zoonosis;

B. external covers;

C. intestinal;

D. respiratory tracts;

E. bloody.

STANDARDS OF FAITHFUL ANSWERS

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

Fill a table: epidemiology and clinical description of felinose

α=3

№	**Main criteria**	**Description of this criterion**:
1	**Epidemiological history**	Children are ill, which are played with cats
2	**Duration of latent period**	On the average to 1-2 weeks
3	**Clinical simptoms**	Scratch - papula - pustula – crust. There are primary affect. Regionary lymphadenitis - swallowing - suppuration - fistula - fever, intoxication, rash.
4	**Methods of specific diagnostics**	The indirect of imunofluorescans, skin allergic test, inoculation of pus into special nutritive environment, PCR. Biopsy matherial of lymph nodes, conjunctive granule and its painted by silvering by method of Warthin-Starry or immunocytochemical method.

3.4.3. Tasks are for self-control

Task 1 (α=2)

62 year old women fell ill acutely with the increasing of temperature up to 39,8^{0}C, a chill, intensive headache, dull ach in her body. She complained for increasing and acute pain in right inguinal area and swallowing of the right shin and skin hyperemia with clear borders.

1. Preliminary diagnosis.

2. Inspection plan.

3. Treatment plan.

Task 2 (α=2)

58 year old patient, who saffers-burst chronically thrombophlebitis of lower extremity, III-d stage obesity. Fever and skin rash appeared after the trauma of foot. Objective: temperature up to 38,8^{0}C, bright red crush skin, hot zone of hyperemia is dearly separated, boarded by no equal fire-like toughs. The inguinal nodes are middle increased.

1. Preliminary diagnosis.

2. Inspection plan.

3. Treatment plan.

Task 3 (α=3)

A veterinary doctor came to his family doctors complaining for considerable increasing and pain of left axillary's lymph nodes and suppurating fistula. Objective: lymph nodes are increased up to 4 cm, not joined. There is fistula in the projection of one of the node, it suppurates. Inoculation of pus into nutritive environment is
negative.

1. Preliminary diagnosis.

2. Inspection plan.

3. Treatment plan.

4. **MATERIALS FOR auditory independent work.****

4.1.The list of practical tasks to be resolved on practical lesson: 4.1. The list of practical tasks to be resolved on practical lesson:

- to master the techniques of examination patients with erysipelas, fellinosis
- to cure the patient with erysipelas
- to differentiate erysipelas with fellinosis and other skin diseases
- to create a plan of laboratory examination
- to interpret the results of specific examination patient with erysipelas
- to recognize erysipelas complications
- to create a plan of treatment for patients with erysipelas and fellinosis

- to prescribe curative drugs for patients with erysipelas and fellinosis
- to fill in medical documentation by fact of diagnosis determination “Erysipelas”, “Fellinosis”
- to resolve a situational tasks

4.2. The professional algorithm as for habits and skills forming in the diagnosis of erysipelas

№	The task	The order of fulfillment	Notes, warnings as for safe control
1.	To cure the patient with Erysipelas	I. Clarifying of patient’s complains	To separate complains, that characterizes main disease symptoms, accompaniment illnesses and complications, select the following syndromes: general intoxication, skin injury, changes of other organs and systems.
.		II. To take the anamnesis of: a) disease	To pay attention on: acute beginning of the disease, fever, headache, infirmity, vomiting, pain, erythematic, swelling.
		b) life	existence of otolaryngology chronic diseases, dermatomycosis, thrombophlebitis, burst disease, cardiovascular diseases, traumas and ulcers
		c) epidemiological	contact with patients with streptococci infection.

		III. To fulfill an objective examination - general status of a patient - skin - lymphatic system - Another organs and systems:	To pay attention on flaccidity, increasing of body temperature; erythema, it's borders, localization, swelling, pain, local temperature increasing, vesicle existence, bullas, hemorrhages; lymph stasis; regional lymphadenitis - existence of otolaryngology chronic focuses, fungous foot, diabetes mellitus, cardiovascular or renal pathology, hepatic cirrhosis. Remember: existence, expressiveness, symptom dynamics are caused by term and severe ness of its flow and depend on age and accompaniment illness of the patient.
2	To prescribe laboratory and accessory examinations, interpret it's	1. General blood examination. 2. Urinalysis. 3. Bacteriological examination.	Neutrophilic leukocytes, RBCS acceleration Without special changes. Inoculation from bullas, erosions β-

	results		hemolytic streptococcus revealing.

5. MATERIALS POST auditory independent work.

Subject of SEIW and SSIW:

Patients with erysipelas on background concomitant diseases

There is the health centre system of patients with often relapses of erysipelas

Felinosis is illness of cat-like scratch of –methods of clinical and laboratory diagnostics

TORCH infection

Actuality of theme:

TORCH (also known as TORCHS or TORCH infection) is a medical abbreviation for the first letters of a set of infectious diseases transmitted from the pregnant to the fetus (perinatal infections) that can lead to severe intestinal development or even fetal death. Infections included in the TORCH complex belong to a group of viral, bacterial infections, or infections caused by the simplest, often accessing the blood flow of the fetus through the chorionic villus. Vertical transmission can occur during any period of pregnancy or during childbirth.

The acronym "TORCH-complex" is explained as follows:

T - Toxoplasmosis (Toxoplasmosis);

O - Other infections (Other); They include HBV, infections caused by the Coxsac virus, syphilis, chickenpox, HIV infection and infection caused by parvovirus B19 (in pediatric practice known as' infectious erythema ',' fifth disease 'or syndrome' traces of slaps ");

R - Rubella (rubella);

C - Cytomegalovirus infection (CMV-infecion);

H - Diseases caused by a herpes simplex virus (He: pes simplex in¬fection).

Re**c**ognition of toxoplasmosis in pregnant women is important because of the risk of transmission to the fetus. This risk is limited almost exclusively to those fetuses whose mother acquire the infection for the first time during gestation. The earlier in gestation a mother is infected with T.gondii, the more severe is the disease in the fetus and newborn, despite the lower frequency with which transmission occurs with shorter period of gestation. Finding include chorioretinitis with blindness, epilepsy, psychomotor disorders and developmental delay, hearing loss, jaundice, rash, hematologic abnormalities and pneumonia. The classic triad of hydrocephalus, chorioretinitis, and cerebral calcification is seen rarely.

Inbred toxoplasmosis arises up in connection with the presence of toxoplasma of certain invasion for mothers. From data of Institute of pediatrics, obstetrics and gynecology of AMS of Ukraine spontaneous abortions took place in 50,5% women from

toxoplasmosis, artificial abortions on call of pregnant - in 19,5%. Deadborns comprises 22% from the number of births. While contamination with nfection of toxoplasmosis pregnant women toxoplasma gets to the fetus in 66% cases.

Toxoplasmosis is dangerous especially for immunocompromised patients: uncontrolled reproduction of toxoplasma can develop defeat of organs, that can lead even to death. Toxoplasmosis is a parasitic disease characterized mainly by a latent or chronic course, by damage to the nervous system, the organs of the reticuloendothelial system, muscles, myocardium and eyes.

Etiology: the simplest Toxoplasma gondii.

Epidemiology: zoonosis with natural foci; intermediate hosts - a man and a number of animals, the final - domestic cats and some wild representatives of the cat family (lynx, cougar, jaguar). Infection occurs when eating raw meat (swallowing cysts contained in meat or oocysts, with which it can be seeded), through infected blood and transplacental.

Pathogenesis: the gate of infection - often the digestive system. The introduction of the pathogen occurs in the lower parts of the small intestine, then with the current of the toxoplasma lymph reach the regional (mesenteric) ls, where inflammatory changes develop with the formation of infectious granulomas. Then toxoplasma enters the blood, spread throughout the body and is fixed in various organs and tissues (liver, spleen, lymph nodes, nervous system, eyes, myocardium, skeletal muscles). In these organs, clusters of parasites are formed in the form of cysts, which can persist in the body for dozens of years and even for life. In places of fixation, inflammatory foci appear, and in some organs (nervous system, skeletal muscles) - necrosis foci, in which calcium salts are then deposited and calcints formed. As a result of the vital activity of the parasite and the release of antigens and allergens, an allergic alteration of the organism occurs (according to the type of hypersensitivity reactions of a delayed type) and antibodies are produced.

Classification of toxoplasmosis:

a) latent (primary and secondary)

b) primary and secondary chronic

c) acute toxoplasmosis

Clinic: incubation period about 2 weeks.

Primary latent toxoplasmosis - primary infection leads to the production of specific antibodies and the formation of non-sterile immunity without any clinical manifestations of the disease.

Acute toxoplasmosis - begins violently, often proceeds according to the type of meningitis (meningoencephalitis, encephalitis), with the development of neuritis of optic nerves, paresis, hemiplegia, after 4-5 weeks - myocarditis. In a number of cases, a typhoid-like form of acute toxoplasmosis with a characteristic exanthema appears on the 4th-7th day (abundant, macular, from pink to dark red) without affecting the central nervous system, although the combination of symptoms (mixed form) is more common.

With the reactivation of a latent infection or against a background of clinically pronounced chronic toxoplasmosis, acute toxoplasmosis begins gradually and is characterized by a CNS lesion of the type of meningoencephalitis that progresses slowly with the gradual involvement of the cranial (more often visual) nerves and myocardium into the pathological process. Later, CT scans can detect cysts in the brain substance. Simultaneously, against the backdrop of the dominant picture of neuroinfection, other signs of toxoplasmosis are revealed: poly-lymphadenitis, hepatolienal syndrome, arthralgia and myalgia.

In both cases with spinal puncture, the cerebrospinal fluid flows under increased pressure, cytosis is lymphocytic, low, IgM can be detected to T. gondii, and in the color of the smear obtained from the centrifuged CSF, according to Romanovsky, trophozoites of toxoplasm.

Acute toxoplasmosis is severe and may be fatal. In the case of recovery, there remain residual phenomena of varying severity (atrophy of the optic nerves, diencephalic disorders, epileptiform seizures, intracranial hypertension, sluggish arachnoiditis, foci of chorioretinitis with visual impairment, calcifications), with timely diagnosis and adequate treatment, complete clinical recovery with the formation of a second latent forms of toxoplasmosis.

The more frequent and less favorable outcome of acute toxoplasmosis is the formation of secondary chronic toxoplasmosis, which also proceeds as primary chronic and is characterized by almost annual exacerbations, rare and short-term remissions, the

presence of residual phenomena and low effectiveness of anti-infective therapy.

Primary chronic toxoplasmosis is characterized by a pronounced polymorphism of clinical manifestations in the absence of pathognomonic symptoms.

The disease begins gradually, the most common signs of general intoxication, lesions of the central nervous system, cardiovascular system, lymphadenopathy, enlargement of the liver and / or spleen, dysfunction of the vegetative nervous system, defeat of the musculoskeletal system and the gastrointestinal tract.

Patients complain of general weakness, headache, adynamy, deterioration of appetite, sleep disturbance, sometimes weight loss. Psychoemotional instability, memory loss, mental performance, neurotic-like functional disorders (phobias, affective disorders, asthenic syndrome) are often observed. The most constant sign is an increase in body temperature to subfebrile digits. Quite often the subfebrile condition lasts for many months, sometimes it is wavy, in women it can be associated with the menstrual cycle. Generalized lymphadenopathy is noted. Peripheral lu are enlarged moderately (1-3 cm in diameter), often sensitive or painful on palpation, in about half of patients mesenteric nodules are involved in the process. Patients complain of pain in the muscles and joints. During the examination, myositis is detected (especially often the muscles of the legs), sometimes with the development of calcifications in the muscles. Radiographically, dystrophic changes in the small joints of the hand can be detected. Patients are often concerned about heartbeat, dull pressing pains in the heart, violation of the rhythm of heartbeats. Objectively noted tachycardia, sometimes extrasystoles, lowering blood pressure, widening the boundaries of the heart, muffling the tone, ECG - conduction disorders, focal and diffuse muscular changes, heart rhythm disturbances. Changes in the respiratory system. Patients note dull pain in the epigastric region, bloating, stool retardation, the phenomena of spastic colitis are characteristic. The defeat of the vegetative and peripheral parts of the nervous system is manifested by acrocyanosis, marbling of the skin, hyperhidrosis, plexitis, changes in the parameters of the ortho- and clinostatic test.

Often there are lesions of the organ of vision in the form of chorioretinitis (central, bilateral). With prolonged course of chronic toxoplasmosis, secondary hormonal

insufficiency may develop with the formation of habitual miscarriages of pregnancy, secondary infertility.

General clinical indicators of blood are poorly informative. Somewhat more often there is normocytosis and mild leukopenia with relative lymphocytosis. Acute phase reactions are negative, ESR - within normal limits.

Chronic toxoplasmosis proceeds wavy. The periods of exacerbation are replaced by short-term remissions, during which the clinical manifestations of the illness subsided, and the working capacity of the patients improves, not reaching the level of healthy individuals.

Latent forms of toxoplasmosis are characterized by the fact that even with a thorough clinical examination of the patient, signs of toxoplasmosis can not be detected. They are diagnosed only with the help of serological reactions or an intradermal test with toxoplasmin.

There is a secondary latent form of toxoplasmosis in people who previously had manifest forms of toxoplasmosis, and primary latent in those cases when the latent form developed without any clinical manifestations of the disease. With the secondary-latent forms, there is an easy onset of exacerbation (with intercurrent diseases, pregnancy, drugs suppressing immunity, and especially in HIV-infected individuals). With the primary-latent form of exacerbations under normal conditions, there is almost no, although a sharp decrease in the protective reactions of the organism can also lead to the transition of the disease to clinically expressed and even generalized (acute) forms.

Congenital toxoplasmosis manifests itself as a picture of neuroinfection accompanied by poly-lymphadenitis (especially mesadenitis), hepatolienna syndrome, arthralgia, myalgia, optic nerve damage, myocarditis development;

Diagnosis: clinic, detection of IgG and IgM in serological reactions, dynamic observation of their titer, setting of intradermal test with toxoplasmin, PCR.

One can confidently talk about infection during pregnancy in the presence of seroconversion, double and more growth of IgG titers during the reaction with an interval of 2-3 weeks and the presence of epidemiological prerequisites for a "fresh" infection, the detection of trophozoites of toxoplasm in biological fluids by PCR

Treatment. In the treatment of acute forms of toxoplasmosis, a combination of two or three ant-and protozoal drugs is used. Adults prescribe chloridine (synonyms: tindurine, pyrimethamine, daraprim) in the first two days of 100-200 mg in two doses, then 75 mg / day + sulfanilamide short-acting drug (sulfadimezin) 2 -4 g / day + tetracycline 1,2 g / day until the 10th day of apyrexia (or t <5 ° C). Every ten days, one or two preparations are changed (it is possible to prescribe delagil 250 mg 3 times / day, clindamycin 450 mg 3 times / day, spiramycin 1 g 3 times / day, trichopolum 0.25 g 4 times / day). Necessarily the appointment of glucocorticoids (predn and zolon 20-40 mg / day per os 12-15 days). The remaining subfebrile condition is not a contradiction for discharge from the hospital. Contraindications to the appointment of daraprima - hypersensitivity, megaloblastic anemia. Adverse reactions - headache, arrhythmia, anorexia, vomiting, diarrhea, leukopenia, thrombocytopenia. Produced in slabs of 0.025 g.

In children, it is preferable to use mordidine 1 mg / kg of body weight per day in two divided doses + bac-trim -120 per 1/2 tablets per 1 kg of body weight per day for 2 doses + calcium folinate 2 mg / day + pre-nisolone 1, 5 mg / kg per day for 2 doses (morning and evening) until the protein in the liquor decreases to less than 1 g / l. Duration of treatment as in adults.

Biseptolum (Biseptolum; synonyms: Co-trimoxazole, Bactrim, Berlocid, Oribact, Oriprim, Se-p-trin) is less active (but less toxic). Contraindications - a violation of liver and kidney function, pregnancy, hypersensitivity. Adverse reactions - vomiting, allergic reactions. Produced in tablets. The adult tablet contains 0.08 g of tri-metoprime and 0.4 g of sulfamethoxazole, in one tablet for children - 0.02 and 0.1 g, respectively.

With chronic forms of toxoplasmosis, complex therapy gives good results (after one course, a stable recovery occurs in 80-90% of patients).

Initially, a course of treatment (7-10 days) is prescribed for any of the etiotropic drugs given above, in combination with nonspecific desensitizing therapy. To this end, use antihistamines (diprazine, dimedrol, suprastin, etc.) or corticosteroids. Prescribe also vitamins, fortifying agents, leukopoiesis stimulants (with leukopenia). In the future, toxoplasminotherapy (vaccine therapy) is carried out. As a vaccine, toxoplasmin is used,

which is based on its ability to stimulate a long-term specific immune response by cellular type.

First, the working dilution is determined (the minimum dilution in the titration sample, to which causes a small skin reaction). To treat toxoplazmin (at the selected dilution) intradermally on the 1st day to 0.1 ml in 3 places, on the second day - 4 injections of 0.1 ml at 4 points, then every day, adding one injection, reach up to 10 injections (on the 8th day of treatment). During the treatment with toxoplasmin, patients are prescribed a general ul-ra-violet irradiation daily, beginning with 1/4 of the bio-dose and bringing up to one biodose. The introduction of o-plasmin toxin results in a specific desensitization and stimulates protective immune.

Long-term treatment of patients with chronic toxoplasmosis chemopreparations (more than 10 days) is not justified, since the pathogen predominantly localizes within cells or cysts and is practically inaccessible to antiprotozoal drugs.

Conduct treatment of concomitant diseases, sanation of foci of a chronic infection, which can have an adverse effect on the state of the immune system. With arthralgia i-ya and myositis, physiotherapy is used. The average duration of treatment is about 3 weeks. The practical methods of treatment of pregnant women are of great practical importance. E c-whether the infection with toxoplasmosis occurred before pregnancy and there are no clinical manifestations of the disease, treatment is not carried out. With pronounced manifestations, it is possible to confine oneself to general c-replenishing treatment and vaccine therapy. If the infection occurred early in pregnancy (the appearance of protivoktoksoplazmoznyh antibodies IgM, the growth of antibody titers, the transition of negative serological reactions into positive), then there is a risk of developing severe congenital toxoplasmosis. In such cases, rovamycin is recommended for 3 million ED / day for 4-6 weeks, starting from the 18th week of pregnancy (the drug does not have a teratogenic effect, is well tolerated) against the background of taking eubiotics.

The prognosis with congenital toxoplasmosis is serious, as the consequences of his stance. The lethally acute forms of toxoplasmosis in people with impaired immunity (AIDS patients, etc.) are very difficult and may end. In chronic forms, the prognosis is favorable, although some may experience recurrence of the disease.

Prevention and activities in the outbreak. Limitation of contact with infected cats, compliance with personal hygiene rules. Prohibition of consumption (testing) of raw meat with minced meat, as well as meat dishes without sufficient heat treatment. Prevention of infection of women during pregnancy (avoid contact with cats and testing raw ground beef, wash hands after preparing dishes from raw meat, etc.). There are no profiles of events in the outbreak.

2.Whole lessons (with pointing of level of mastering which is planned):

2.1.Student must know: ***à-2***

- etiology of toxoplasmosis, specific of different germ forms;
- specific of toxoplasma life cycle;
- epidemiology of toxoplasmosis;
- pathogenesis of disease;
- clinical classification of toxoplasmosis;
- variants of toxoplasmosis clinical displays whyle different forms of infectious process;
- specific of toxoplasmosis course in pregnant and inbred toxoplasmosis;
- specific of toxoplasmosis course in HIV-infected patients;
- complications and consequences of toxoplasmosis;
- laboratory diagnosis of toxoplasmosis;
- differential diagnosis of toxoplasmosis with other diseases;
- treatment principles of patients with toxoplasmosis;
- principles of disease prophylaxis.

2.2. A student must be able: à-3

· to gather anamnesis of illness with the estimation of epidemiology information;

· to inspect a patient and find out basic symptoms and syndromes of toxoplasmosis;

· to substantiate clinical diagnosis for on time referral of patient in the hospital;

· to differentiate toxoplasmosis;

· to fill in a medical document by fact of “toxoplasmosis” previous diagnosis indication (an urgent report for the epidemiology department of SES);

· to work out a plan of laboratory and additional examination of patient;

· to interpret the results of laboratory inspection for determination of process activity and determination of necessity in etiology treatment prescription

· to work out an individual plan of treatment taking into consideration epidemiology data, process activity, presence of complications, severity of the state, allergy anamnesis, concomitant pathology;

· to work out a plan of epidemiological and prophylactic measures in the focus of infection;

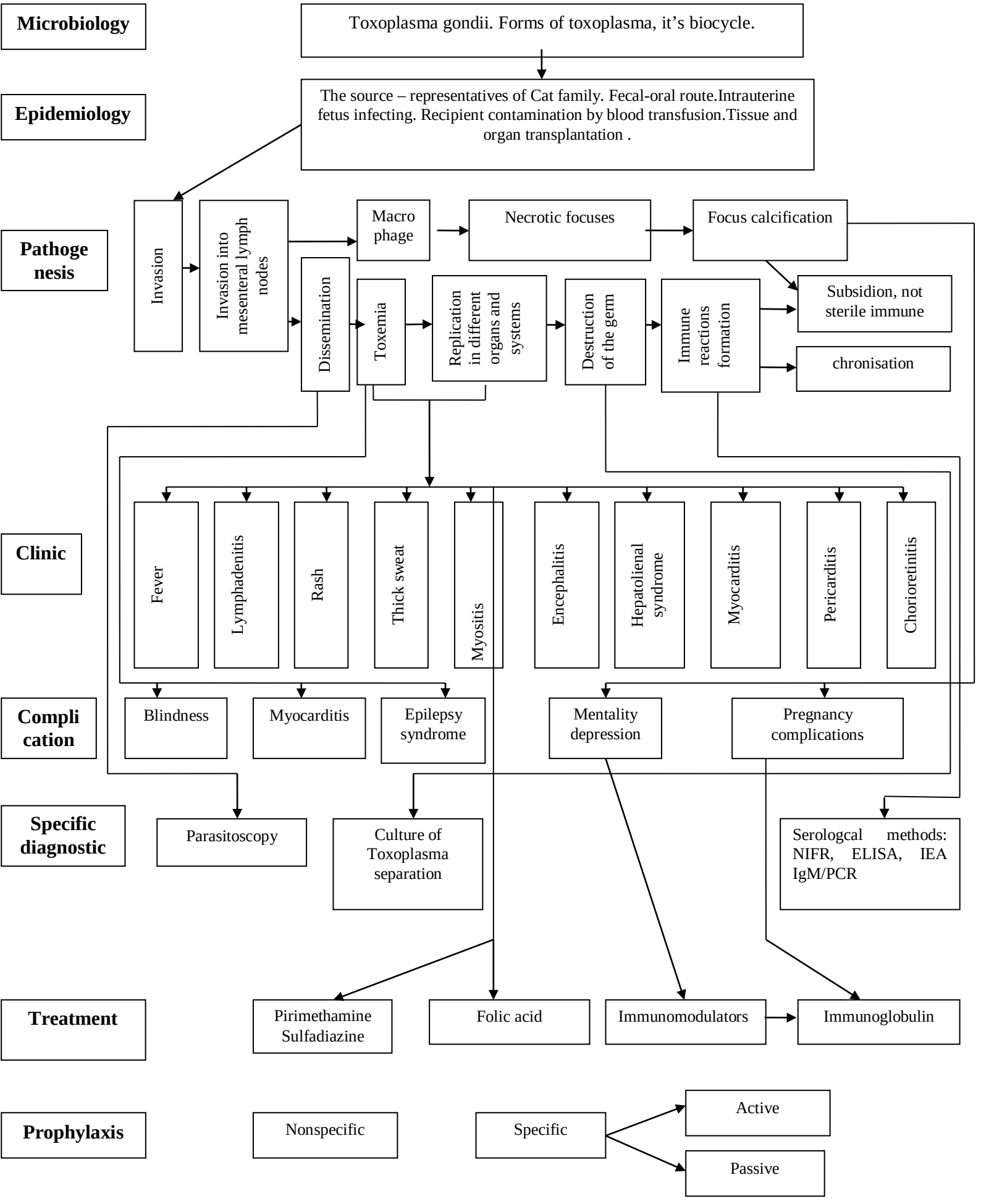
3. Materials of independent work.

3.1.Basic knowledge, ability, skills, necessary for a study themes

Discipline	**To know**	**To be able**
Previous disciplines		
Microbiology	Properties of Toxoplasma gondii, of specific methods of toxoplasmosis diagnostics	To interpret the results of specific methods of toxoplasmosis diagnostics
Physiology	Parameters of human physiology norms of organs and systems,normal indexes of laboratory examination (general blood picture, urinalysis, biochemistry of blood, electrolytes and others like that)	To estimate information of laboratory inspection.
Patophysiology	Violation mechanism of organ’s functions and	To interpret pathological changes as a result of

	systems at different genesis pathological states.	laboratory inspection at violations of function of organs and systems of different genesis.
Immunology and allergology	Basic concepts of object, role of immune system while toxoplasmosis, influence on term of elimination of the germ from human organism. Immunological aspects of toxoplasmosis chronic forms .	To estimate information of immunological researches.
Epidemiology	Epidemiologycal process (source, mechanism of infection, ways of transmission) of toksoplazmosis; prevalence of pathology in Ukraine and all over the world	To collect epidemiology anamnesis, to fulfill anti epydemy and prophylactic measures in the focus of infection.
Neurology	Pathogenesis, clinical signs of toxoplasmos meningoencephalitis	To fulfill clinical inspection of patient with the affection of nervous system.
Ophthalmology	Pathogenesis, clinical signs of toxoplasmos chorioretinitis	To fulfill clinical inspection of patient with affection of eyes.
Obstetrics-gynecology	Clinics-laboratory signs of toxoplasmosis of pregnant, inbred toxoplasmosis	On time iagnostic of toxoplasmosis at pregnancy and in neonatal period
Internal diseases	Methods and basic stages of patient's clinical inspection	To collect anamnesis, fulfill clinical inspection of patient, find out pathological symptoms and syndromes. To analyse datas.
Clinical pharmacology	Pharmacokinetics and pharmacodynamics, side effects of antiprotozoal drug preparation, sulfanilamides, antibiotics, possibilities of nosotropic therapy	To prescribe treatment depending on age, individual features of patient, activity of process. To choose the optimum mode of reception and dose of preparation, write recipes.

Torch-infection. Toxoplasmosis.



6 Materials are for self-control

6.1. A question is for self-control

1. What group of infectious diseases does toksoplasmosis belong according to the source of infection?Ways of toxoplasmosis transmission.
2. Biocycle of Toxoplasma gondii.
3. Pathogenesis of toxoplasmosis.
4. Clinical classification of toxoplasmosis.
5. Basic symptoms of aquired toxoplasmosis (acute, second-latent, primary chronic and the second-chronic toksoplasmosis).
6. Specific of toxoplasmosis in HIV-infected persons.
7. Specific of inbred toxoplasmosis.
8. Features of motion of newborn toxoplasmosis.
9. Specific of toxoplasmosis in ptregnant.
10. Complications of toxoplasmosis.
11. Consequences of toxoplasmosis.
12. Additional methods of inspections at toxoplasmosis.
13. Specific diagnostics of toxoplasmosis.
14. Estimation of laboratory indexes patients with toxoplasmosis.
15. Diagnosis substantiation of toxoplasmosis different forms.
16. Differential diagnostics with other infectious diseases.
17. Treatment principles of patients with toxoplasmosis.
18. Patogenic therapy of toxoplassmosis.
19. Specialities of toxoplasmosis treatment in pregnant.
20. Treatment of patients is with inbred toxoplasmosis.
21. Specific of treatment HIV-infected patients with tosoplasmosis.
22. Prophylaxis of toxoplasmosis.

6.2. Tests are for self-control

To choose right answers: **a=2**

1) The causative agent of toxoplasmosis is: A. Toxoplasma gondii B. Trichinella spiralis C. Trichocephalus trichiurus D. Brucella ovis E. Bacillus antracis

2) The final host with toxoplasmosis are: A. cats B. pigs C. diseased person D. rodents E. environmental objects

3) Intermediate host with toxoplasmosis are: A. birds B. rodents C. cattle D. dogs E. all true

4) Transmission routes of toxoplasmosis are all except: A. transmissible B. food C. transplacental D. percutaneous E. hemotransfusion

5) Transplacental infection with toxoplasmosis occurs in the case of: A. primary-chronic toxoplasmosis B. secondary-chronic toxoplasmosis C. infection during pregnancy D. infection 3 months before pregnancy E. infection 6 months before pregnancy

6) In toxoplasmosis, organs and systems are affected all but: A. bone marrow B. eyes C. skeletal muscles D. lungs E. central nervous system

7) Acute acquired toxoplasmosis is characterized by all the symptoms except: A. diarrhea B. fever C. exanthema D. meningeal symptoms of E. lymphadenopathy

8) For chronic acquired toxoplasmosis is characteristic: A. hydrocephalus B. convulsive syndrome C. calcification in the brain D. chorioretinitis E. all true

9) Malformations of the fetus develop when infected with toxoplasmosis: A. in the first trimester of pregnancy B. in the second trimester of pregnancy C. in the third trimester of pregnancy D. infection 3 months before pregnancy E. malformations are not characteristic

10) For the treatment of acute toxoplasmosis, pregnant women are prescribed: A. ceftriaxone B. spiramycin C. biseptol D. metronidazole E. doxycycline

11) Which groups of lymph nodes increase with toxoplasmosis: A. occipital B. cervical C. axillary D. inguinal E. all true

12) Which way is the most common infection of a person with toxoplasmosis: A. when using raw minced meat B. when raw water is used from unknown water bodies C. by airborne droplets D. when in contact with a sick person E. all right

13) In what color is the protoplasm of toxoplasma stained using the Romanovsky-Giemsa method: A. yellow B. red C. blue D. ruby red E. pink

14) A positive intradermal test with toxoplasmin indicates: A. the infection that just occurred B. about the presence of acute toxoplasmosis C. about the presence of congenital toxoplasmosis D. about the presence of chronic toxoplasmosis E. about the presence of sensitization to the antigen of toxoplasm

15) For the purpose of etiotropic therapy, patients with toxoplasmosis can be appointed: A. fanzidar B. furazolidone C. finlepsin D. ofloxacin E. Biltricidum

16)What drug should be administered concomitantly with etiotropic therapy with toxoplasmosis: A. ascorbic acid B. iron preparations C. cyanocobalamid D. folic acid E. vitamins of group B

17) In antropurgic foci the source of infection in toxoplasmosis are: A. cats B. man C. chickens D. ducks E. pigs

18) Where does the maturation of toxoplasmosis oocysts: A. in the human body B. in water C. in soil D. on food E. in feces

19) To determine the activity of the process, A. can be used to determine specific IgM B. Determination of specific IgG C. Reaction of Paul-Bunel D. Widal's reaction E. Wright's reaction

20) The sexless cycle of development of toxoplasm in the animal body is called: A. gametogony B. sporogony C. Schizogonia D. zigothonia E. all right

21) Maturation and formation of invasive forms of toxoplasm are: A. schizogonia B. endodiogeny C. gametogonia D. sporogonia E. all right

22) The gametogony occurs in the body: A. human B. mice C. young cats D. cattle E. geese

23) A characteristic sign of eye damage in chronic acquired toxoplasmosis are: A. conjunctivitis B. iridocyclitis C. creeping corneal ulcer D. glaucoma E. chorioretinitis

24) How clinical symptomatology is characteristic of the secondary latent form of toxoplasmosis: A. subfebrileity B. calcifications C. sclerogenous lymph nodes D. old foci of chorioretinitis E. all true

25) What dose of toxoplasmamine is administered with the intradermal sample: A. 0.05 ml B. 0.5 ml C. 5 ml D. 0.1 ml E. 1.0 ml

26) Herpes simplex viruses of the 1st and 2nd types belong to the subfamily: A. α-herpesviruses B. β-herpesviruses C. γ-herpesviruses D. influenza viruses E). All right

27) Patients with chickenpox are isolated at home: A. for 6-8 hours B. for 1-2 days C. for 3-4 days D. for 5 days from the moment of the last rashes E. for 8 days.

28) Specify the time of follow-up for contacts with chickenpox: A.5 days B. 14 days C. 21 days D. 45 days E. 180 days

29) The drugs of choice for the treatment of CMV infection are: A. acyclovir B. ganciclovir C. interferon D. ribavirin E. albendazole

30) The main element of the rash in herpetic infection: A. papula B. pustula C. vesicle D. roseola E. petechia

STANDARDS OF FAITHFUL ANSWERS

1.A 2. A 3. E 4. A 5. C 6. D 7. A 8. E 9. A 10. B 11. E 12. A 13. C 14. E 15. A 16. D 17. A 18. C 19. A 20. C 21. D 22. C 23. E 24. E 25 D 26. A 27. D 28. C 29. B 30.C

Presence of clinical symptomatic of toxoplasmosis different forms:

Form\ Symptom	Acute aquired	Chronic aquired	Acute inbred	Chronic inbred	Latent
Acute beginning	+	-	+	-	-
Myalgia	+	+	+	+	-
Myocarditis	+	+	-	+	+\-
Chorioretinitis	-	+	-	+	+\-
Polylymphoadenopathy	+	+	+	+	+
Hepatosplenomegaly	+	+	+	+	+\-
Endocrine system affect	-	+	-	+	-
Encephalitis	+	+\-	+	+\-	-

6.3. Tasks are for self-control

Task 1 **a=2**

23 year old patient referred to an infectiologst with complaints for subfebril temperature of body during the last 2 months, general weakness, irritability, headache, sight worsening. While examining: temperature 37,5, small neck, inguinal lymph nodes are palpated. Tones of heart are hollowed, tachycardia up to 90 shots for a minute, hepatosplenomegaly.1. What additional information of epidemiology anamnesis is necesarry?2.Formulate a previous diagnosis.3. Plan of patient's inspection.

Task 2 **a=2**

28 year old sick woman referred to the reception department of infectious hospital complaining for general weakness, periodic increasing of body temperature up to 37,3-37,4 , insignificant headache. While examining : polylimphoadenopathy, hepatosplenomegaly, tachycardia. By means of IEA method increased levels of IgM are found.1. Formulate a diagnosis. 2. Plan of patient's inspection. 3. Plan of patient's treatment.

Task 3 **a=3**

14 year old patient entered an infectious hospital with complains for head ache, increasing of body temperature up to subfebril , attacks of cramps which became more frequent lately. She is ill during last 4 years. While examining: all groups lymphoadenopathy. Heart tones are hollowed, HBF- 100 beats a minute. The lower edge of liver comes out from under the costal edge on 5 cm, lower edge of spleen - on 1 cm. A Nose-lip fold is smoothed on the left. Reflexes of tendons are alive, there are no pathological reflexes. Attacks of cramps happen for 2-3 times per hour. Consciousness during attacks is stored KGR with toksoplazma antigen is positive.

1. To propose a previous diagnosis.2. Additional methods of inspection3. To work out a plan of treatment

7. Materials are for audience independent work.**

7.1. List of educational practical tasks which must be executed on practical employment:

- To lay hands on the method of inspection sick on toxoplasmosis
- To conduct examinations of patient with toxoplasmosis
- To conduct differential diagnostics of toxoplasmosis
- To work out a plan of laboratory examinations
- To interpret the results of specific examinations of patient with toxoplasmosis
- To work out a plan of treatment sick on toxoplasmosis
- To define medical tactic in the case of toxoplasmosis for pregnant.

7.2. The professional algorithm as for habits and skills forming in the diagnosis of toxoplasmosis:

№	The task	Sequence of fulfillment	Remarks, warnings as for self control
1	To manage the methodic of clinical examination of patient To cure a patient	I. Clarifying of patient's complains	To separate complains, that characterize main disease symptoms: general intoxication, organs affects.
		II. To gather the anamnesis of: a) disease	To pay attention on gradual beginning, term, sequence of arising, dynamics of • fever • expressed sweating • sleep disordes • lymphoadenopathy • rash • other symptoms
		b) life	To reveal past diseases and vaccination anamnesis.
		c) epidemiological	To reveal information as for fecal-oral route of contamination, to pay attention on cat pets existence at home.
		III. To fulfill an objective	Remember: presence, expressiveness, dynamics of symptoms are caused by term and activity of disease flow, depend on the age, accompaniment pathology of a patient.

		examination 1. general status 2. skin, mucous membranes, lymphatic nodes 3. digestive system: - tounge exam - percussion of abdomen - palpation of abdomen 4. Cardiovascular system: - pulse - heart auscultation 5. Respiratory system: - lungs auscultation 6. Nervous system 7. Eyeground	To pay attention on: • infirmity, adynamia • body temperature • pale skin • maculo-papulous rash • polylymphoadenopathy To pay attention on: • lasting subfebril temperatute • hepatolienal syndrome To pay attention on: • unstable arterial pressure • bland hollowness of heart tones To pay attention on: • signs of bronchitis in some patients Signs of meningoencephalitis. Signs of chorioretinitis.
2	To prescribe laboratory and accessory examinations, interpret it's results	1. General blood examination. 2. Urinalysis. 3. Ultrasound diagnostics of abdomen organs 4. Parasitoscopy. 5. Intracutaneous test 6. Serological methods: IEA (IgM) 7. PCR	To pay attention on typical changes: normo- or basocytopenia, lymphomonocytosis. Absence of considerable changes at typical flow. Hepatolienal syndrome. Tachyzoites can be possibly found in biological liquids. Becomes positive from 4-6 week of infecting Appear from the first week of disease Toxoplasmosis DNA revealing in blood and other biological liquids.

8. Materials of of independent work.

Subject of UDRS and NDRS:

· The pregnant have features of diagnostics of tosoplasmosis

· Modern methods of specific diagnostics of toxoplasmosis

- Features of motion of toxoplasmosis are at the purchased immunodeficit
- Modern principles of treatment of patients with toksoplasmosis

Medicamentous disease (anaphylactic shock, serum sickness, Lyell's syndrome, Stevens-Johnson syndrome). Dysbiosis of intestine.

1. Actuality of theme: In recent decades, side effects of drugs remains a pressing problem as clinical medicine in general and the practice of doctors, infectious disease in particular. With thousands of drugs currently used can not be called any drug which, together with the therapeutic effect would not have side effects varying degrees of severity. Therefore, clinicians in different specialties often meet with patients, where complications of pharmacotherapy were either a history or developing during treatment. Avalanche-like growth side effects of pharmacotherapy promotes the widespread use of drugs, not always for the doctor. Contribute allergysation population varied nutritional supplements, antibiotics and hormones in food.
From 2001 to 2006, pharmacovigilance department of the State Pharmacological Center MoH Ukraine received 8220 spontaneous reports side effects of drugs, which often occur when using cardiovascular (26%) and antimicrobial (21%). However, these official statistics are very incomplete, since the real distribution of complications of drug use is unknown.

ANAPHYLACTIC SHOCK

Anaphylactic shock (AS) is a type of allergic reaction of the immediate type that occurs when the allergen is reintroduced into the body, characterized by rapidly developing common manifestations: a decrease in blood pressure, body temperature, blood coagulability, CNS disorder, increased vascular permeability and spasm of smooth muscle organs.

AS can develop with the introduction of any drug into the body. Most often, shock reactions occur on the introduction of sera, immunoglobulins, plasma proteins, polypeptide hormones (ACTH, insulin), penicillin and other drugs. The frequency and timing of the development of AS is influenced by the way the allergen is introduced into the body. With parenteral administration, it is observed more often and develops within an hour. However, anaphylactic shock can develop later, 1 -3 hours after contact with the allergen, as it is absorbed. The incidence of ASH increases with age. This is associated

with an increase in sensitization as various agents react. With age, it proceeds more severely, as the compensatory possibilities of the organism decrease.

The dose of the allergen in the development of shock does not matter.

The basis of the pathogenesis of AS is the response mechanism. In response to the ingestion of an allergen, reactants are formed (Ig E, Ig G). They are fixed on mast cells (labrocytes) and basophils, thereby creating a state of sensitization. Repeated exposure to the same allergen leads to its combination with the formed responses, which causes the release of mediators. As a result, the vascular tone falls and collapse develops. The permeability of the suction vessels of the microcirculatory bed increases, which contributes to the release of the liquid part of the blood into the tissues and the thickening of the blood. As a result, the BCC decreases. Usually, the patient comes out of shock himself or with medical help. With insufficient homeostatic mechanisms, the process progresses, metabolic disturbances in tissues associated with hypoxia are added.

It is necessary to remember the possibility of pseudoallergia. A number of drugs, including blood substitutes, γ-globulins, either cause direct release of histamine and some other mediators from the lambrocites and basophils, or include an alternative pathway of complement activation with the formation of its active fragments, some of which also stimulate the release of mediators from mast cells . In protein preparations, aggregation of molecules can occur. These aggregated complexes can cause an immunocomplex type of injury. Unlike anaphylactic shock, it is called anaphylactoid.

CLINIC

Clinical manifestations depend on the form of anaphylactic shock.

The fulminant form develops 1 to 2 minutes after the administration of the allergen. The patient loses consciousness, cramps appear, the pupils dilate. Skin pale or cyanotic, cold. Breathing becomes difficult, agonal. A sharp drop in blood pressure, pulse on the radial arteries is not determined. This form ends with death within 8 - 10 minutes.

The severe form develops after 5 to 7 minutes. The patient has a feeling of heat, lack of air, headache, pain in the heart, the fear of death. The patient quickly loses consciousness. The rest of the clinic is similar to a lightning-fast form. The prognosis is serious: when there is no help, death occurs.

The form of moderate severity develops 30 minutes after the allergen is administered. In this case, allergic rashes appear on the skin. The clinical symptomatology is very diverse and depends on the variant:

1) cardiogenic (most frequent) - pain in the heart. Feeling of heat, blood pressure decreases, tachi, cardiac arrhythmia, skin becomes marble;

2) asthmatic (asphyxia) - bronchospastic syndrome or laryngeal edema;

3) cerebral - a strong headache, psychomotor agitation, loss of consciousness, ship;

4) abdominal - severe pain in the abdomen, vomiting, diarrhea.ЛЕЧЕНИЕ

The outcome of anaphylactic shock depends on timely and adequate therapy.

With a lightning-fast form, treatment begins with resuscitation - indirect cardiac massage and artificial ventilation.

First of all, it is necessary to stop further supply of the allergen:

 Place a tourniquet above the injection site;

 put ice on the injection site;

 cut off the injection site with a solution of adrenaline (0.1% - 1 ml diluted in 10 ml of sodium chloride saline).

The most effective means for arresting anaphylactic shock are adrenaline, norepinephrine and mesethon.

Adrenaline (0.1% - 1 ml) is administered intravenously, intramuscularly or subcutaneously, depending on the shape of the shock. With the preservation of low blood pressure, adrenaline can be repeated after 15 minutes.

If there is no effect, infusion therapy is necessary. First, an isotonic sodium chloride solution is introduced in a volume of 1000 ml. If the response does not occur, it is advisable to use plasma-substituting hemodynamic drugs.

Corticosteroids are recommended for use in any period of shock. In the acute period 30 - 60 mg of prednisolone or 125 mg of hydrocortisone is administered subcutaneously, in severe cases - intravenously. These doses can be repeated every 4 hours before the acute reaction is stopped. In the future, to prevent allergic reactions by immunocomplex or delayed type and prevention of allergic complications, it is recommended to use

glucocorticoids inside for 4-6 days, with a gradual decrease in dose by ¼ - ½ tablets per day. Duration of treatment and dose of the drug depends on the patient's condition.

For relief of bronchospasm in addition to adrenaline, it is recommended to enter eufil-lin 2,4% -10 ml with the appearance of stridor breathing and the absence of the effect of complex therapy, it is necessary to produce a tracheotomy. Diuretics for swelling of the lungs against the background of collapse are not shown, since the kidneys do not fulfill their function. To correct heart failure use cardiac glycosides (strophanthin or korglikon). The convulsive syndrome is stopped by a relanium or sodium oxybutyrate. When psychomotor agitation is recommended droperi¬dol 2,5 - 5 mg.

In anaphylactic shock caused by penicillin, 1000000 ED of penicillinase is injected intramuscularly once into 2 ml of saline. With anaphylactic shock, bicillin penicillinase is administered for 3 days to 1,000,000 units.

With anaphylactic shock, it is traditionally recommended to use antihistamines, the effectiveness of which requires confirmation.

SERUM disease

SERUM disease - a systemic allergy is not a disease, an egg is caused by the introduction of heterogeneous serums or dasgs from them and is characterized by inflammatory lesions of blood vessels and connective tissue. Antibodies involved in the development of serum sickness are of the IgG and lgM classes. The process takes place in three phases: in phase I (2-3 days), the concentration of antigen is drastically reduced, so the intravenous antibody administered is distributed over the intra-and extracellular protein pools. In Phase II (6-8 days), it gradually decreases due to catabolism of the antigen in the body (depending on the type of antigen and animal species). In the III phase (4 days) again there is a sharp decrease in concentration until the complete disappearance of the antigen. The rapid extinction of antigen is due to the appearance of LI inflammation with antibodies that were formed during this time. In the case of the interaction of antibodies with soluble antigens, soluble CIPs are formed, and only with certain correlations of antigens and antibodies in their composition (sometimes there is an abundance of antibodies) they are excited. If the component of the complement system is bound, the precision of the PIC is dissolved. Soluble CICs are usually rapidly released by the

kidneys. However, the deficiency of the complement, as well as the excess of antigen, predispose to the formation of insoluble CICs. Such complexes are slowly removed by the macrophage system. Then there is a shortage of complement, as with the excess of the TEC, its reserves are depleted. The CIC is accumulated along the main cross-sections (basal membranes). When it observed the general symptoms (fever) and local manifestations, which depend on the place of deposition of the CIC vasculitis (with their presentation on the vascular wall with subsequent involvement in the inflammatory process), arthritis (with deposition of the CIC in the joints), nephritis (with the deferral of the CIC in the kidneys).

Serum sickness is characterized by a greater variety of clinical manifestations. In mild cases, the increase in body temperature usually does not exceed 1 LS for 1-2 days, in more severe cases the body temperature reaches 39-40 ° C and lasts for 7-10 days. The nature of the temperature curve is incorrect, intermittent. Lymphoenalapatiya with the most common symptom of serum sickness. An especially rapid increase in regional lymph nodes located near the serum administration site is evident. The second sign of the frequency of detection is the skin rash, which is characterized by polymorphism. Most often, it is a urticaria, which is accompanied by a severe itching. The affliction of joints depends on the severity of the course of serum sickness with different frequencies. Patients complain of pain and limitation of movement in the joints. The pathological process is mainly lesioned in the knee, abdominal, abdomen, elbow and ray wisdom joints, as well as in small joints of the hands and feet, rarely in the intervertebral joints. From the side of the CCS, there is a certain decrease in AT, due to the weakening of the tone of the sympathetic nervous system, in combination with bradycardia or tachycardia. Possible violations of the conduction system of the heart, the development of myocardiogenes, as well as hepatitis, glomerulonephritis.

SUCCESSION OF APPLICATION OF MEDICINAL PREPARATIONS IN THE PRACTICE OF INFINITY

The severity of the serum sickness may be different: from the rudimentary form, which manifests itself by individual clinical features, to severely1 forms with sharply

pronounced leading symptoms of the disease (fever, rash, enlargement of the lymphatic nodes, sharp general weakness) and severe manifestations from different organs .

For treatment, HCS is prescribed (prednisone 0,006-0,009 g and isomyostomy or intravenous; dexamethasone 0,0008-0,0016 g intravenously), membrane stabilizers (ketotifen 0,0001 g 2-3 times a day), antihistamines (suprastin in 2 ml of 2% solution 2-3 times a day). Disinfection and simultaneous elimination of antigen and mediators are achieved with the help of hemosorption, echterosorption.

STEVENS-JOHNSON SYNDROME

Stevens-Johnson syndrome belongs to the systemic allergic reactions of the slowed-down type; This is a difficult variant of multiform exudative erythema, in which simultaneous with the defeat of the lining of the mucous membranes there is a defeat of the skin.

The reason for the development of Stevens-Johnson syndrome may be various drugs: antibiotics, especially the ienicillin number, NSAIDs, sulfanilamide preparations, vitamins, more often B, ascorbic acid, barbiturates, vaccines, etc. Also, today, discussing the possibility that this syndrome, as well as multivariate exudative erythema, causes mycoplasma infection.

The main source in the development of hypersensitivity of the retarded type in medication and allergies play T-lymphocytes, which have on their surface specific receptors to preparations shiergenov. At the same time, CICs are formed, which are largely due to the activation of the complement, leading to multiple organ lesions, including mucous membranes and skin. Sitsdrome Stevens-Johnson - the result of mixed reactions of several types (shunokompleksniy, hypersensitivity of the slowed-down type).

Clinical picture is manifested by symptoms of severe swollen intoxication and erosive-ulcerative defeat of the mucous membranes of the oral cavity, the nose of the throat, eyes, genital organs and rectum. The body temperature rises to 40 ° C and more. Patients complain of headache, general weakness, arthralgia. Often bothering with sore throat when swallowed. On the lips there are blisters, which crack quickly and form thick blood peeled thick blood. On the mucous membrane of the cheeks, soft and hard palate, tongue, pharynx from the bubbles, there are large painful erosions that merge with each other, and

ulcers leading to abundant salivation. The surface of the ulcers is often covered with a rather thick yellow necrotic film that is not removed. Around the erosion with a bright red rim width of 1-2 mm, expressed diffuse swelling and hyperemia of the mucous membrane. Opening your mouth and swallowing liquid meals: extremely difficult. Defeat of the mucous membranes of the nose is accompanied by the formation of bloody crust at the entrance to the HJC and frequent nasal bleedings. Eye symptoms are manifested by the development of bilateral vesicular conjunctivitis, which in severe cases can lead to keratitis. In the distal region of the rectum, painful ulcers and erosions that bleed are developed. At the 4-6th day of the disease there is a rash in the skin - first in the symmetrical areas of the rear of the brushes, forearms and shiiches, then in the front of the 29th GENERAL INSECTOLOGY The upper legs and legs are more distinct on the face (in the circle of the coronary artery, on the neck, trunk) The clinical picture of the rash is very polymorphic (see Figure 5, see colored adhesive); simultaneously, various forms and stages of disintegration of the elements can be observed: spots, papules and blisters that can merge. Hardness The course of the syndrome is correlate: with the number of blisters. After 1 - 2 days, the center of the spots is slightly depleted, pale, becomes a bluish-purple tint. The disease lasts 4-6 weeks or more, goes badly to treatment, relapses often occur. Possible lethal consequences in case appearance of symptoms of meningo- encephalitis The process may be complicated by the development of corneal erosion, blindness, proctitis, esophageal stenosis, urethral stricture in men, vulvovaginitis, vaginal sinus, myocarditis, toxic hepatitis, nephritis. In the treatment of Stevens-Johnson syndrome ed should cancel all drugs, except for those necessary for health po¬kazannyamy patient. Assign a hypoallergenic diet (liquid, rubbed food). The volume of infusion therapy is calculated in accordance with the vital needs of the body, but not less than 40 ml / kg body weight quorum. It is expedient to have lallazmaferez with a 20-50% bcc. GCS should be administered intramuscularly, based on prednisolone 0.0001- 0.0005 g / kg per day. For eye treatment, you can use the ophthalmologist I drop in the affected eye 1-4 times a day; azelastin in 1 drop 2 times a day; with severe lesions - corticosteroid eye drops and ointments (prednisolone 0.5% in 2 drops 3 times a day). The oral cavity should be treated with hydrogen peroxide after each meal. Mucous membranes of the urinary and

reproductive systems are treated with ointment on the basis of GCS 3-4 times a day. Antihistamines are prescribed in mid-therapeutic doses. Patients should be placed in a separate lobe and create sterile conditions with the prevention of bacterial complications.

LAYLA'S SYNDROME

Layla's syndrome is toxic epidermal, and necrosis, which is characterized by the syndrome of "burned skin". Its immediate cause may be antibiotics, sulfanilamide preparations, barbiturates, pyazolone derivatives and some other medicines. Two possible variants of the course of toxic epidermal necrolysis: a spontaneous course with a fatal outcome; less severe course with the development of a toxic-infectious syndrome and a recovery for 10-15 days, a lethal consequence is also possible. The process often begins as a common urticaria, but does not respond to treatment with gestamyshmi means. On the skin there are itchy blisters. Poor condition of patients deteriorates rapidly: characterized by prolonged high fever, marked symptoms of intoxication (headache, general weakness, joint pain, nausea, etc.). On the background of pain and burning of the skin on the face, trunk, mucous membranes there is an erythematous swollen rash of various sizes, often draining. Soon, there are numerous large, flaky vesicles with serous or serous-hemorrhagic contents that merge with the formation of large erosions, massive exudation, which leads to dehydration and deterioration of the patient's condition. The syndrome of endogenous intoxication is expressed as much as possible. There is an increase in headache, loss of consciousness. Developed by the current encephalopathy, acute liver failure, gastroduodenal insufficiency.

COMPLICATIONS OF THE APPLICATION OF MEDICAL DRUGS IN PRACTICE INFECTIVITY (GNE), toxic myocarditis. Often joins sepsis. Mortality at Lyell's syndrome reaches 50-80%. Treatment of patients should be carried out in a resuscitation unit and identically to such at burn conditions; strict asepsis is a prerequisite. Take care of the affected skin and mucous membranes kami The areas of necrosis are treated with antiseptic agents (3% solution of hydrogen peroxide, etc.), hipschina or buckthorn oil. In the eyes, 0.01% of dexamethasone solution is dropped in 1 drop 3 times a day. Over the centuries, lay 1% hydrocortisone muj. Conduct active de-toxication measures. The main purpose of infusion therapy is detoxification, normalization of water-electrolyte balance,

correction of those propoeteinsemia, prevention and treatment of DIC. Assign GCS in high doses intravenously Drop, To improve the rheological properties of blood enter heparin, pentoxifylline. Due to the danger of joining the secondary infection using broad-spectrum antibiotics. Sorbents are prescribed internally or injected through a nasogastric tube probe 4-6 times a day. Widely used extracorporeal methods of detoxification of the organism.

DYSBIOSIS

This term reflects quantitative and qualitative changes in the microflora in the colon. Dysbiosis of the large intestine may be post-infectious (more often after persistent intestinal infections); medicamentous (more often after the use of antibiotics of a wide spectrum of action); alimentary (in case of unbalanced nutrition with deficiency of food fibers); radiation. This pathology occurs in various somatic diseases (ulcerative colitis, diffuse polyposis, etc.). The development of dysbiosis in the large intestine is facilitated by endocrine dysfunctions, immunodeficiencies, psychoemotional suturing, environmental degradation. In the English literature, the term "bacterial overgrowth syndrome", the bacterial overgrowth syndrome, is used to denote the expansion of microbial growth in the small intestine. It is diagnosed in cases where the amount of microorganisms in the empty intestine exceeds 105 in 1 ml. The dysbiosis of the small intestine breaks up with muscle failure - the switch of the ileocecal region or its resection, with strictures of the intestines, resection of the segment of the small intestine with an anastomosis side-by-side, etc. By the nature and severity of the clinical symptoms, there are three forms: compensated (without clinical manifestations); subcompute spray (localized); decompensated (generic). The last firm is divided into uncomplicated and complicated by some authors. Clinical symptomatology at 1 and II degrees of dysbiosis of the colon, as it is true, is absent. In this period, dysbiosis is basically a microbiological concept. Under subcompensated form it is possible to observe subfebrile, decrease in appetite, disturbance of motility of the digestive canal. In the case of a decompensated form of dysbiosis of the large intestine, a whole spectrum of clinical manifestations arises: secretion or diarrhea, or their alternation, flatulence, food and drug allergy, abdominal pain, degeneration of water and mineral metabolism, etc. With antibiotic-associated

diarrhea, pseudomembranous colitis can develop as a result of colonization of the large intestine Clostridium difficile.31 GENERAL INFECTIONS The clinical symptoms of dysbiosis of the small intestine may not be present, but often the syndrome of relapsing secretion diarrhea is often found. When pronounced microbial contamination of the small intestine appears flatulence, discomfort, abdominal pain, steatorrhea and creator, B12-deficiency anemia, deficiency of vitamins K and A. In severe cases, sepsis may develop. The main method that confirms the diagnosis of dysbiosis of the large intestine, is a bacteriological study of feces. This method makes it possible to establish the quantitative and specific composition of the microflora dominant at that time in the intestine, to detect the change of the obligated microflora to the conditionally pathogenic. It is known that the bacteria colonizing the ileum gut 'are representatives of 17 rolls, 45 kindling and 400-500 species. The ratio of anaerobes and aerobes is 10: 1. The total number of anaerobes reaches 1013- 1014 microbial cells in 1 g of intestinal contents. These include gram-positive forms (bifidobacteria and schizophrenia); Gram-negative are anaphylactic anaerobes (bacteroids, fusobaccariae, paiseria) and andigy forms. Frequent concomitant microflora is 8-10%; mainly not lactobacillus and enterobacteria. The final microflora is represented by the conditionally pathogenic bacteria of the enterobacteria family (klebsiella, tsitrobakter, enterobacter, protius, etc.). There are four degrees of dysbiosis of the colon: I degree - the number of anaerobes, lactobacillus does not change; increases or decreases the number of E. coli, conditionally pathogenic flora within the norm. II degree - the number of anaerobes at the lower limit of norm or reduced; decreases the number of normal E. coli; increases the density of atypical strains, may appear hemolysing colonies of E. coli and staphylococcus; the number of conditionally pathogenic flora increases. III degree - decreases the number of bifidumb bacteria; E. coli are mostly represented by atypical strains, the amount of lactobacilli is reduced; the number of conditionally pathogenic flora continues to increase. There was no degree of bifidobacteria; E. coli is represented by a small number of atypical strains; conditionally pathogenic flora grows in associations, although one strain can predominate (hemolysis whose staphylococcus aureus, protius, sinushishna paulichka, etc.). Classification of intestinal dysbiosis according to the microbiological characteristic includes the following types: 1)

bifidumdeficiency (reduction or absence of bifidumbacteria); 2) esherichia (increase, decrease or appearance of atypical strains of the E. coli); 3) staphylococcal; 4) Protective; 5) fungal (candidiasis); 6) encerococci; 7) due to Pseudomonas aeruginosa; 8) Associated. Dysbiosis of the small intestine is diagnosed by direct sowing on bacterial media of the contents of the small intestine, which is obtained by a special thin-intestinal probe. An increase in the total number of microorganisms (more than 105 microbial cells in 1 ml of intestinal contents) confirms the diagnosis. Correction of the colon dysbiosis requires an integrated approach: 1) adequate treatment of the underlying disease, which caused the development of dysbiosis; 2) restoration of impaired functions of the colon; 3) increasing the overall resistivity of the macroorganism by stimulating its immunological and non-specific defense; 4) Correction of dysbiosis of the large intestine using functional nutrition, pre-, pro- and synbiotics, as well as (according to strict indications 32 NON-SPECIFIC PREVENTION OF INFECTIOUS DISEASES) of intestinal antiseptics and other antibacterial and anti-parasitic agents. At stages I and II dysbiosis prescribe the consumption of products of natural origin, having the ability to restore the disturbed microbial cenosis of the colon. They include products of plant, animal and microbial origin containing bifidobacteria and lactobacilli, food fibers, natural antioxidants. Wanted to consume soy milk, pectins, proteins, vitamins, minerals, which are often called "nutritional drugs". An important component is the food fibers that increase the volume of feces, stimulate the passage of food chyme, removing constipation, serve as a source of short-chain fatty acids. Along with food fibers, it is recommended to consume dairy products (kefir, sour, yogurt, cheese, etc.). Prebiotics are used to correct dysbiosis. Prebiotics are components of food that are not digested and are a substrate for the selective growth of the habitat microorganisms, primarily bifido- and lactobacilli. Prebiotics include lactulose and other oligoccrids. Lactulose is a nutrient substrate for sugrolytic bacteria. Prebiotics should be combined with probiotics. These are preparations made on the basis of living microorganisms - representatives of volunteer microflora: bifido-, lacto-and colibacilli, enterococci. Probiotics should be considered only as a means of recovery of eubiosis of the large intestine. At III and IV degrees of dysbiosis, the need for the administration of antibacterial agents is suppressed by the inhibition of

opportunistic and pathogenic microflora of the large intestine, since independent recovery of eubiosis in these cases does not happen. It is necessary to start with those antibacterial agents, which selectively suppress conditionally pathogenic microorganisms and thereby contribute to the growth and reproduction of bonds of the microflora.

2. Learning Objectives classes (with the level of assimilation, which is planned)

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

have an idea: the complications of the use of drugs, pathogenesis and classification of complications, principles of diagnosis, treatment, emergency treatment and prevention of side effects of drugs. read: statistics related to the prevalence of complications as a result of drugs, frequency of their occurrence, mortality in Ukraine and in the world today.

2.2. The student must know:

• complications arising from the actions of drugs;
• pathogenesis of;
• classification of complications;
• principles of treatment;
• tactics in case of emergency conditions;
• Weather complications arising from the use of medications
• principles of prevention.

2.3. The student should be able to:

• collect allergy history and history of illness;
• examine patients and detect symptoms of complications of drugs;
• To assess the dynamics of the major clinical manifestations;
• carry out differential diagnosis with other diseases;
• make medical records into the adverse implications of drugs;
• interpret the dynamics of laboratory examinations.
• make recommendations regarding the regime, diet, inspection, supervision of patients.

2.4. Creative level (for the most capable students):

• develop creative skills of students during clinical trials, analysis of scientific sources;
• involve students to work in student science club chair;
• to propose themes for UDRS and NDRS of the most pressing issues, such as: "Problems

of prevention of complications as a result of drugs"

3. Educational goals (individual goals):

Develop the deontological idea while studying the topic. Be able to follow the rules of conduct at the bedside, the principles of medical ethics. Master skills to establish a psychological contact with patients.

On the basis of the theme to develop a sense of responsibility for the timeliness and accuracy of professional action.

4. Track Session:

Side effects of drugs:

1. Effects related to direct pharmacological action of LP.
2. Toxic effect of drugs
3. Interaction of drugs which may arise as a result PDLZ
4. Allergic reactions
5. Idiosyncrasy
6. Drug Violations biological properties of an organism (bacteria overgrowth, bakteriolizysu reaction, influence on the immune system)
7. Unusual effects of drugs
8. Teratogeny and tumorigenesis.

Differential diagnosis:

Symptoms	serum sickness	syndrome Stevens-Johnson	syndrome Layyela
Patanatomichno		spongiosis, intracellular edema. Blister formed under the epithelium, with necrosis	erosion of surface layers of the epidermis and epithelium, edema rostkovoho layer violation of intercellular connections with

			blistering
The temperature	high	high	high
Rash	Polymorphic rash is often maculopapular	The skin makulo-papular, bullous rash with hemorrhagic component. On the mucous membranes of the mouth, nose, conjunctiva, urethra-necrotic lesions ulser	erythematous-bullous skin lesions and mucous membranes. Detached epidermis (epidermonekroliz) , which resembles a burn
Itching	Yes	No	No
C-M Nikol'skii	Rarely	can be high	positive
The defeat of the internal organs	not always	not always	often in the form of glomerulonephritis, myocarditis, hepatitis, etc.
CNS lesions	possible toxic damage	possible toxic damage	always accompanied by toxic damage

Principles of treatment:

1. Desensitizing drugs (dimedrol, suprastyn, Phencarol, klarytyn)
2. Anti-inflammatory
3. Vitamin therapy
4. Calcium Preparations
5. Corticosteroids

6. Massive detoxification therapy

7. Local treatment that aimed at the elimination of edema and inflammation accelerate epithelization of affected areas (pain relievers, antiseptic agents, proteolytic enzymes, keratoplasty)

5. Materials methodological support classes:

5.1. Control materials for the preparatory phase of training.

5.1.1. Test questions for the individual survey

1. What are the possible complications from the use of drugs in the practice of infectious disease.

2. Etiology of complications.

3. Pathogenesis of adverse drug zasobiv.

4 Action. Clinical classification of complications of therapy and pharmacotherapy in the practice of infectious disease doctor.

5. Systemic manifestations of side effects of medications.

6.Pobichni effect of drugs on the part of: skin, gastrointestinal tract, nervous, cardiovascular, endocrine and urogenital systems.

7. Mental illness of complications of the treatment.

8. S-m Stevens-Johnson. Pathogenesis. Clinic. Principles of treatment.

9. Layela syndrome. Pathogenesis. Clinic. Principles of treatment.

5.1.2. -2α Tests Level 2: Choose the correct answer;

1) The development of Lyell's syndrome is caused by: A. Anaphylactic type of allergic reaction of B. hypersensitivity of delayed type C. cytotoxic type of allergic reaction D. immunocomplex type of allergic reaction E. all true

2) The allergic reaction of delayed type hypersensitivity develops within: A. 1-2 hours. B. 5-10 hours C. 24-48 hours D. 48-96 hours. E. 30 minutes

3) The symptom of Nikolsky is most typical for: A. Stevens-Johnson syndrome V. toxic epidermal necrolysis of S. serum disease D. toxic-allergic dermatitis. E. Anaphylactic shock

4) Which groups of drugs can lead to the development of Lyell's syndrome ?: A. antibiotics B. sulfonamides S. barbiturates D. pyrazolone derivatives E. all right

5) Choose the drug, the appointment of which there is the greatest risk of dysbiosis: A. ceftriaxon B. fluconazole C. titriazolin D. anaprilin E. lacidophilus

6) The most common defeat of internal organs occurs when: A. Stevens-Johnson syndrome B. serum disease S. Lyell syndrome D. toxic-allergic dermatitis E. edema Quincke

7) Itching of the skin is a constant sign: A. Lyell's syndrome of B. serum disease S. Stevens-Johnson syndrome D. Nodal eczema E. edema Quincke

8) Polymorphic, patchy-papular rash is characteristic for: A. Lyell's syndrome of B. serum disease S. Stevens-Johnson syndrome D. edema Quincke E. All true

9) Which drug should be prescribed to the patient when an allergic reaction occurs: A. loratadine V. claritin S. diazolin D. cetrin E. all right

10) Which drug should first be prescribed to a patient when an anaphylactic shock occurs: A. countercrack B. loratadine S. adrenaline D. furosemide E. hydrocortisone

11) Clinical manifestations of serum sickness are: A. fever B. polymorphic rash C. joint pain D. enlargement of the lymph nodes E. all true

12) The allergic reaction of hypersensitivity of immediate type develops in the course of: A. from a few seconds to 6 hours B. 5-10 hours C. 24-48 hours D. Within a day E. within a week

13) Indicate a drug that is contraindicated to a patient with renal insufficiency A. penicillin V. gentamicin S. amoxicillin D. ceftriaxone E. levofloxacin

14) A patient has a history of an allergic reaction to penicillin. On which of the listed antimicrobial drugs is most likely the development of an allergic reaction: A. ceftriaxon B. levomycetin succinate S. amikacin D. lacidophil E. loratadine

15) The risk factors for the occurrence of side effects of drugs are: A. intolerance of a drug in a history of B. simultaneous administration of two or more drugs of the same group C. concomitant administration of two or more drugs of different groups without regard for their interaction D. anaphylactic shock in an anamnesis E. all listed is true

16) A patient with a history of peptic ulcer 12 duodenal ulcer. Which of the drugs is contraindicated? A. clarithromycin B. prednisolone S. ranitidine D. alamegel E. omeprazole

17) What disease is accompanied by necrosis of superficial layers of the epidermis? A. Lyell's syndrome V. Stevens-Johnson syndrome C. toxic-allergic dermatitis D. chicken pox E. systemic lupus erythematosus

18) For the treatment of Stevens-Johnson syndrome apply: A. plasmapheresis V. prednisolone C. infusion therapy D. antihistamines E. all true

19) For the treatment of serum sickness, all are used except for: A. Glucocorticoids B. adrenaline C. suprastin D. enterosorbents E. hemosorption

20) The patient had nausea, dyspnea, tinnitus in 60 seconds after the introduction of antidiphtheria serum, Objectively: unconscious patient, pale skin, cold sticky perspiration on the face, Hell 60/0 mmHg, HR of 120 beats in minutes, thready, heart sounds are deaf. What complication arose? A. Lyell's syndrome B. serum sickness S. Stevens-Johnson syndrome D. Quincke edema E. anaphylactic shock

STANDARDS OF FAITHFUL ANSWERS

1.B 2. C 3. B 4. E 5. A 6. C 7. B 8. B 9. E 10. C. 11 E 12. A 13. B 14. A 15. E 16.B 17. A 18. E 19. B 20.E

Tasks are for self-control

Task 1 **a=2**

Patient M., age in 1971, was in the hospital with a diagnosis of "pneumonia". Received combination therapy consisting of gentamicin in a single dose of 80 mg 3 times daily over pneumonia eufilin to 200 mg three times a day, furosemide 40 mg per day for the correction of blood pressure (hypertension in patients). Within 10 days after initiation of therapy developed acute renal failure and the patient died. In the history of chronic pyelonephritis, hypertension.

Task

1. Explain the reason for the ACF.
2. Errors in treatment? Ask the optimal treatment plan.

Task 2 **a=2**

Patient S., 57 years, routinely conducted endoscopic cholecystectomy. In the postoperative period assigned to combined drug therapy: heparin in 5000 OD 4 times a day, pancreatitis 300 OD 1 per day subcutaneously, ciprofloxacin 200 mg twice daily,

cefotaxime 1 g 2 times a day, Riboksin 10 mg 3 times per day intravenously. After two days the patient's condition worsened because of bleeding from the bed of the gall bladder (platelets 68 x109 / l, clotting time 44 minutes). An relaparotomy. After the surgery the patient was in a coma for weeks with signs of pneumonia and progressive brain edema. He died after 15 days of the planned operation carried out. In history it is known that 6 years ago the patient had the operation - correction of complex heart defects, after which for the past 6 years, he always took indirect anticoagulants - fenilin to 0.03 g-2 times a day.

Task

1. What are the risk factors of bleeding were not included?

2. How could prevent complications that happen? **Task 3** **a=2**

Patient in 1922 was appointed furazolidon in therapeutic doses for treatment dysbacteriosis, which showed liquid stools without pathological impurities. After 5 days of treatment the patient revealed jaundice, increased ALT activity twice above normal, bilirubin 25.1 mmol / l (by indirect fraction).
When ultrasound revealed liver enlargement (dynamic 1 cm +1.5 cm, 2 cm). Ehostruktura homogeneous. As a result of the discontinuation of the drug and the treatment lipoic acid, vitamin E, sorbent patient's condition and normalized liver function tests returned to normal.

Tasks?

1.Chy can be associated with deterioration of patient intake furazolidone? Justify.
2.How advisability of furazolidone in this case as a preparation for correction dysbacteriosis?

7. Materials are for audience independent work.

7.1. List of educational practical tasks which must be executed on practical employment:

- To conduct examinations of patient with toxoplasmosis
- To conduct differential diagnostics of medicamentosus diseases
- ·· To work out a plan of treatment sick of anaphylactic shock
- To define medical tactic in the case serum diseases

ESPECIALLY DANGEROUS INFECTIONS

Actuality of theme:

Infectious diseases continue to be a serious problem and a threat to humanity. Particularly important among them are especially dangerous infections - a group of diseases to which quarantine measures are applied in accordance with international health and sanitary regulations. This group of infectious diseases is able to rapidly spread in a short period and cause severe clinical forms that cause high mortality. The epidemic features of especially dangerous infections are noted in the form of sporadic cases, local outbreaks, epidemics and pandemics.

In 2005, at the 58th World Health Assembly, WHO expanded the list of these diseases and divided them into several groups. The first group is "diseases that are unusual and can have a significant impact on the health of the population": smallpox, poliomyelitis caused by wild poliovirus, pandemic influenza, SARS. The second group is "a disease, any event with which it is always evaluated as dangerous, since these infections can have a serious impact on the health of the population and spread rapidly on an international scale": cholera, pulmonary plague, yellow fever, hemorrhagic fever Lassa, Marburg, Ebola, West Nile fever. This includes infectious diseases "that represent a particular national and regional problem", such as Dengue fever, Rift Valley fever, meningococcal infection.

Quarantinable diseases is a group of infectious diseases in which various degrees of quarantine are imposed. Quarantine can be imposed by the state throughout its territory (smallpox, pulmonary plague), at the level of the region, city, district, or institution (children's airborne infections).

To date, the relevance of bioterrorism is high, which poses a serious threat to the security of all countries. Modern technologies allow creating unidirectional biological weapons based on "slow" and "sleeping" viruses with large latent periods, modified pathogens of common or especially dangerous infections.

Plague is an acute zoonotic natural focal infectious disease with a predominantly transmissible mechanism of transmission of the pathogen, which is characterized by intoxication, damage to the lymph nodes, skin and lungs.

The causative agent is the gram-negative polymorphic immobilized rod Yersinia pestis, which belongs to the family Enterobacteriaceae, the genus Yersinia. Has a capsule, it does not form a spore. Facultative anaerobic. It grows on simple nutrient media with the addition of hemolyzed blood or sodium sulfate, the optimum temperature for growth is 28°C.

Yersinia pestis has more than 20 antigens, but the main role is played by thermolabile capsular and thermostable somatic, to which the V and W antigens belong. The main pathogenicity factors of the pathogen are exotoxin, endotoxin, as well as enzymes of aggression: coagulase, fibrinolysin and pesticides.

Rodents - marmots (tarbagans), ground squirrels, voles, gerbils, and also lagiformes (rabbits, pikas) play a leading role in preserving the causative agent in nature. The main reservoir and source in antropurgic foci are gray and black rats, less often house mice, camels, dogs and cats. Of particular danger is a person who has a pulmonary form of the plague.

The vector of the infection is a flea that can transmit the pathogen 3-5 days after infection and retains the infectiousness up to a year. Mechanisms of transmission: transmissible (with a bite of infected flea), contact (through damaged skin and mucous membranes in contact with infected animals or products of their vital activity), fecal-oral (when eating insufficiently thermally processed meat of infected animals), aspirating (a patient with pulmonary forms of the plague).

The susceptibility of a person is absolute in all age groups and under any mechanism of infection. After the transferred disease develops unstable immunity.

Natural foci of infection exist on all continents, with the exception of Australia: in Asia, Afghanistan, Mongolia, China, Africa, South America, where a large number of cases are registered each year.

Tularemia is an acute zoonotic bacterial natural focal infectious disease with various mechanisms of transmission of the pathogen, which is characterized by fever, intoxication, inflammatory changes in the entrance gate of infection, regional lymphadenitis.

The causative agent of tularemia, Francisella tularensis, belongs to the genus Francisella, the Brucellaceae family. Gram-negative polymorphic immobilized rod, spore and capsules do not form. Facultative anaerobic. Francisella tularensis grows on nutrient media with the addition of cysteine or egg yolk, rabbit blood, tissue extracts and other growth stimulants. The microorganism contains somatic (O) and enveloped (Vi) antigens, the main pathogenicity factor is endotoxin.

The source of the causative agent of infection are about 150 species of animals. The main reservoir and source of infection are rodents, rabbits, rabbits, water rats, muskrats, hamsters, etc. Among domestic animals, the reservoir of infection may be sheep, pigs, cattle, horses. A sick person cannot be a source of infection for others. Natural foci of tularemia exist on all continents of the Northern Hemisphere, in Western and Eastern Europe, in Asia, and in North America.

The carriers of infection are ticks, mosquitoes, and flies. In the human body, the pathogen can penetrate various mechanisms: transmissible (with the bite of infected insects), contact (in contact with infected rodents and water), alimentary (with the use of infected, thermally unprocessed products and water), aerosol (by inhaling the infected dust).

People's susceptibility to tularemia is high (reaches 100%). The infection of a person occurs mainly in the summer-autumn period. After the transferred disease the stable, long, but not absolute immunity forms.

Anthrax is an acute saprozonotic infectious disease with a predominantly contact mechanism for the transmission of the pathogen.

The causative agent of infection is the gram-positive fixed bacillus Bacillus anthracis, which belongs to the genus Bacillus, the family Bacillaceae. Aerobe. It grows on simple nutrient media, with access to oxygen. Has a capsule, forms spores, and when it enters a living organism, it is a vegetative form. Bacillus anthracis contains two capsular and one somatic polysaccharide antigens. The main factor of pathogenicity is exotoxin, which includes a protective antigen, a lethal factor (has cytotoxic effect and causes pulmonary edema) and an edema factor (causes tissue edema).

The reservoir of infection is the soil in which Bacillus anthracis persists for a long time and accumulates. Sources of infection for humans - large and small cattle, horses, camels, as well as wild animals (rabbits, wolves, bears, arctic foxes, etc.).

Anthrax develops when the vegetative forms or spores of the pathogen enter the body. Mechanisms for the transmission of pathogens to humans - contact (contact with infected animals, as well as livestock products contaminated with B. anthracis), aspirating (by inhaling the infected dust), fecal-oral (with the use of meat of the infected animal) and transmissible (through stings bites, flies lighters, mosquitoes). Sick people do not pose a danger to others. Immunity in those who had recovered is unstable, there are cases of repeated diseases.

Anthrax at the moment in the form of sporadic cases is recorded on all continents and in all countries. However, periodically anthrax outbreaks are observed in countries where livestock are well developed - Asia, Africa, South America.

2. Learning objectives of the lesson (indicating the level of mastery that is planned):

2.1. The student must have a presentation (to read): ***α - 1***

- to have a general idea of the place of especially dangerous and quarantine infections in the structure of infectious diseases, the prevalence in different regions of the world and different age groups, to get acquainted with statistical data on morbidity, mortality, frequency of complications, long-term consequences of the transferred infections;
- to get acquainted with the history of scientific study of especially dangerous and quarantine infections.

2.2. The student should know: ***α – 2***

1. Etiological factors that cause diseases of a group of especially dangerous and quarantine infections, the prevalence of these diseases in different regions of the world.

2. Epidemiology of especially dangerous and quarantine infections.

3. Pathogenesis of anthrax, plague, tularemia

4. Clinical manifestations with a typical course of anthrax, plague, tularemia.

5. Laboratory diagnostics of especially dangerous and quarantine infections, rules for the collection of biological material from patients with these diseases.

6. Principles of treatment of anthrax, plague, tularemia.

7. Rules for discharging patients with anthrax, plague, tularemia.

8. Principles of personal prophylaxis of especially dangerous and quarantine infections.

9. Prognosis for plague, tularemia, anthrax depending on the clinical form of the disease.

10. The tactics of the doctor's behavior in case of suspected quarantine infection.

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

1. Observe the basic rules of work at the bedside of the patient.

2. To collect the anamnesis of the disease with the evaluation of epidemiological data

3. To examine the patient and identify the main symptoms and syndromes of anthrax, plague, tularemia, to substantiate a clinical diagnosis.

4. On the basis of clinical examination, it is timely to recognize possible complications of anthrax, plague, tularemia, urgent conditions.

5. Draw up a plan for laboratory and instrumental examination of the patient.

6. Interpret laboratory test results

7. Correctly evaluate the results of specific diagnostic methods.

8. Develop an individual treatment plan, taking into account the epidemiological data, the stage of the disease, the presence of complications, the severity of the course, allergological history, concomitant pathology; provide first aid

9. Give recommendations on the regimen, diet, examination, observation in the period of convalescence.

10. Obtain medical documentation on the fact of establishing a preliminary diagnosis of anthrax, plague, tularemia (emergency notification).

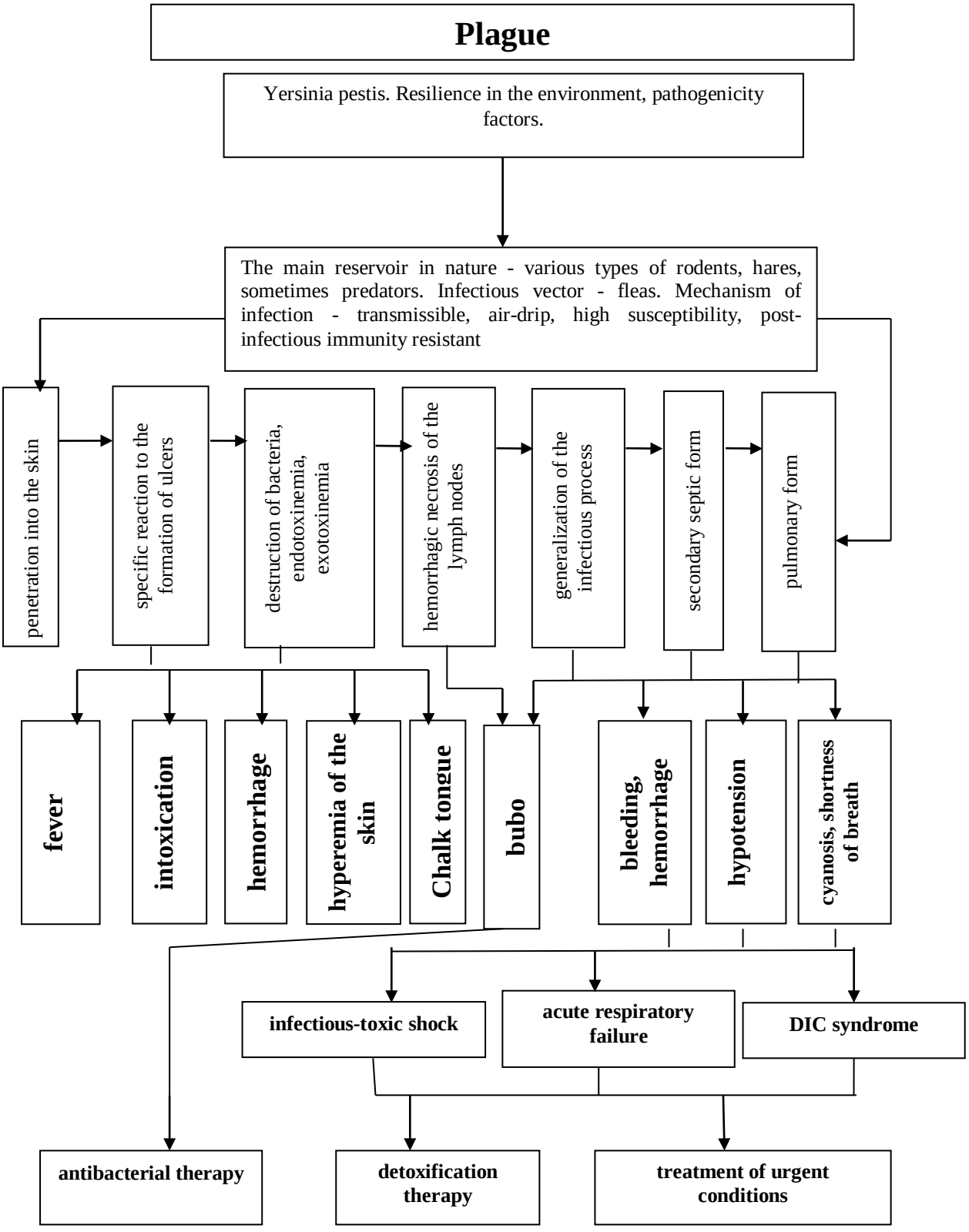
3. Materials for classroom independent work.

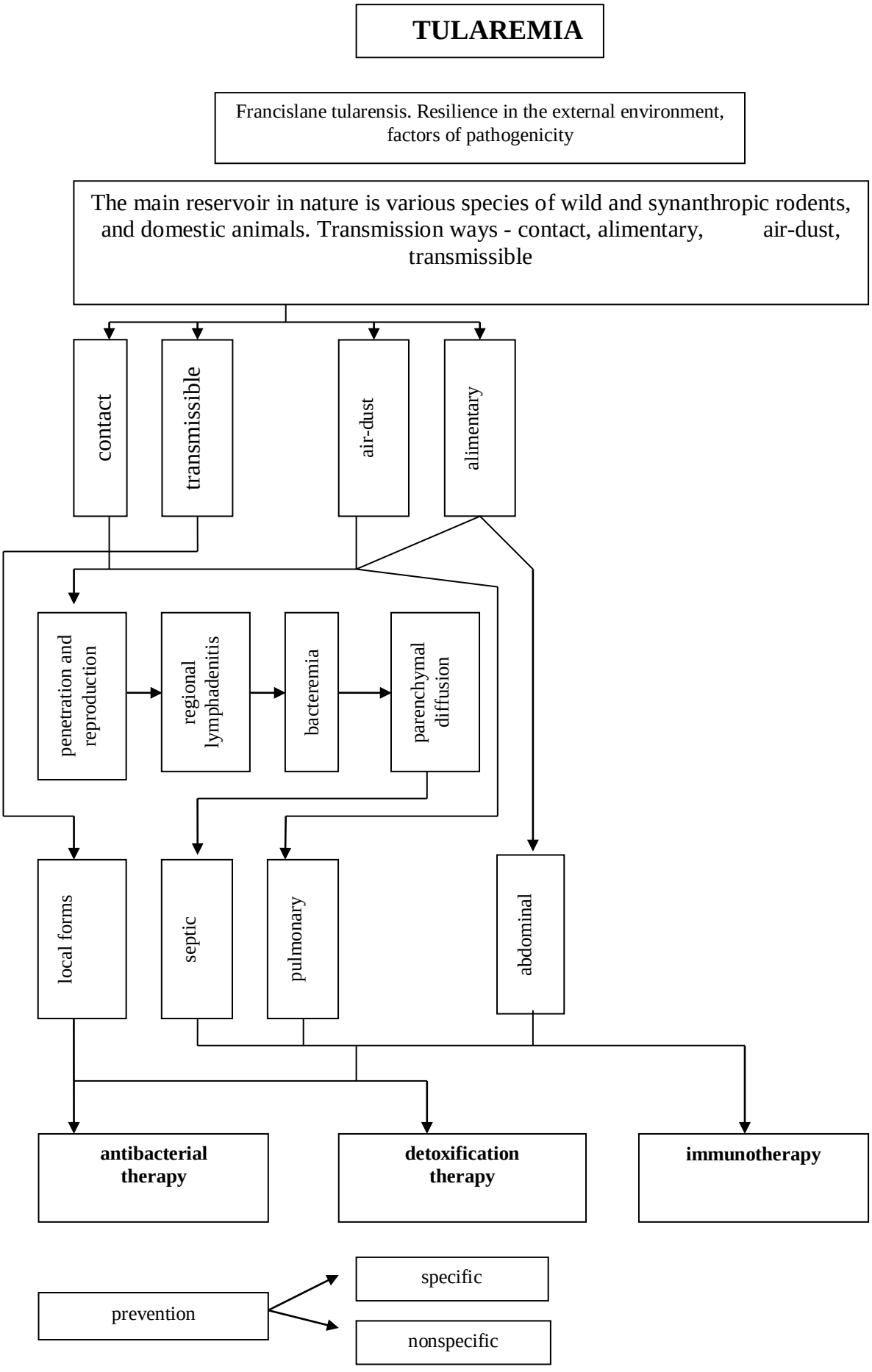
3.1. Basic knowledge, skills, skills necessary for studying the topic (interdisciplinary integration)

Discipline	Know	Be able to
Microbiology	Properties of pathogens of especially dangerous and quarantine infections, features of the serological response depending on the timing of the disease, rules and timing of material intake for specific diagnostics.	Interpret the results of specific and non-specific research methods.
Propaedeutics of internal diseases	Main stages and methods of clinical examination of a patient	Collect anamnesis, conduct a clinical examination of the patient for organs and systems, identify clinical signs of pathology, and analyze the findings.
Epidemiology	The epidemic process (source of infection, mechanisms and transmission routes) of diseases that are regulated by international sanitary regulations, prevalence in the world. The WHO strategy for the prevention and treatment of these infections.	Collect an epidemiological anamnesis, carry out an analysis of the compliance of clinical and epidemiological data.
Immunology and allergology	The role of immunity in the infectious process, the effect on the elimination of the pathogen from the patient's body. Immunological aspects of complications.	Evaluate the data of immunological studies.
Physiology	Parameirs of the physiological norm of human organs and systems, laboratory examination parameters in norm (general analysis of blood, urine, cerebrospinal fluid, biochemical blood tests, parameters of acid-base state, electrolytes, etc.)	Assess laboratory data.

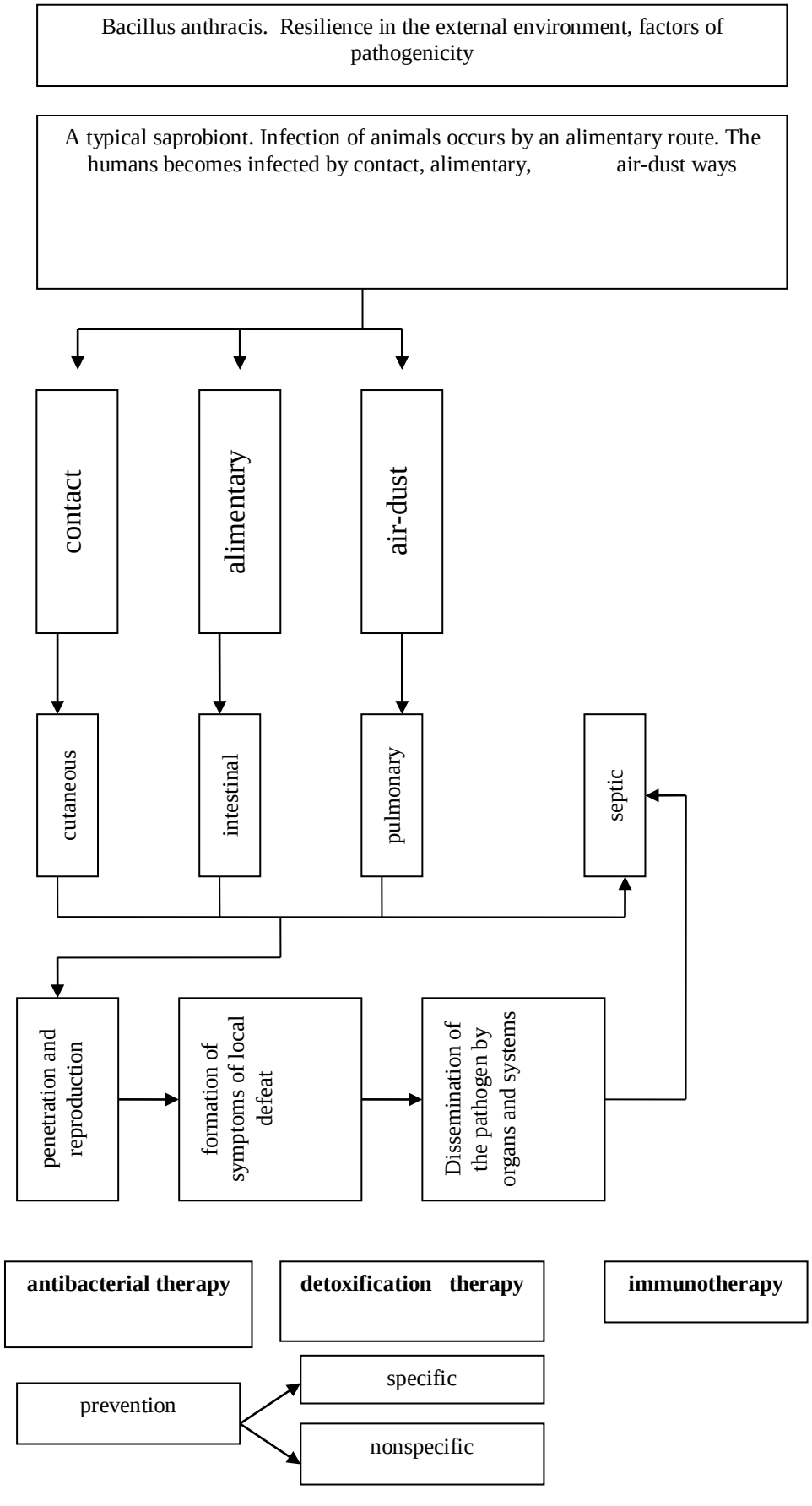
Neurology	Clinical and laboratory-instrumental signs of meningitis, encephalitis, toxic encephalopathy.	Conduct a clinical examination of a patient with CNS damage. Carry out a lumbar puncture.
Clinical Pharmacology	Pharmacokinetics and pharmacodynamics, side effects of the main medicinal etiotropic drugs, preparations of pathogenetic therapy.	Prescribe treatment, depending on the age, individual characteristics of the patient, choose the optimal regimen of priomy and the dose of the drug, prescribe a prescription.
Resuscitation and intensive care	Urgent conditions: acute respiratory failure, acute renal failure, infectious-toxic shock.	Timely diagnose and provide emergency care in emergency conditions: acute respiratory failure, acute renal failure, infectious-toxic shock.
Surgery	Clinico-laboratory signs of purulent-inflammatory complications. Rules for the provision of emergency care.	Timely diagnose complications, appoint an appropriate examination, provide emergency therapy.

3.2. Structural-logical scheme of the content of the class




TULAREMIA
Francislane tularensis. Resilience in the external environment, factors of pathogenicity
The main reservoir in nature is various species of wild and synanthropic rodents, and domestic animals. Transmission ways - contact, alimentary, air-dust, transmissible
contact
transmissible
air-dust
alimentary
penetration and reproduction
regional lymphadenitis
bacteremia
parenchymal diffusion
local forms
septic
pulmonary
abdominal
antibacterial therapy
detoxification therapy
immunotherapy
prevention
specific
nonspecific

ANTHRAX


Bacillus anthracis. Resilience in the external environment, factors of pathogenicity
A typical saprobiont. Infection of animals occurs by an alimentary route. The humans becomes infected by contact, alimentary, air-dust ways
contact
alimentary
air-dust
cutaneous
intestinal
pulmonary
septic
penetration and reproduction
formation of symptoms of local defeat
Dissemination of the pathogen by organs and systems
antibacterial therapy
detoxification therapy
immunotherapy
prevention
specific
nonspecific

3.4. Materials for self-control

3.4.1. Questions for self-control **α=1**

1. To what group of infectious diseases belong Siberian ulcer, plague, tularemia?
2. Source of infection in anthrax, plague, tularemia.
3. Ways of transmission of anthrax, plague, tularemia.
4. Factors of pathogenicity of pathogens of anthrax, plague, tularemia.
5. Antigenic structure of the causative agent of the plague.
6. Carriers of the plague.
7. Stages of pathogenesis of plague.
8. Local changes in the bubonic form of the plague.
9. Clinical forms of plague and their features.
10. Peculiarities of the pathogenesis of the plague depending on the place of penetration of the pathogen.
11. Peculiarities of the pathogenesis of anthrax depending on the place of penetration of the pathogen.
12. Features of pathogenesis of tularemia depending on the place of penetration of the causative agent.
13. Local changes in skin type of anthrax.
14. Characteristics, time of occurrence and peculiarities of the dynamics of rash during the plague.
15. Pathogenesis of fever in tularemia.
16. Clinical manifestations of violations on the part of the nervous system at the plague.
17. Clinical manifestations of disorders of the nervous system in tularemia.
18. Clinical manifestations of disturbances from the nervous system in anthrax.
19. Clinical manifestations of lesion of respiratory organs in pulmonary form of plague.
20. Clinical manifestations of skin form of plague.
21. Changes from the cardiovascular system in anthrax.
22. Complications of anthrax.
23. Causes of death in the plague.
24. Hemogram in the plague.

25. Methods of specific diagnosis of plague.

26. Conditions for the appointment of hemoculture in a plague.

27. Etiotropic plague therapy. Preparations, their doses, ways of introduction.

28. Etiotropic therapy of anthrax. Drugs and doses.

29. Pathogenetic plague therapy. Drugs, ways of administration.

30. Specific prophylaxis of the plague.

31. Clinical and epidemiological features of tularemia, differential diagnosis of plague and tularemia.

32. Emergency prophylaxis of plague and anthrax.

33. Rules for extracting from the hospital of a plague patient.

34. Organizational tactics of the doctor in case of suspicion of a plague or anthrax.

35. The notion of infectious diseases, which are regulated by international sanitary rules**.**

3.4.2. Test level: choose the right answers **α=2**

1. What antibiotic is used for etiotropic therapy of plague?

A. Erythromycin

B. Streptomycin

C. Penicillin

Д. Azithromycin

E. Levofloxacin

2. For laboratory diagnosis of plague, everything is used, except for:

A. Bacteriological study of the bubonic punctate

B. Reaction of indirect hemagglutination

C. Bacteriological examination of blood

Д. Immunofluorescence reaction

E. Bacteriological examination of bile

3. The plague is characterized by all transmission mechanisms, except for:

A. Vertical

B. Transmissible

C. Contact

Д. Airborne

E. Fecal-oral

4. The causative agent of tularemia is:

A. Coxiella burneti

B. Bacillus anthracis

C. Francislane tularensis

Д. Brucella melitensis

E. Yersinia pestis

5. The clinical forms of tularemia are everything, except for:

A. Arthritic

B. Bubonic

C. Ulcer-bubonic

Д. Eye-bubonic

E. Anginous-bubonic

6. The main sources of infection in tularemia are all, except for

A. Cattle

B. Sheeps

C. Rodents

Д. Sick man

E. Small cattle

7. The main factor of pathogenicity in anthrax is

A. Endotoxin

B. Exotoxin

C. Neuraminidase

Д. Hemagglutinin

E. Fibrinolysin

8. The drug of choice for etiotropic therapy of anthrax is:

A. Streptomycin

B. Gentamicin

C. Penicillin

Д. Doxycycline

E. Azithromycin

9. The cutaneous form of anthrax is characterized by:

A. Severely painful ulcer

B. Moderately painful ulcer, swelling of soft tissues

C. Painless carbuncle, edema of jelly-like consistency

Д. Erythema with clear boundaries in the form of "tongues of flame"

E. Roseous and petechial rash

10. Especially dangerous infections include all diseases, except for:

A. Plague

B. Anthrax

C. Cholera

Д. Tularemia

E. Chickenpox

11. From the first days of the disease, the phenomena of toxic encephalopathy are characteristic for:

A. Lymphogranuloma

B. Plague

C. Anthrax

D. Tuleremia

E. Purulent lymphadenitis

12. The mechanism of development of infectious-toxic shock in the plague:

A. Expansion of the vessels of the microcirculatory bed with their increase permeability

B. Regional release of histamine

C. Activation kallikrein-kinin system and intravascular proteolysis

D. Centralization of blood circulation

E. All is true.

13. Isolation of contact with plague is carried out:

A. No necessity

B. For 6 days

C. For 14 days

D. For 21 days

E. For 1 month

14. The causative agents of which diseases have a vegetative and spore form:

A. Erysipelas

B. Tularemia

C. Plague

D. Anthrax

E. All is true.

15. What disease is characterized by the presence of a painless ulcer, located on a dense ground, covered with a dark scab:

A. Tularemia

B. Anthrax

C. Plague;

D. Erysipelas

E. Erysipeloid

16. What disease is characterized by the presence of Stefansky's symptom (presence dense jelly-like consistency of painless edema):

A. Tularemia

B. Plague

C. Anthrax

D. Erysipeloid

E. Erysipelas

17. Patient A., during the last three months complains of a periodic increase the temperature. In the axillary region present a conglomerate of painless lymph nodes in the diameter of 4-5 cm, has a dense consistence:

A. Plague

B. Tularaemia

C. Anthrax

D. Lymphogranulomatosis

E. Erisipeloid.

18. The patient K. was acutely ill: T-38.5°C, weakness. On the right brush hands on a dense base painless covered with a dark crust sore diameter 1.5 cm, surrounded by a dense consistency of edema. Palpable regional lymph nodes 1 cm in diameter, mobile.

A. Plague

B. Tularemia

C. Anthrax

D. Lymphogranulomatosis

E. Erisipeloid.

19. The patient N., was acutely ill: within 5 days T-38-38.5°C. On the right hip ulcer 1 cm in diameter, the bottom of the ulcer is dense, covered with yellow exudate. In the groin area to the right palpable moving moderately painful lymph node in a diameter of 5 cm. Lymphangitis.

A. Tularaemia

B. Anthrax

C. Plague

D. Lymphogranulomatosis

E. Erisipeloid.

20. A material for bacteriological research in suspicious of the plague may be:

A. Punctate from the bubo

B. Contents vesicles, pustules, ulcers

C. Sputum

D. Blood

E. Everything is true.

Standards of correct answers

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

3.4.3. Tasks for self-control α=2

Task 1

Patient M., 38 years old, was admitted to the infectious department on the second day of the illness with complaints of an increase in body temperature to 40°C, chills, general weakness, headache. 5 days before the illness hunted the hare, independently chopped the carcass of the hare. Objectively: general condition of moderate severity, consciousness is not disturbed, temperature 38.7°C, blood pressure 120/60 mm Hg, pulse 60 beats/min. Skin covers are clean, no rashes. Painful formation with a diameter of up to 4 cm, not soldered with surrounding tissues, palpates in the inguinal area to the right. Scleritis, conjunctivitis. The tones of the heart are muffled, rhythmic. In the lungs a rigid breath is heard. When palpation the abdomen is soft, painless. The lower edge of the liver protrudes 2 cm from the edge of the edge arc. The spleen is not palpable. Meningeal signs are not revealed. In the general analysis of blood: leukocytes are $10,4x10^9$/l, rodenuclear neutrophils – 12%, segmental neutrophils – 49%, lymphocytes – 28%, monocytes – 10% eosinophils – 1%, erythrocyte sedimentation rate - 42 mm/h.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.

Task 2.

Patient K, 48 years old, was admitted to the infectious department on 2 days of illness with complaints of chills, an increase in body temperature to 40°C, myalgia, headache, weakness, pain in the inguinal area to the right, thirst. He works as a ratcatcher, lastly he has been deratization 3 days before the illness. Objectively: the condition is heavy, the body temperature is 39.7°C, the blood pressure 80/40 mmHg, the Ps 114 in min., arrhythmic. In the mind, it is slowed down. Hyperemia of the face, dryness of the mucous membranes. The tones of the heart are sharply muffled. In the lungs weakened vesicular breathing, no wheezing. The liver and spleen are not enlarged. In the groin area to the right, a node with a diameter of up to 10 cm is detected, sharply painful with palpation, without clear borders, the skin above it is red with a cyanotic tinge. In the general analysis

of blood: L-18x10^9/l, ESR - 54 mm/h, Hb-122g/l, rodenuclear neutrophils -28%, segmental neutrophils -60%, lymphocytes -11%, monocytes -1%.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.

Task 3.

Patient 63 years old, farm keeper. Hospitalized at the clinic on the 4th day of the illness with complaints of general weakness and swelling of the face. The patient noticed a "bubble" on his right cheek, which quickly increased in size, there was a swelling of the face and neck. Subsequently, the vial burst and an ulcer was formed in its place. The swelling has grown, it has become difficult to breathe. Body temperature reached 39.0°C, general weakness increased.

Objective: On the right cheek in the region of the mandible - an ulcer up to 5 cm in diameter. Around it is a crown of small vesicles with hemorrhagic contents. Pronounced edema of face and neck. In the right submaximal region, a significantly enlarged (up to 4 cm) painless lymph node is palpated. Pulse - 90 beats/min, blood pressure - 100/60 mm Hg. The tones of the heart are muffled.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.

Task 4.

The surgeon turned a man 38 years old, a farmer. 6 days ago on the rear of the right hand there was a very itchy reddish nodule, which the next day turned into vesicle. Then around new vesicles appeared, swelling of the brush quickly developed. The temperature rises to 38.5°C, no pain in the hand. From the anamnesis it is known that the patient participated in the slaughter of cattle. Objectively: a state of moderate severity, body temperature 38 ° C. The right brush and the lower third of the forearm are sharply swollen, on the back of the brush of a crust of dark brown color, up to 5 cm in diameter, around the vesicles in the form of a crown, filled with hemorrhagic fluid. Palpation is painless. Axis lymph nodes are enlarged to the right.

1. Clinical diagnosis.

2. Plan test.

3. Treatment.

Task 5.

Patient G., 32 years old, recently returned from Thailand. On the 3rd day Illness sick patient, speech is indistinct. Body temperature is 40.2°C. On the skin of the right leg is sharply painful ulcer 3-3.5 cm in size, covered a dark scab, with a red-purple inflammatory shaft around. From under a scrotal scar appears to have scant purulent-serous discharge. In the right inguinal he area is palpated with a dense, slow-moving tumor-like formation, sharply painful on palpation. The skin above it is hyperemic, tense. The number of respiratory movements is 36. The heart sounds are deaf, rhythmic. Pulse-130 bpm, weak filling and tension. BP -90/60 mmHg. The abdomen is soft, painless. Meningeal symptoms are negative.

1. Clinical diagnosis.

2. Plan test.

3. Treatment.

4. Materials for classroom independent work.

4.1. A list of training practical tasks that must be performed on a practical lesson:

• To use the method of examination of a patient with plague, anthrax, tularemia.

• Conduct a cure for a patient with anthrax, tularemia, and plague.

• Perform differential diagnosis of plague, anthrax, tularemia.

• To prepare a plan for laboratory examination of patients with plague, tularemia, and anthrax.

• Interpretation of the results of a specific examination of a patient with plague, anthrax, tularemia.

• Recognize the complications of the plague, anthrax, tularemia.

• To prepare a plan for treatment of a patient with plague, anthrax, tularemia.

• Determine the medical tactics in the event of emergencies.

• To issue medical documentation on the diagnosis of plague, anthrax and tularemia.

4.2. Professional algorithm for the formation of skills and skills in the diagnosis of anthrax, plague, tularemia

№	The task	Sequence of execution	Remarks, warnings regarding self-control
1.	To master the technique of clinical examination of a patient with plague, tularemia, anthrax	I. Determine the patient's complaints	Identify complaints that characterize syndromes: - general intoxication - organ lesions - skin lesions
2.	Carry out the patient's curation.	I. Find out anamnesis: 1. Anamnesis of the disease 2. Anamnesis of life 3. An epidemiological history	Pay attention to the dependence of the realization of a possible route of transmission of infection from the first manifestations of the disease. To reveal the transferred diseases. Identify data on the implementation of the transmission mechanism, pay attention to the patient's stay in regions with an increased risk of contracting the plague, to expose the linkage of diseases from professional activities
		II. Conduct an objective examination of the patient. 1. General inspection: - the general condition of the patient; - skin, mucous oropharynx, lymph nodes	Remember: the presence, severity, and dynamics of symptoms are due to the duration and severity of the course of the disease, depending on the patient's age, concomitant pathology, mechanisms of infection. Pay attention to - lethargy, adynamy, inhibition of the patient;

		2. Digestive system: - percussion of the abdomen; - palpation of the abdomen; 3. Cardiovascular system: - pulse - blood pressure; - auscultation of the heart. 4. Respiratory system: - auscultation of the lungs. 5. Nervous system	- body temperature; - color of the skin; - presence, localization, the nature of the rash, enlarged lymph nodes, the nature of their lesions, possible changes in the oropharynx Pay attention to - "Chalky tongue" (plague) - hepatosplenomegaly Pay attention to - tachycardia - moderately lowered blood pressure (a significant reduction indicates a complication); - moderate deafness of heart tones. Pay attention to - possible signs of pneumonia, pulmonary edema of the lungs; - the nature of the sputum In severe course excitement, nonsense, hallucinations are possible
3.	Appoint laboratory and additional research methods, interpret the results.	1. General blood analysis	Pay attention to changes in the hemogram: leukocytosis, neutrophilia, possible anemia, thrombocytopenia, significant acceleration of the rate of erythrocyte sedimentation.

		2. General urine analysis	Lack of significant changes in the typical course, with severe course of signs of toxic kidney damage
		3. Ultrasound examination of the organs of the abdominal cavity	Hepatosplenomegaly
		4. **for anthrax** - bacteriological examination of the contents of the boil (or sputum, blood, stools when suspected of the corresponding clinical forms)	Isolation of the pathogen, its identification
		- reaction of Ascoli (thermoprecipitation)	Used to carry out laboratory tests with skin and animal meat
		- immunofluorescence reaction	It allows to detect antigens of the causative agent of anthrax
		5. **for plague** bacteriological examination of the contents of the lymph node, sputum, blood, feces in case of suspected clinical forms	Isolation of the pathogen, its identification
		- bacterioscopy of the corresponding preparations, including with the help of fluorescent serum	The ability to quickly obtain results

		- a direct hemagglutination reaction, a neutrality reaction, an indirect immunofluorescence reaction	It is necessary to examine in dynamics with the growth of antibodies titer 4 times

5. Materials for out-of-class independent work

5.1 Themes for the research work of students:

• Peculiarities of plague and anthrax in modern conditions

• Modern methods of specific diagnosis of plague and anthrax.

• Anthrax as a biological weapon

HEMORRHAGIC FEVER

Actuality of theme: viral hemorrhagic fevers are a group of natural focal infectious diseases that are recorded on all continents of the world, except for Australia. Diseases are characterized by a specific lesion of the human hemostatic system, multiple organ failure with the development of severe intoxication, hemorrhagic syndromes, high lethality.

The causative agents of viral hemorrhagic fevers are RNA-containing viruses that belong to four families: Arenaviridae, Bunyaviridae, Filoviridae and Flaviviridae. n accordance with the International Health Regulations (WHO, 2005), these diseases are considered especially dangerous to human infectious diseases. The causative agents of viral hemorrhagic fevers are now considered potential agents of bioterrorism.

Hemorrhagic fevers **Lassa, Marburg** and **Ebola** are zoonotic natural-anthropurgic viral infectious diseases characterized by severe hemorrhagic syndrome. Causative agents - RNA-containing viruses. The Lassa fever virus belongs to the Arenaviridae family, the causative agents of Marburg and Ebola fevers belong to the family Filoviridae.

The source of the infection of the Lassa fever is the multinipples rat Mastomys natalensis, Marburg and Ebola fevers are African green monkeys. Lassa fever occurs in Western (Sierra Leone, Nigeria, Senegal, Mali, Guinea) and Central (Zaire, Burkina Faso) Africa; fever Marburg - in Zimbabwe, Kenya; fever Ebola - in Zaire, Sudan. The secondary source of infection is the infected person. People who live in rural areas are more often ill. In a sick person, the virus is contained in the blood, mouth and nasal secretion, urine. Diseases occur more often in families and other closely-communicated groups of the population.

The mechanism of transmission of the pathogen is diverse. The infection of humans from animals occurs by the fecal-oral mechanism (when urine, nasal and oral secretions of infected animals enter food, water), airborne and air-dust mechanisms, transmission of the pathogen by contact is established when blood or urine drops enter microtraumas on the skin and mucous membranes. Transmission factors can be virus-contaminated medical instruments.

Hemorrhagic fevers Lassa, Marburg and Ebola are related to nosocomial (nosocomial) infections. The natural susceptibility of people is high and does not depend on sex and age.

Yellow fever is a natural focal disease with a transmissible mechanism of transmission that is characterized by acute febrile flow, general intoxication, hemorrhagic syndrome and necrotic liver damage.

The causative agent of yellow fever belongs to the genus Flavoviruses, the family of Togaviruses. The virus grows well on tissue media, especially on the chorionalantoic medium, which is used to produce the main vaccine strain 17-D.

Yellow fever is recorded: in Africa: Angola, Benin, Burkina Faso, Gabon, Burundi, Cameroon, Côte d'Ivoire, Democratic Republic of the Congo, Ghana, Guinea-Bissau, Guinea-Bissau, Kenya, Congo, Côte d'Ivoire, Liberia, Niger, Nigeria, Tanzania, Rwanda, Sao Tome and Principe, Senegal, Somalia, Sudan, Sierra Leone, Togo, Uganda, Central African Republic, Chad, Equatorial Guinea, Ethiopia; on the American continent: Bolivia, Brazil, Venezuela, Guyana, Colombia, Panama, Peru, Suriname, French Guiana, Ecuador.

There are two types of foci: jungle (primary or natural, zoonotic) and urban (secondary, or synanthropic, anthroponous). The carrier of the virus in natural foci are various animals - monkeys, opossums, armadillos, anteaters, marsupials, rodents. The carriers of the virus are mosquitoes of certain species. In Africa, the main carrier of the virus are monkeys, the mosquito is vector of the virus. In the countries of South America, the main carrier of the virus are monkeys, mammals, leading a terrestrial way of life. The transmission of the virus involves mosquitoes of some species.

The source and carrier of infection in the urban type of disease is only a sick person, the main carrier of the virus from a sick person to a healthy one is a mosquito. The expression of a person is possible even if the patient's blood enters the skin, which has injuries and micro-trauma, or mucous membranes of a healthy person.

2. Learning objectives of the lesson (indicating the level of mastery that is planned):

2.1. The student must have a presentation (to read): ***α - 1***

1. About the statistical data on the prevalence of hemorrhagic fevers Lassa, Marburg, Ebola, as well as yellow fever.

2. About specific methods of research, which are used to diagnose hemorrhagic fevers, rules of material intake.

2.2. The student should know: ***α – 2***

1. Etiological agents that cause hemorrhagic fever, the prevalence of these diseases in different regions of the world.

2. Epidemiology of hemorrhagic fevers.

3. Pathogenesis of hemorrhagic fevers.

4. Clinical manifestations in the typical course of hemorrhagic fever Lassa, Marburg, Ebola, yellow fever.

5. Laboratory diagnostics of hemorrhagic fevers, rules for harvesting biological material from patients.

6. Principles of treatment of hemorrhagic fevers.

7. Rules of discharge of patients with hemorrhagic fevers.

8. Principles of personal prophylaxis when suspected of hemorrhagic fevers.

9. Prognosis of hemorrhagic fevers.

10. Tactics of the doctor's behavior in case of suspicion of hemorrhagic fevers.

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

1. Observe the basic rules of work at the patient's bed.

2. Collect anamnesis of the disease with the evaluation of epidemiological data

3. To examine the patient and identify the main symptoms and syndromes of hemorrhagic fever Lassa, Ebola, Marburg, yellow fever, substantiate the clinical diagnosis.

4. On the basis of clinical examination, it is timely to identify possible complications of hemorrhagic fevers, urgent conditions.

5. To plan a laboratory and instrumental examination of the patient.

6. Interpret the results of laboratory examinations.

7. Correctly evaluate the results of specific diagnostic methods.

8. To make an individual plan of treatment taking into account epidemiological data, the stage of the disease, the presence of complications, the severity of the condition, allergic history, concomitant pathology, to provide emergency care

9. To give recommendations on the regime, diet, examination, observation in the period of reconvalescence.

10. Obtain medical documentation on the fact of establishing a preliminary diagnosis of hemorrhagic fevers (emergency notification).

3. Materials for classroom independent work.

3.1. Materials for self-control

3.1.1. Questions for self-control **α=1**

1. To which group of infectious diseases are haemorrhagic fevers Lassa, Marburg, Ebola, yellow fever?
2. Source of infection in hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
3. Ways of transmission of hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
4. Pathogenicity factors of pathogens of hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
5. Sources of infection and carriers of hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
6. Pathogenesis of hemorrhagic fevers Lassa, Marburg, Ebola.
7. The pathogenesis of yellow fever.
8. Clinical manifestations of hemorrhagic fever Lassa.
9. Clinical manifestations of hemorrhagic fevers Marburg and Ebola.
10. Clinical manifestations of yellow fever.
11. Changes in the hemostatic system in hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
12. Complications causes of death in hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
13. Hemogram with hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.
14. Methods of specific diagnosis of hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.

15. Principles of therapy for hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever. Preparations, their doses, ways of administration.

16. Specific prevention of yellow fever.

17. Clinical and epidemiological features of hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever, differential diagnosis.

18. Rules for discharge from the hospital of patients with hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.

19. Organizational tactics of the doctor for suspected hemorrhagic fevers Lassa, Marburg, Ebola, yellow fever.

3.1.2. Test level: choose the right answers **α=2**

1. The source of infection with yellow fever is:

A. Sick man

B. Mice

C. Dogs

Д. Parrots

E. Sheeps

2. What is the main mechanism for transmission of yellow fever?

A. Air-droplet

B. Contact

C. Fecal-oral

Д. Transmissible

E. Parenteral

3. What is the main method of specific diagnosis of yellow fever?

A. Bacteriological culture of blood

B. The complement fixation reaction

C. Bacteriological examination of bile

Д. Agglutination reaction

E. Sputum microscopy

4. What is used for the specific prevention of yellow fever?

A. Specific immunoglobulin

B. Specific serum

C. Anatoxin

Д. Killed vaccine

E. Live vaccine

5. What is being done for the non-specific prevention of yellow fever?

A. Deratization

B. Boiling and sterilization

C. Disinsection

Д. Disinfection

E. Nonspecific prevention is not carried out

6. Name the timing of hemorrhagic syndrome with hemorrhagic fever Marburg:

A. 12 hours after the onset of fever

B. On day 3-4 of the disease

C. At the end of the second week of the disease

Д. At the end of the first week of the disease

E. In the first hours of the disease

7. What is used for the purpose of etiotropic therapy for hemorrhagic fever Marburg?

A. Etiotropic therapy is not developed

B. Acyclovir

C. Ribavirin

Д. Neurromidase inhibitors

E. Tetracycline

8. What is the main mechanism of infection with the Marburg fever?

A. Air-droplet

B. Contact

C. Fecal-oral

Д. Transmissible

E. Vertical

9. Who is the natural reservoir of the Marburg fever?

A. Mice

B. Rats

C. Monkey

Д. Dogs

E. Cattle

10. The main method of prevention in contact (suspicion) for Marburg fever?

A. Acyclovir

B. Doxycycline

C. Vaccination

Д. Use of a protective suit

E. Specific immunoglobulin

11. The drug of choice for etiotropic therapy of Lassa fever is:

A. Ribavirin

B. Acyclovir

C. Doxycycline

Д. Specific immunoglobulin

E. Etiotropic therapy is not developed

12. Specify a characteristic change in the hemogram in patients with Lassa fever:

A. Lymphocytosis

B. Lymphopenia

C. Thrombocytosis

Д. Increased hemoglobin level

E. Aneosinophilia

13. Lassa fever is transmitted from person to person by all mechanisms, except for:

A. Sexual

B. Contact

C. Parenteral

Д. Airborne droplets

E. All options are correct

14. The causative agent of the Lassa fever belongs to the family:

A. Herpesviruses

B. Flaviviruses

C. Bunyaviruses

Д. Togaviruses

E. Arenaviruses

15. What material is used for a specific diagnosis of Lassa fever?

A. Blood

B. Cerebrospinal fluid

C. Sputum

Д. Urine

E. All biological fluids

16. What is the most informative method of specific diagnosis of Ebola?

A. Microscopy of a thick drop of blood

B. PCR

C. Microscopy of a thin drop of blood

Д. Bacteriological examination of blood

E. Bacteriological examination of urine

17. Who is the main source of infection with Ebola?

A. Mice

B. Rats

C. Dogs

Д. Monkey

E. Mosquitoes

18. What is the main method of specific prevention of Ebola?

A. Live vaccine

B. Killed vaccine

C. Specific immunoglobulin

Д. Ribavirin

E. Specific prevention is not developed

19. The exanthema of Ebola is represented by:

A. Spots and papules

B. Vesicles

C. Roseola

Д. Ulcers

E. Spots

20. What is the main mechanism for the transmission of Ebola?

A. Fecal-oral

B. Air-droplet

C. Contact

Д. Airborne dust

E. Vertical

Standards of correct answers

1.	A	6.	B	11.	A	16.	B
2.	Д	7.	A	12.	B	17.	Д
3.	B	8.	B	13.	Д	18.	E
4.	E	9.	C	14.	E	19.	A
5.	C	10.	Д	15.	E	20.	C

3.1.2. Tasks for self-control **α=2**

Task 1.

Patient A, 25 years old was hospitalized in an infectious hospital on the third day of illness. Complaints on admission to high fever, headache, pain in the muscles, lower back, nausea, vomiting. Objectively: icteric sclera, photophobia, lacrimation. The skin of the face and neck is hyperemic, the skin is dry, hot to the touch. Hepatomegaly. Pulse is frequent, mild. From the epidemiological anamnesis: a week ago he returned from South Africa, where he was bitten by mosquitoes.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.
4. Prevention.

Task 2.

The patient, 25 years old, returned from Liberia 3 days ago, where he served in the UN peacekeeping unit. On the plane, he felt unwell, a headache, a sore throat. At home he

measured the body temperature, which was subfebrile. The patient did not seek medical help, he took aspirin on his own. On the second day of the disease headache and general weakness increased, body temperature rose to 39.5-40°C. The ambulance was taken to the infectious diseases hospital.

On admission, the patient was vomited three times, twice a loose stool without pathological impurities, a patient coughing, sputum with a blood admixture. Objectively: the body temperature is 40°C, the swelling of the face and neck, generalized lymphadenopathy, edema of the posterior pharyngeal wall, hyperemia of the pharyngeal mucosa ulcers. Frequency of respiratory movements is 25 / min. Breath vesicular, in the lower sections weakened, moist sonorous rales. Pulse - 90 / min., BP 90/60 mm Hg. The heart sounds are muffled. The liver is +7 cm, sensitive for palpation.

1. Provisional diagnosis.
2. Plan test.
3. Carry out differential diagnostics.
4. Treatment.

Task 3.

A 26-year-old patient turned to the clinic. Objectively: generalized lymphadenopathy, more pronounced increase in the cervical lymph nodes. Along with the petechial rash, papules and spots are noted. Tachycardia, the borders of the heart are expanded, heart sounds are muffled, blood pressure is lowered. Shortness of breath, cough, stitching pain in the side, shortening of the percussion sound, dry and wet wheezing. Radiographic examination of the lungs revealed an infiltrative change. Necrotizing pharyngitis is revealed on examination of the mucous membrane of the oropharynx. The stool is plentiful, watery. The liver is enlarged, painful on palpation. From the side of the nervous system - meningeal signs are positive (with the normal composition of the cerebrospinal fluid). The patient complains of severe headache, dizziness, tinnitus.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.

Task 4.

The patient of 35 years was acutely ill, there was a fever, a headache, expressed generalized myalgia, prostration. From the first days of illness there was vomiting and a loose stool of a watery nature. On examination: pharyngitis, conjunctivitis, inflammatory lesions of the genital organs. On 4-5-th day of the disease, there was a maculopapular rash. Manifestations hemorrhagic diathesis at the second week. There were bleeding from the gums, small intestine, urinary tract. In vomit masses - the admixture of blood. Parallel signs developed polyorganous lesions - liver, kidney, myocardium. High fever decreased after 8-10 days of illness, but gave a second peak by the end of the second week from the onset of the disease. Diarrhea is long, preserved and after normalization of body temperature, leading to severe dehydration.

1. Clinical diagnosis.
2. Plan test.
3. Treatment.

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