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Department of Infectious diseases

METHODOLOGICAL GUIDE

CONGO-CRIMEAN HEMORRHAGIC DISEASE FEVER

for students studying in the specialty
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Congo – crimean hemorrhagic disease fever-2020

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Among infectious diseases, due to their high contagiosity and severe, often catastrophic course, there is a group of diseases called especially dangerous (OOI) or conventional. In cases of detection of patients suspicious of OOI, the tactics of first-contact doctors, who are most often doctors of the ambulance service and district therapists, are strictly regulated by the Directive orders of administrative bodies. The main goal of this tactic is to prevent the spread of the disease and provide the patient with the necessary medical care as quickly as possible. Failure to comply with these rules entails serious legal liability of the doctor.

OOI includes cholera, plague, smallpox, and contagious viral hemorrhagic fevers, including Congo-Crimean hemorrhagic fever (CCHF). Foci of Congo-Crimean hemorrhagic fever were registered in Krasnodar, Stavropol territories, Astrakhan and Rostov regions, on the territory of Central Asian republics and Kazakhstan, as well as on the territory of Bulgaria, Yugoslavia, Hungary, a number of Asian and African countries. Due to extensive international and interregional contacts, it is possible to import these diseases to non-endemic areas, so all doctors should be well aware of early clinical manifestations and methods of working with patients suspected of OOI.

Introduction

The purpose of the lesson is to master the early, suggestive clinical signs of Congo-Crimean hemorrhagic fever (CCHF) and the tactics of diagnostic and anti-epidemic actions of the district therapist, as a first-contact doctor; familiarization with the principles of treatment, prevention and methods of laboratory confirmation of this disease.

Generalities.

As a result of studying the topic, the student should know:

1) Etiology and epidemiology of Congo-Crimean hemorrhagic fever.
2) Pathogenesis and pathological anatomy of KGL.
1) Clinical classification and characterization of various clinical forms of the disease.
3) Methods of laboratory diagnostics of KGL.
4) Methods of etiopathogenetic therapy.
5) Measures in the focus of infection and prevention issues.

The student should be able to:

1) Examine the patient and identify clinical and epidemiological signs indicating the disease of KGL.
2) Carry out the first measures aimed at limiting the spread of infection in the focus.
3) Inform about the detection of a patient who is suspected of taking KGL.
4) Organize patient care and provide first aid at the pre-hospital stage.
5) Carry out measures for personal prevention of the quarantine disease.

History

KGL is an acute transmissible infectious disease of viral etiology, endemic to a number of geographical zones, characterized by fever, severe intoxication and hemorrhagicsyndrome.

KGL was discovered and isolated in an independent nosological form in 1944-1945, when there was an outbreak of a severe febrile disease with a pronounced hemorrhagic syndrome in the Crimea. In 1945-1949, cases of the disease with hemorrhagic syndrome were described under various namesin the Krasnodar and Stavropol territories, Astrakhan and Rostov regions, on the territory of the Central Asian republics and Kazakhstan. During the same period, foci of cchl were identified in Bulgaria, Yugoslavia, Hungary, and a number of Asian and African countries.

Etiology

The causative agent of KGL was discovered in 1945 by M. L. Chumakov. At his suggestion, the disease was called "Crimean hemorrhagic fever".

Strains of the virus were isolated from the blood of patients and from the tick Hyalomma marginatum margi-natum. KGL virus belongs to arboviruses, it is part of the Bunyaviridae family Bunyaviridae(genus nairovi-rus). Spherical virions with dimensions of 90-100 nm have an outer shell containingmy lipids. The virus genome is represented by three fragments of single-stranded RNA. The KGL virus quickly dies when exposed to ether, chloroform; at t=56° C, it completely loses its infectability after 5-10', and when boiled-almost instantly (b / W 30"). The virus is well preserved in a frozen state in a material containing proteins. It is unstable to commonly used disinfecting solutions, has medium resistance to environmental factors (insolation, humidity...) Most virus strains do not cause cell destruction in an infected culture. The KGL virus is characterized mainly by cytoplasmic localization in cell culture. Specific antigen luminescence is detected by the method of fluorescent antibodies (MFA) also in the nuclei and nuclei of cells. The antigen can also be found extracellularly.

The viral etiology of KGL in the Astrakhan region was first established in 1955 by Chumakov, A. P. Belyaeva and E. V. Leshchinskaya. In 1977, a strain of the KGL virus "Drozdov" named after the patient was isolated from the blood of a KGL patient in Astrakhan. In 1968, the Smirnova CE. the immunological relationship of the "Drozdov" strain with other strains of the KGL virus isolatedin the Rostov and Samarkand regions (the "Kash"and "Khoja"strains) was established with the co-author.

In 1956, in the Congo, a viral agent called the Congo virus was isolated from the blood of a fever-stricken boy, which is similar in antigenic structure and biological properties to the KGL virus. In 1969, the antigenic affinity between KGL and Congo viruses was established. Unlike KGL, the disease caused by the Congo virus occurs without a hemorrhagic component, relatively rarely detected in humans, but the virus is often detected in animals.

Strains of KGL - Congo viruses cause disease and death of newborn white mice and rats with intracerebral infection, which is used to isolate the pathogen from patients and ticks. Adult white mice, rats, and other animals do not get sick after in-

oculating them with the KGL virus, but they respond to infection with the production of specific antibodies, so they are used toobtain specific immune serums.

Epidemiology

KGL is a zoonotic natural-focal infectious disease. Its characteristic feature is that it is confined to certain landscapes: semi-desert, steppe, forest-steppe, and soldered areas. In General, such areas are unsuitable forplowing and are used as grazing grounds for livestock that feed adult STAtions Of hyalomma Ixodes ticks Hyalomma. The main reservoir of the virus is many types of pasture mites, which transmit the virus to their offspring by transovarian and transfase pathways and during metamorphosis. Animals serve as a reservoir of the virus and the period of viraemia to infect ticks parasitizing on them.

Birds that feed on the ground are mass feeders of larvae and nymphs of two- and three-host tick species, whose imagos usually parasitize ungulates. However, birds infected with the KGL virus do not have virosemia, so their epizootological role in the foci of KGL is to maintain the number of vector populations, in local territorial dessimination (of the virus) and its transcontinental introduction.

In ticks, the virus is stored throughout life, including during the off-season period. The causative agent of KGL is transmitted to humans by transmissible and contact pathways, possibly aerogenic.

The main epidemiological role in human transmission of the KGL virus belongs to adult ticks (imagos), which often attack humans. Adult ticks are parasitic on cattle, sheep, goats, etc. The larvae and nymphs are fed by birds and hares. The height of parasitization of ticks H.marginatum occurs in April-July, withmaximum activity in may. The mites ' habitats are pastures, animal milking sites, rook nesting sites, gardens, vegetable gardens, hay and straw ricks. Climate conditionshave a significant impact on the existence of ticks. Under favorable climatic conditions, there is an increase in the number of mites.

Human infection with KGL occurs as a result of sucking a tick or when crushingticks removed from livestock with their hands. Less common are cases of infection with the KGL virus through contact with the blood of a sick person or animal. Patients with KGL are the most dangerous forokruzhayushchy during the first days of the disease, especially from the moment of bleeding.Health workers and family members of patients with CRL are usually infected during the first aid and caregiving of patients. There are also cases of intra-laboratory infection of personnel when working with infected material containing the KGL virus.

The incidence of KGL is characterized by spring-summer seasonality, which is due to the rise in the number of ticks - carriers of the virus.

KGL is more common in rural areas, and less common in urban areas when they go out for field work, fishing, or recreation.

People are susceptible to KGL regardless of age. The most common cases are young and middle-aged people (from 20 to 50 years old), who are attacked by ticks due to their profession. Men get sick more often.

The incidence of KGL is sporadic, with less frequent outbreaks. It is characterized by a low level of collective immunity to the virus in healthy people. Among the

population, there is the formation of a small immune layer from the number of patients with KGL. When examining reconvalescents, it was found that antibodies to the virus persist for more than 5 years afterthe disease was transferred. After the disease, people acquire lifelong immunity to KGL.

The endemic area of the KGL is the Astrakhan region. Among the patients, the majority were men (78%), women were 22%. However, a significantnumber of elderly people (32%) were ill. Among those who applied, 63% of patients report casesof tick scurvy.

Pathogenesis and pathomorphology of KGL

The virus enters the human body when blood is sucked by an infected tick. The pathogen is fixed and multiplies in the vascular endothelium, epithelial cells of the liver, kidneys and reticuloendothelial cells. At the same time, vasculitis develops with mainly edematous and destructive damage to the microcirculatory bed up to fibrillar necrosis and loosening of membrane structures. After the accumulation of the virus in the RES, virosemia occurs, which causes a General infectious manifestation of the initial period of the disease. As a result of the direct action of the virus on the vascular endothelium, as well as in connection with the defeat of the hypothalamic region and the adrenal cortex, vascular paresis occurs, their permeability increases, and hemostasis is disrupted. Clinically indicated hemodynamic and coagulation shifts are manifested by symptoms of hemorrhagic diathesis.

To date, many aspects of pathogenesis remain unclear.

Pathomorphological changes have a certain dynamics of development, coinciding with the clinical stages of KGL.

In the incubation period, a non-specific inflammatory reaction develops in the tick bite zone, which is indistinguishable from the cellular composition of exudate from bites of healthy ticks. The second period (1-4 days of illness), corresponding to the beginning of the disease in the clinic, is characterized by dystrophic-necrotic changes in organs and tissues. These processesoccur simultaneously and mutually enhance tissue destruction. They are caused by sharp intoxication due to the accumulation of viral antigen in a number of organs and its cytopathogenic action.

The third period is generalized inflammation with a pronounced hemorrhagic component (5-9 days of illness).

It is characterized by generalized inflammatory changes with a predominance of the hemorrhagic component. On the skin and mucous membranes there is a large-spotted or hemorrhagic rash, in the internal organs - multiple hemorrhages. Hemorrhagic wall infiltration in a number of hollow organs was combined with the presence of a large amount of liquid blood in their lumen. At the same time, localized edema of subcutaneous fat in the lumbar region, as well as the fat capsule of the kidneys and retroperitoneal tissue was detected.

the pathological essence of the disease is manifested primarily by circulatory disorders and increased vascular permeability. These processes are the main links in theGeneral chain of generalized serous-hemorrhagic inflammation, which cause a pronounced hemorrhagic syndrome.

With KGL, various hemorrhagic manifestations develop: hemorrhages on the

skin and mucous membranes, in internal organs, bleeding gums, hemorrhages inthe injection sites, nasal, uterine, gastrointestinal bleeding.

The period of secondary changes in the organs (10-12 days of the disease). This period is expressed only in severe course of KGL. Long-term local changes, most often caused by circulatory disorders or inflammation caused by the addition of secondary bacterial flora, come to the fore. The tendency to bleed decreases. However, during the autopsy, hemorrhages of various sizes and shapes are found everywhere. Edema reaches the highest degree of severity in the parathyroid and adrenal tissues of the lumbar region. A small amount of transudate is found in the pericardium, abdominal and pleural cavities. Histologically, there are pronounced alterative - necrotic changes in all organs, which are usually manifested by necrotic and fat destruction, as well as small-focal necrosis. Fibrinous necrosis of vascular walls is a morphological reflection of changes in the reactivity of the body and hypersensitivity of tissues. Often necrotic changes are associated with dyscirculatory disorders. Probably, necrosis is caused by the direct damaging effect of the virus.

In 2\3 patients who died from KGL, bronchopneumonia was noted, but it was of crucial importance in the pathogenesis in isolated cases. Pneumonia was hemorrhagic or purulent in nature. Along with hemorrhagic phenomena, autoinfection against the background of fullness and pulmonary edema is important.

The clinical picture was dominated by symptoms of insufficiency of one or more organs (lungs, heart, liver).

The reparative period usually begins with the 8th day of illness. Regeneration occurs without the development of foci of sclerosis. At autopsies of corpses. in the case of those who have died from KGL, the external examination of the corpse is of diagnostic significance.

Characteristic are hemorrhages in various areas of the skin, from purplish-purple to brownish-red. In the places of injection, applying the tourniquet, there are extensive hemorrhages and bruises. The main hemorrhagic changes in KGL are caused by a pronounced hemorrhagic syndrome. During histological examination, hemorrhagic disorders and increased vascular permeability were observed in all organs. The vessel walls were edematous, the endothelium swollen, sometimes necrotic. Argyrophilic fibers are swollen, fragmented. Violations of the tinctorial properties of the elastic shellof the nipples and capillary basal membranes were observed. In ganglionic cells was observed shrinkage cytoplasma, pyknosis of nuclei, neuronophagia. In gipotalamicescoy area was observed circulatory disturbances: congestion, perivascular hemorrhage, stasis, stromal edema, swelling pericellular. The ganglion cells are swollen, some of them with tigrolysis and pycnosis of the nuclei. In theparasympathetic division of the autonomic nervous system, the changes were less pronounced, but even there there was cell proliferation and vacuolation.

In all internal organs: heart, lungs, stomach, intestines, kidneys - during microscopic examination, diapedesis hemorrhages, hyperemia, stasis, edema were detected. In some cases, necroses were detected In the liver parenchyma, in the kidneys - granular dystrophy of the epithelium fromthe branched tubules, in isolated cases - necrosis of the epithelium. Hyaline cylinders were found in the lumen of the tubules. The basement membranes of the glomerular capillaries were swollen. In the internal or-

gans, there was a sharp tissue anemia, serous edema. In the brain - foci of hemorrhages in subcortical nodes. The heart muscle on the incision is dull, flabby, grayish-yellow or grayish-brown in color.

Hemorrhages were constantly observed throughout the gastrointestinal tract, starting with the oral cavity, where hemorrhages were detected on the hard palate, gums, and tongue. The mucosa of the stomach, small and large intestines had a brownish-red or purplish color with hemorrhages. In the lumenof the larynx and intestines, most of the deceased contained blood (up to 1-1. 5 liters). The liver remained normal size, or was moderately enlarged. The liver parenchyma on the incision was dull, sometimes with a clay tint. Kidneys, as a rule, were of normal size, sometimes dense, with the formation of hematomas. The spleen is also full-blooded, with hemorrhages in the parenchyma and on the surface of the organ.

KGL clinic

During the infectious process in KGL distinguish the following periods of illness:

- incubatory
- initial (pre-hemorrhagic)
- the period of hemorrhagic manifestations with organic changes (the height of the disease)
- period of convalescence.

One of the characteristic symptoms that largely determine its severity and outcomeis hemorrhagic manifestations. However, some patients (7-9%) may not have hemorrhagic syndrome. In this regard, there are clinical formsof the disease with and without hemorrhagic manifestations.

KGL, depending on the degree of intoxication and severity of hemorrhagic manifestations, can occur in three forms:

- heavy
- medium weight
- easy

As with other arbovirus infections, the disease process can proceed in the erased and inapparent forms.

In General, the clinical classification is as follows:

I. KGL with hemorrhagic syndrome:

1. Severe form:

a) no cavity bleeding

b) with cavities bleeding

2. Moderate form:

a) no cavity bleeding

b) with cavities bleeding

3. Easy shape.

II. KGL without hemorrhagic syndrome:

1. Medium-heavy form
2. Mild form.

III. Inapparently form (subclinical)

Clinically, the form without hemorrhagic manifestations presents significant difficulties for diagnosis and is characterized by fever and toxicosis. The diagnosis is usually made after detection of antibodies to the KGL virus in the serum of patients.

The incubation period for KGL is 1-14 days, usually 3-7 days. When analyzing the incidence of KGL according to the data of The Astrakhan Regional infectious diseases hospital, no mild forms of the disease were detected. Medium-heavy forms prevailed (60%), in which non-profuse nosebleeds were noted. Severe forms were observed in 40% of cases and were accompanied by copious oral bleeding (nasal, gastrointestinal, uterine) and other complications.

The disease begins acutely, some patients can even indicate the hour when they fell ill. Those who are ill have a terrific chills and fever. The temperature rises to 39°-40°. in the pre-hemorrhagic period, which is often difficult to diagnose, there are a number of non-specific symptoms that occur in other infectious diseases: joint pain, muscle aches, body aches, severe headaches, weakness, bruising, rapid fatigue, possible catarrhal phenomena, pain in the calf muscles, in the abdomen, repeated vomiting, low back pain. In many patients there is hyperemia of the face and neck, sclera, conjunctivitis, SPECTARIS-ness of the sclera, subscleral hemorrhage.

A constant symptom of KGL is fever (39° - 40°), which has a "two-humped" temperaturecurve. Characteristic for KGL is a decrease in temperature at the time of bleeding and hemorrhage, more often on the 3-5 day of the disease.

On the 3rd-5th day of the disease, the peak period of the disease occurs, characterized by hemorrhagicmanifestations: on the skin and mucous membranes, hematomas at the injection sites. Along with the rash, patients have bleeding gums, nosebleeds, gastrointestinal bloodflows (vomiting of "coffee grounds", tar-like stool). Bleeding continues fromseveral hours to 2-5 days. In some patients, there may be bleeding from the ears, bloodspitting when coughing, and uterine bleeding. With the appearance of hemorrhagic syndrome, the condition of patients deteriorates sharply. Their appearance changes. Hyperemia of the face is replaced by paleness, acrocyanosis is noted.

One of the main manifestations of hemorrhagic syndrome in KGL is a rash. It appears on 2-4 days, is located symmetrically on the trunk, limbs, is represented by petechiae and roseoles. More often it appears on the side surfaces of the trunk, thighs, shins, feet, palms, upper limbs. Petechial rash does not protrude above the skin surface and does notdisappear when pressed. Elements of a rounded shape, with clear borders, without a tendency to merge, are purple, rich purple or bright red. Often there was an erythematous-roseolous rash of pale pink color, prone to merging with the formation of extensive erythematous sites. The borders are not clear, the shape is rounded or irregular. In most cases, white or mixed dermography was detected. At the same time with the appearance of a rash or 1-2 days before, patients revealed enanthema on the mucous membrane of the soft and hard palate, the mucous membrane of the cheeks.

Primary affects at the site of tick bites in most cases were single and were de-

tected on the lower extremities. There were no subjective sensations at the bite sites. Primary affects had a rounded shape, bright red color, from 5-8 mm in diameter, did not rise above the surface of the skin. In the center of the affect, there is a brownish crust with a narrow border of epithelial detachment along the periphery. After the resolution, pinpoint skin atrophy was detected. Often affect is accompanied by regional lymphadenitis. The lymph nodes are tightly elastic in consistency, painless, no bigger than a pea in size.

Changes in the cardiovascular system during KGL are characterized by muted-tones, deafness, and a decrease in blood PRESSURE. The pulse rate in most patients does not correspondto an increase in temperature, 50-60 beats per minute. There may be a sharp increase PSin PS (120-130 beats per minute). Vascular collapse may occur. The ECG reveals dystrophic changes in the myocardium, impaired conduction.

Respiratory organs are also involved in the pathological process: pneumonia can develop.

Changes in the gastrointestinal tract are constantly observed: dry mouth, anorexia, nausea, vomiting. There may be bloody vomiting, tarry stools, a" flaming throat", loose, bleeding gums. Dryness of the tongue, overlaid with brown plaque, soreness on palpation in the right hypochondrium is detected. In some cases, there was an increasein the liver, as well as intense jaundice, which can be parenchymal or hemorrhagic.

A significant role in the pathogenesis of KGL is assigned to kidney damage. Patients complain ofpain in the lumbar region. There is a positive symptom of Pasternatsky, in some cases-oliguria. In KGL, functional renal failure is often detected, and manifestations of acute renal failure are explained by massive bleeding and hematomas in the peri-renal fiber, characterized by an increase in the concentration of creatinine, residual nitrogen and urea in the blood, up to the development of anuria.

Lesions of the Central and autonomic nervous systems are a characteristic feature of KGL. All patients complain of headache, dizziness, weakness, adynamia, and lethargy. In some patients, there is a rigidity of the occipital muscles and a positive-Kernig symptom. Possible loss of consciousness, delirium, psychosis.

The disorder of the autonomic nervous system is expressed by hyperemia of the skin, mucous membranes, dryness of the oral mucosa, bradycardia, hypotension, narrowing of the pupils, sluggish reaction to light . The vascular nature of the CNS lesion is characteristic. This is evidenced by the absence of an inflammatory reaction in the cerebrospinal fluid.

Changes in peripheral blood are of great diagnostic significance: from the first days, leukopenia is characteristic, but leukocytosis is also possible, and thrombocytopenia also occurs. Often, a sharp drop in the number of platelets during KGL coincides with bleeding. In most patients with hemorrhagic syndrome, a decrease in the number of red blood cells and hemoglobin was observed, anemia developed, the number of red blood cells decreased to 1.8 x 10 g/l. The period of convalescence is characterized by normalization of temperature, disappearance of intoxication, reverse development of hemorrhagic syndrome. Recovery in many people is delayed. Pathological changes in blood and urine parameters persist for a long time.

The mortality rate for KGL is 6% - 12%, and for the hemocontact path of in-

fection - up to 60%. Prognostic criteria of severe and complicated course of KGL.

- fever over 39°C;
- prolonged fever, presence of a "double-humped" temperature curve;
- athralgia myalgia;
- presence of primary affect at the site of tick bite;
- abundant, spreading to the palms and feet, petechial-roseolous rash;
- tachycardia and tone deafness;
- abdominal pain, nausea, vomiting, loose stools;
- hepatomegaly, jaundice;
- pain in the lumbar region, oliguria;
- sudden General weakness, lethargy, drowsiness;
- mucosal bleeding and bleeding in the early stages of the disease;
- repeated and massive bleeding at the height of the disease;
- leucocytosis and thrombocytopenia at the beginning of the disease;
- elderly and senile age;
- staying in a Hyper-endemic zone;

Laboratory diagnostics.

Laboratory diagnostics of KGL is carried out by virological, serological and molecular-biological methods. Isolation and identification of the virus is carried out in the first days of the disease and in the period of Virology. Priority methods are PCR. KGL virus RNA is detected by reverse transcription polymerase chain reaction.The volume agglomeration reaction (RW) with an antibody polymer dry diagnosticum is also used to detectthe KGL antigen. For virological research, the blood of patients, the liquor and internal organs of those who died from KGL are used.

Serological methods are widely used for diagnostics: ELISA, RCS, RAO. Pairedblood sera of patients are examined by ELISA for the presence of IgM antibodies to the KGL virus using test systems.

Blood for serological studies is selected twice: on 5-7 and on 10-14 days from the beginning of the disease. For the diagnosis of KGL based on the detection of specific antibodies, it is sufficientto accurately detect IgM in one serum at a titer of at least 1:800. In all the observed patients, the diagnosis of KGL was confirmed by the detection of IgM antibodies by ELISA with a 4.8-fold increase in titer dynamics.

Differential diagnosis

As the reference diagnostic criteria of the KGL in the epidemiological survey is taken into account:

- stay in a natural hearth;
- spring and summer seasonality;
- tick bite;
- professional seasonality.

Laboratory data in the pre-hemorrhagic period:

- leukopenia with neutrophil shift;
- relative lymphocytosis;
- thrombocytopenia;

moderate increase in the number of red blood cells; proteinuria;

- microhematuria;
- cylindruria.

Laboratory data in the hemorrhagic period:

↑ thrombocytopenia, leukopenia, lymphocytosis, ↓CO ESR, increasing anemia, ↘prothrombin index, ↓amount of blood fibrinogen, ↘specific gravity of urine, proteinuria, macro-and microhematuria, cylinders.

KGL should be differentiated from Astrakhan rickettsiosis fever(ARL), hemorrhagic fever with renal syndrome (HFRS), Ku fever, leptospirosis, meningococcemia, typhoid and typhoid fever, West Nile fever and other arboviral infections, as well as thrombocytopenic purpura, hemorrhagic vasculitis, surgical and hemorrhagic diseases.

Astrakhan rickettsiosis fever is similar to KGL. Common to them are: epidemiological data, a number of clinical symptoms: acute onset, fever, intoxication phenomena, primary affect and skin rash. But the differences are that the primary affect in KGL is smaller (0.5-0.8 cm), in the center light, with a crust. Affect in ARL - a pink spot of 0.5-1.5 cm, in the center of it - erosion with a dark brown crust. The rash in ARL is more abundant, localized on the trunk and limbs, including the palms and feet. The rash in ARL is polymorphic: roseolous-papular, erythematous and only sometimes hemorrhagic. Fever a long "double-humped" curve is not characteristic. The diagnosis of ARL is confirmed by the reaction of indirect immunofluorescence with a specific antigen (RNIF).

Hemorrhagic fever with renal syndrome has a lot in common with KGL. Clinic similar, but in HFRS possible prodrome (2-4 days), then there ↑t^0 to 39-40 ° - 41°C, symptoms of lesions of the nervous system, vision: pain in eyeballs, photophobia, "grid" or "fog" before the eyes, reduced visual acuity, extensive subscleral hemorrhage. Characterized by pain in the lower back, a positive symptom of Pasternatsky. The fever lasts for 7 - 9 days. From 3 to 5 days, there is a period of hemorrhagic manifestations and renal failure. Hemorrhagic syndrome has the same manifestations as in KGL, but the leading one in the clinic is nephrotic symptoms: a sharply positive symptom of Pasternatsky, lower back pain, oliguria, proteinuria, hematuria, cylindruria, anuria, isohypostenuria develops. The urine contains high levels of creatinine, urea, and residual nitrogen. Uremia may develop. HELL↑↑, VD↑. To confirm the diagnosis, the method of fluorescent antibodies and enzyme immunoassay is used.

Often, patients with KGL are sent to the hospital with a suspicion of KU fever. There are a number of similarities: fever, Arthro - and millii, sclerenchyma, hepatomegaly. But Ku fever differs from KGL by a longer febrile period (up to 2-3 weeks or more), accompanied by repeated chills, sweating, retroorbital pain, the absence of exanthema and enanthema. They are characterized by changes in the respiratory system (Bronchitis, pneumonia). Almost constantly observed hepatosplenomegaly, possibly jaundice of the skin and sclera. The main difference between Cu fever and KGL is the absence of a hemorrhagic syndrome. In the blood: normocytosis or moderate leukocytosis, ↑ESR. Thrombocytopenia for cu fever is not characteristic. To confirm

Cu fever, an RSC with a Burnet antigen is used.

KGL has to be differentiated with leptospirosis, since they have a number of clinical similarities: acute onset, two-wave fever, intoxication, hemorrhagic syndrome. Pathognomonic signs of leptospirosis are muscle pain, especially in the calf muscles. On palpation: sharp soreness, restriction of movement. The rash in leptospirosis is polymorphic: erythromatous-roseolous, spot-papular, urticarial, small-point, petechial. Items tend to be mergers and forming erythematous field. After disappearance, peeling is observed, often bran-like. Often, herpes rashes on the lips and wings of the nose, jaundice of the skin and sclera are joined. Characteristic of kidney damage: pain in the lumbar region, a positive symptom of Pasternatsky, oliguria, anuria, the development of acute renal failure. It is possible to attach meningial syndromes, changes in the liquor. The diagnosis of leptospirosis is confirmed in the laboratory: detection of leptospir in blood, urine, CSF, isolation of leptospir culture. From 5-7 days, serological reactions (RSC and RL) are performed.

West Nile fever (WNF) is a naturally occurring focal arbovirus disease. The vectors are mosquitoes. There are a number of similarities, in particular, in the initial period. Lzi differs from KGL in its polymorphism of clinical manifestations: acute onset, high temperature, pain in the eyes, arthralgia, muscle pain, catarrhal phenomena from the upper respiratory tract, lymphodenopathy, CNS damage, repeated vomiting, drowsiness, hyperesthesia, inappropriate behavior. Typical: tremor of the hands, twitching of the muscles of the face and extremities, paresis and paralysis are possible, serous meningoencephalitis is characteristic, the appearance of rashes, individual hemorrhagic elements is possible. ELISA, rtga, and PCR are used for diagnostics.

Meningococcemia has a number of features in common with KGL; a distinctive feature is the appearance of a hemorrhagic rash on the first day of the disease. Localization of the rash-first on the thighs and buttocks, then on the trunk, upper limbs, the rash has a star-shaped shape, rises above the skin, blue or purple in color. Possible necrosis of the skin, fingertips, and earlobes. The diagnosis is confirmed bacteriologically and bakteriostaticheski, productive is the serological method of research, Phragmites.

Typhoid fever and KGL have many common features, but typhoid fever is more commonly reported in winter. With typhus, there is no primary affect, the fever is long (10-16 days), the symptoms of Central nervous system damage are more pronounced, and the development of typhoid status is possible: delirium, disorientation, hallucinations. There is a tremor of the tongue, lips, fingers, "symptom of the tongue" (Govorova-Godelier): when trying to show the tongue, the patient pulls it out with difficulty. The diagnosis is confirmed RSK, Phragmites antigen of Provasca.

KGL should be distinguished from typhoid fever and paratyphoid A and B. typhoid fever is characterized by a gradual onset: fever increases in a ladder-like manner, a rash appears on the 7-8 day, sparse, roseolous. Characteristic symptoms from the gastrointestinal tract: tongue overlaid on the back, bloating, rumbling in the ileocecal area, scavenger's symptom, constipation, diarrhea, intestinal bleeding: tar-like stool with blood clots of dark cherry color.

KGL often has to be differentiated with non-communicable diseases, accompanied by a rash, bleeding gums, and mucous membranes.

Platelet-derived purpura (Werlhof's disease). The similarity lies in the rashes that continue undulating. Frequent hemorrhages and bleeding of mucous membranes of the oral cavity. In some cases, there are hemorrhages in the membranes and substance of the brain, focal and meningeal symptoms, and a disorder of consciousness. There is blood in the liquor.Post-traumatic anemia develops. In the blood: pronounced thrombocytopenia, often critical, clot retraction is sharply reduced or absent. The bleeding time is increased. The amount of fibrinogen is normal.

With hemorrhagic vasculitis (Schonlein-Henoch's disease). The body temperature is raised sharply, the main symptom is a hemorrhagic rash, its location is symmetrical, it is possible to merge hemorrhages, there are often extensive hemorrhages with necrotic areas. There is a characteristic swelling of the joints, serous-hemorrhagic effusion, more often in the knee joint, hemorrhages in the peritoneum are accompanied by acute abdominal phenomena. Possible hemorrhagic nephritis, meningeal symptoms. The clinic is caused by pathology on the part of the vascular wall, not the blood. Platelet count is normal, retraction and bleeding time are not changed. In the course of differential diagnosis of KGL, acute surgical and gynecological diseases accompanied by bleeding, nausea, vomiting, and pain are excluded. Here, the epidemiological history and laboratory diagnostics, as well as the absence of primary affect, are of great importance.

Treatment of KGL

Treatment involves, first of all, hospitalization in separate wards or boxes. Strict bed rest is prescribed. Transportation of patients during the period of severe bleeding is contraindicated.

Diet-complete, sparing (table #10 on Pevsner), including vitamins P, C, A.

Prevention of bedsores is important.

Etiotropic therapy will be performed in the early stages (5-7 days). The antiviral drug "Ribavirin Meduna" is prescribed at a dose of 15 mg / kg. The recommended dose of 1000 mg per day in 2 doses (2 capsules in the morning and 3 in the evening) - 10 days. It is also prescribed "Cyclopheron" in a single dose of 0.5 g. in\in 1 time a day according to the scheme: for 1,2,4,6,8,12 days.

For ↑иммун immune potential - 100-300 mg of blood plasma reconvalescents, hyperimmune specific equine γглоб-globulin in a dose of 0.5-7.5 ml. Heterologous γglobulin is injected by the method Besredka.

Pathogenetic therapy is aimed at combating intoxication, hemorrhagic syndrome and infectious - toxic shock.

Detoxification therapy is prescribed: intravenously administered solution of 5% glucose, hemodesis, saline solutions in combination with cocarboxylase, ascorbic acid, potassium preparations.

Hemostatic therapy involves the introduction of freshly frozen plasma, gelatinol, and albumin. Hemotransfusions are performed: fresh citrate blood, erythrocyte and leucocyte mass, cryoprecipitate up to 10 times a day, platelet mass.

At the same time, drugs that seal the vascular wall are prescribed - 10% calcium gluconate solution, 5% ascorbic acid solution, ascorutin, nicotinic acid, proteolysis inhibitors, fibrinolysis. When bleeding: vicasol, dicinol, androxone 1-4 ml per day

3 times a day. Fibrinogen is prescribed when the fibrinolytic activity of the blood increases.

Local pressure bandages, tamponade, cold. When tamponade is used epinephrine (1: 2000 - 1:50000) and tampons hemoglobin or hemostatic sponge.

To maintain adequate hemodynamics, cardiamine, caffeine, sulfokamfokain, korglikon, strofantin are prescribed. With the aim of desensitizing appointed antihistamines: diphenhydramine, diazolin, pipolfen, tavegil, suprastin, prednisolone.

With a severe course of KGL, corticosteroid drugs are prescribed: prednisone, dexamethazole. Prednisone b\V from 1-3 mg per 1 kg of weight. The course of treatment is 7-10 days. When vomiting-0.5% solution of novocaine 1 UF. spoon 3-4 times a day, cerucal 2 ml intravenously. If complications develop, anti - bacterial therapy is prescribed.

Complications of KGL

Complications are divided into early (nasal, gastrointestinal, uterine bleeding, infectious-toxic shock, DIC-syndrome, myocarditis, hepatitis) and late (post--hemorrhagic anemia, pneumonia, pulmonary edema, encephalopathy, brain edema, subarachnoid hemorrhage, acute renal failure, soft tissue abscesses). The most common complication is focal pneumonia. There is bradycardia, tachycardia, hemoptysis, increased cough, chest pain, wet wheezing.

Prevention

Non-specific prevention of KGL provides for transmissible transmission of infection: anti-mite treatment of the territory, livestock in order to reduce the numberof carriers, regulating the number of main feeders of ticks and reservoirs of viruses (hares, rooks, hedgehogs). Livestock rooms are treated with 1.5% water solution of chlorophos, 40% baitex solution, 10% fury solution. Collected ticks are destroyed in kerosene, in a can of water or burned.

In the centers of KGL, personal prevention measures should be observed: wearing an anti-tick costume. In the case of suction of the tick, it is necessary to drip vegetable oil on it and after 10-15 minutes, turning from side to side, remove the tick. To repel ticks, it isnecessary to use repellents.

Linen and clothing of the patient are collected in an oilcloth bag and subjected to disinfection in a steam chamber (at t° + 120°C for 30 minutes). To disinfect the patient's secretions containing blood, use dry chlorine lime, mix and leave for 2 hours. The chambersare treated with a 3% solution of chloramine. Precautionary measures should be observed at all stages of examination of the patient, sampling of material, during laboratory tests. When opening corpses of those who died from KGL, it is necessary to ensure full protection of the skin and mucous membranes from the ingress of cadaver material.

A significant place in the complex of measures for the prevention of KGL should be occupied by sanitary and educational work, especially in endemic areas, at enterprises and branches of animalhusbandry and agriculture.

Control question.

1) Properties of the causative agent of PT.
2) Natural foci of quarantine infections.
3) Features of KGL epidemiology.
4) The pathogenesis of PT.
5) Suggestive symptoms of KGL.
6) Methods of laboratory diagnostics of KGL.
7) Differential diagnosis of KGL.
8) Principles of treatment of patients with KGL.
9) Actions of the 1st contact doctor in identifying patients with suspected

Control tasks.

Task № 1.

Patient O. 45 years old, a geologist, fell ill a week after returning from Africa. In the first two days, I noticed malaise, pain in my eyes, and a subfebril temperature. Then the body temperature began to increase and reached 39-40°C, joined a strong headache, muscle pain, nausea, sore throat, stomach, in connection with which on the fourth day of the disease went to the doctor. At survey: the condition is quite severe, the patient is somewhat agitated, inadequate. Scleritis, conjunctivitis, bright hyperemia of the face. On the mucous posterior wall of the pharynx, tonsils, soft palate on a hyperemic background, erosions are visible, at the bottom of which there are yellowish dense overlays. The cervical lymph nodes are enlarged, not soldered to the skin. There are separate hemorrhages on the skin of the torso and limbs. In the lungs, the breath is hard, there are no wheezes. Heart tones are muted, PS 96 beats per minute, BP 90/50 mmHg. Urine with a reddish tinge, urinates rarely.

Before the disease, I worked for two weeks in Sierra Leone, sleeping in villages where there were many rats.

What kind of disease can we talk about?

Task № 2.

Patient X. 32 years old fell ill while returning from a business trip to Uganda (Central Africa), where he stayed for two weeks; in the course of work, he went out into the jungle. When boarding the plane, I felt ill: chills, headache, pain in the muscles of my back, lower limbs, and lower back appeared. During the flight, my state of health continued to deteriorate: nausea appeared, I was vomiting several times, I was worried about thirst, photophobia, and my urine turned a reddish hue. Immediately upon arrival, he was taken to the airport's medical center. At survey: the condition is severe, the temperature is 40°C, the patient is restless, groans, tries to stand up, understands the speech addressed to him poorly, does not answer questions, but performs commands. The face and neck are hyperemic. The eyes are "bloodshot" and glisten. At the time of examination - nosebleed. In the lungs, breathing is vesicular, there are no wheezes. 20 breaths per minute. The heart tones are dull, rhythmic. Pulse 126 beats per minute, blood PRESSURE 100/60 mm Hg. the Mucous membrane of the mouth and tongue is clearly hyperemic, edematous. The abdomen is soft and painless. The liver protrudes from under the costal arch by 0.5 cm, the spleen could not be palpated. Questionable rigidity of the nape of the neck and Kernig's symptom.

What are the most likely diseases to think about?

Answers

Task№1.

The doctor should be alerted first of all by information about the arrival of the patient from West Africa. Such an epid anamnesis makes one suspect a contagious viral hemorrhagic fever. Given the headache, myalgia, constant increase in fever and intoxication, nausea, phenomena of ulcerative necrotic pharyngitis, cervical lymphadenitis, hemorrhagic syndrome, bradycardia, hypotension, oliguria, you should think of Lassa fever or yellow fever.

Task № 2.

Based on the acute onset of the disease, rapidly progressing severe intoxication - headache, myalgia, nausea, vomiting, agitation and inadequacy of the patient, bright hyperemia of the face, the mucous membrane of the eyes and mouth, hemorrhagic syndrome, tachycardia, deafness of heart tones, hepatomegaly, one can suspect Ebola hemorrhagic fever (or Marburg), for which Central Africa is an endemic zone. It is necessary to conduct differential diagnostics with tropical malaria, leptospirosis, hemorrhagic fever with a renal symptom.

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