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METHODOLOGICAL GUIDE
INFECTIOUS MONONUCLEOSIS

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INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis (IM) is an acute benign lymphoproliferative disease, the etiological agent of which is the Epstein – Barr virus (EBV), which belongs to the family of human herpes viruses.

Clinically, the disease was described more than a hundred years ago by N. F. Filatov as an "idiopathic inflammation of the cervical glands", but its existence as an independent nosological form has so far been disputed, and the characteristic hematological changes were regarded as a leukemoid reaction.

In early childhood, MI occurs asymptotically or under the guise of respiratory infections, so 90% of adults can detect antibodies to EBV in their blood serum. In adolescents and young adults who do not have immunity, primary EBV infection is clinically manifested as MI. In the practice of a district doctor, MI occurs quite often, occurs under the mask of acute respiratory viral infection or lacunar angina, and for 6 patients with angina, there is 1 patient with MI (according to statistics).

Proper diagnosis of THEM is also necessary due to the existence of the so – called mononucleosis-like syndrome (MS) - a complex of clinical and hematological signs that are characteristic of THEM and are present in other infections (HIV, cytomegalovirus, adenovirus infections, yersiniosis, toxoplasmosis, etc.), as well as in lymphogranulomatosis, leukemia, rheumatoid arthritis, SLE, allergic reactions, intoxication). In this regard, the diagnosis of MI requires a clear assessment of the entire complex of clinical and hematological symptoms from the doctor.

Purpose of the lesson: develop students ' skills in clinical diagnostics of IM AT different stages of the disease, familiarize them with the principles of laboratory diagnostics and treatment of patients.

Medical practical skills mastered by students on the topic.

The student should be able to:

- correctly collect an anamnesis of the disease, make an examination of the patient, identify the characteristic clinical symptoms and syndromes of the disease;
on the basis of anamnestic data and clinical presentation to formulate a diagnosis for THEM;
- carry out a differential diagnosis with diseases accompanied by mononucleosis-like syndrome;
- correctly assess the severity of the patient's condition;
- evaluate hemogram parameters, results of specific and non-specific serological reactions;
- make an outpatient treatment plan for the patient;
- make a plan of rehabilitation measures in the period of convalescence.

Etiology

The causative agent is a DNA-genomic virus of the genus Lymphocryptovirus of the subfamily Gammaherpesvirinae of the family Herpesviridae. The virus is able to replicate, including in B-lymphocytes; unlike other herpes viruses, it does not cause cell death, but rather activates their proliferation. Virions include specific Ah: capsid (VCA), nuclear (EBNA), early (EA) and membrane (MA) Ah. Each of them is formed in a certain sequence and induces the synthesis of the corresponding at. In the blood of patients with infectious mononucleosis, at to capsid Ah first appear, later at to EA and MA are produced. The pathogen is poorly resistant in the external environment and quickly dies when drying, under the influence of high temperature and disinfectants.

Infectious mononucleosis is only one form of infection with Epstein-Barr virus, which also causes Burkitt's lymphoma and nasopharyngeal carcinoma. Its role in the pathogenesis of a number of other pathological conditions is insufficiently studied.

Epidemiology

The source of infection is a sick person, even with erased forms of the disease, all virus carriers. The disease is low-contagious. Low contamination is associated with a high percentage of immune individuals (over 50%), the presence of erased and atypical forms of mononucleosis, which are usually not detected. The infection is widespread in the form of asymptomatic and erased forms, since antibodies to the virus are found in the majority of the adult population. A person's susceptibility to EBV is high, but mild and obliterated forms of the disease predominate. By the age of 40, almost all people are infected with EBV, but clinically expressed forms of IT develop rarely. Children up to 6 months old are immune to them due to the presence of passive immunity, up to 1 year old get sick very rarely. In children under 3 years of age, primary infection is more often under the guise of acute respiratory infections or asymptomatic, so children from 3 to 14 years of age, adolescents and adults under 30 years of age are mostly ill. Therefore, sometimes infectious mononucleosis is also called a disease of "students". The incidence of MI is sporadic. Transmission of infection occurs through airborne droplets, as well as through direct contact with saliva, kissing and indirect contact through salted household items, toys. Infection is caused by crowding of the population, the use of common dishes, etc. It is also possible to infect the child during childbirth, sexually, with hemotransfusions. The virus is released into the external environment within 18 months after the initial infection, which is proved by studies of material taken from the oropharynx. Spring and autumn seasonality. Immunity after an infection is strong, but not sterile. Repeated cases of IM are not observed, but possible chronization and reactivation of the infection with a weakening of the immune system. In recent decades, there has been a growing trend in the incidence of MI. Immunodeficiency States contribute to the generalization of infection.

Pathogenesis

Primary replication of the virus occurs in the epithelium of the oropharyngeal and nasopharyngeal mucosa, salivary gland ducts, and in lymphoid formations. Then there is hematogenic and lymphogenic dissemination of the virus. Penetration of the virus into the upper respiratory tract leads to damage to the epithelium and lymphoid tissue of the mouth and nasopharynx. Note edema of the mucous membrane, an increase in tonsils and regional lymph nodes. In subsequent Virology, the pathogen is introduced into B-lymphocytes; being in their cytoplasm, it spreads throughout the body. B-lymphocytes are the only cells that have surface receptors for the virus. During the acute phase of the disease, specific viral antigens are detected in the nuclei of more than 20% of circulating B-lymphocytes. After the infectious process subsides, viruses can be detected only in single B-lymphocytes and epithelial cells of the nasopharynx. Some of the affected cells die, and the virus that is released infects new cells. Both cellular and humoral immunity is disrupted. This can contribute to superinfection and layering of secondary infection. Epstein Barr virus has the ability to selectively affect lymphoid and reticular tissue, which leads to systemic hyperplasia of lymphoid and reticular tissues, in connection with which atypical mononuclears appear in the peripheral blood. Develop lymphadenopathy, edema of the nasal and oropharyngeal mucosa, enlarged liver and spleen.

Virus replication in B-lymphocytes stimulates their active proliferation and differentiation into plasmocytes. The latter secrete immunoglobulins of low specificity. Simultaneously, in the acute period of the disease, the number and activity of T-lymphocytes increase. T-suppressors inhibit the proliferation and differentiation of B-lymphocytes. Cytotoxic T-lymphocytes destroy virus-infected cells by recognizing membrane virus-induced Hypertension. However, the virus remains in the body and persists in it throughout the subsequent life, causing a chronic course of the disease with reactivation of the infection with a decrease in immunity.

The severity of immunological reactions in infectious mononucleosis allows us to consider it a disease of the immune system, so it belongs to the group of diseases of the AIDS-associated complex.

Clinical picture

The incubation period varies from 5 days to 1.5 months. Possible prodromal period, which does not have specific symptoms. In these cases, the disease develops gradually: for several days, there is a subfebril body temperature, malaise, weakness, increased fatigue, catarrhal phenomena in the upper respiratory tract – nasal congestion, hyperemia of the oropharyngeal mucosa, enlargement and hyperemia of the tonsils. In children, the onset is usually acute, but can be subacute and gradual, which is more often observed in adults. In typical cases, the picture of the disease unfolds completely by the end of the 1st week. It is customary to distinguish a typical and atypical course of MI.

Distinguish light and medium-heavy course of the disease, observed in most patients; severe forms are rare. There are acute and lingering forms of the disease. In recent years, chronic forms of the disease have also been described.

At the acute onset of the disease, the first symptom is a rise in body temperature to 38-39° C, which is accompanied by moderate General malaise, mild catarrhal phenomena, the addition of sore throat, an increase in lymph nodes. By the end of the 1st week, all the characteristic symptoms are detected. With the gradual onset of the disease, the lymph nodes increase and become sensitive, subfebrility, malaise occurs, after a few days, sore throats join and the body temperature rises. At the same time, often an increase in temperature is associated with the appearance of plaque on the tonsils.

Depending on the severity of the disease, fever lasts from 3 to 4 days to 2 to 3 weeks or more, often after a decrease in body temperature, a long subfebrility persists. The level and duration of fever generally correspond to the severity of the disease. In mild cases, subfebrility is noted, in medium-heavy cases-the body temperature reaches 39° C, the fever lasts 3 to 4 weeks. An increase in body temperature at the acute onset of the disease can be accompanied by chills. Intoxication is expressed weakly or moderately, but in severe cases, sharp muscle weakness, anorexia are possible. Symptoms of intoxication are usually short-lived. There is no typical temperature curve for infectious mononucleosis. Body temperature decreases more often lytically, which coincides with an improvement in the General condition and with a decrease in the severity of other symptoms of the disease. After the main wave of fever, subfebrile body temperature is often maintained. The most characteristic increase is in the lateral cervical lymph nodes. They are often visible to the naked eye. Anterolateral, submandibular, axillary and other groups of lymph nodes also increase, but somewhat later and slightly. In adults, the femoral-inguinal lymph nodes are sometimes affected, which may be related to sexual infection. As a rule, the symmetry of the lesion is observed. The size of the lymph nodes varies from 1-2 to 3-5 cm in diameter. The lymph nodes are moderately painful, of dense elastic consistency, not soldered to each other and to the surrounding tissues. They never catch up. Sometimes there is soft tissue edema around the neck lymph nodes. After 2-3 weeks, the size of the lymph nodes decreases, they become dense, but they can remain enlarged for up to 2-3 months or more. There may be an increase in mediastinal as well as mesenteric lymph nodes. At the same time, abdominal pain, flatulence, and runny stools may appear. With an increase in mediastinal lymph nodes, patients may be disturbed by coughing, pain in the heart area of varying intensity and duration.

An increase in the size of the liver and spleen is also a characteristic symptom of the disease. The liver increases from the first days of the disease, maximum on the 2nd week. Normalized size of the liver after 3 to 5 weeks. In addition to liver enlargement, there may also be clinical manifesta-

tions of hepatitis: nausea, decreased appetite, a feeling of heaviness in the right hypochondrium, weakness, darkening of urine and the appearance of jaundice of the sclera and skin. As a rule, jaundice is short-lived, in the midst of the disease. It is accompanied by an increase in the amount of bound bilirubin and a moderate increase in the activity of transferases, thymol samples, and hyperfermentemia is possible even in the absence of jaundice. Functional tests of the liver are normalized by the 15th-20th day of the disease, but may remain changed for 3 to 6 months. At the height of the disease, 25% of patients develop a rash. Its appearance is associated with taking ampicillin. The rash can be small-point, spot-papular, sometimes (with a severe course of the disease) petechial. In these cases, other manifestations of hemorrhagic diathesis are also possible. Changes in heart activity are usually poorly expressed (tachycardia, moderate hypotension).

The spleen also increases in the first days of the disease, often very significantly, but its size decreases earlier than the liver - in the 3rd – 4th week of the disease. The spleen is dense to the touch, sensitive on palpation.

The picture of catarrhal tonsillitis is a typical manifestation of the disease. Tonsils are moderately hyperemic, edematous. Sometimes their edema is expressed so sharply that they close in the middle line, making it difficult to breathe. At the height of the disease, lacunar and follicular angina are possible, rarely necrotic and fibrinous. The appearance of plaque is associated with the addition of a bacterial infection. The attacks last for 3-7 days. The entire lymphopharyngeal ring, in particular the nasopharyngeal tonsil, is involved in the process. Which is accompanied by a nasal sound, nasal congestion, puffiness of the eyelids and face. Granulose pharyngitis is also a typical symptom. If it is possible to examine the posterior wall of the pharynx, then its edema and hyperemia with the phenomena of hyperplasia of lymphoid tissue are detected; in some patients, the posterior wall of the pharynx may be covered with thick mucus. In 3-4 days after the onset of the disease, loose, curd-like deposits of various sizes appear on the tonsils, which are easily removed with a spatula. In some cases, plaque can be localized on the back wall of the pharynx, at the root of the tongue and even on the epiglottis. Changes in the throat are accompanied by fever. The duration of the pharyngeal lesion is 10-15 days; with timely and adequate treatment, angina passes faster.

In patients with removed tonsils, the reaction of the pharyngeal lymphoid tissue manifests as an increase in the lateral rolls and granules of the posterior pharyngeal wall.

Peculiar changes are noted on the part of the blood. In the first days of the disease, moderate leukopenia, neutropenia, lymphocytosis are observed, and plasma cells appear. On the 4th-5th day of the disease and later, leukocytosis develops to $10-12 \times 10^9/l$, the number of lymphocytes, monocytes and plasma cells increases, and peculiar atypical mononuclears appear, characterized by a large polymorphism in shape and structure.

In most cases, atypical mononuclears are found in blood in the first days of the disease, but especially their number increases during the height of the disease. Less often, the appearance of mononuclears can be noted on the 8th-11th days of the disease. These cells persist for several weeks, but gradually their number decreases.

Among the "white blood" cells, the proportion of mononuclears ranges from 10 to 50% or higher. In some cases, at the height of the disease, all mononuclears can be atypical, and their number correlates with the severity of the disease. Plasma cells are often found in the blood. ESR can increase up to 20-30 mm/h.

Infectious mononucleosis in most patients ends in recovery after 2-4 weeks. However, in some patients, lymphadenopathy, hepatosplenomegaly, and atypical mononuclear cells in the blood persist for a long time, which indicates a prolonged, and possibly chronic course of infection. The latter is characterized by persistent lymphadenopathy and EBV-hepatitis, splenomegaly, interstitial pneumonia, hypoplasia of the bone marrow, and sometimes uveitis.

Chronic mononucleosis. Long-term persistence of the infectious mononucleosis pathogen in the body is not always asymptomatic, some patients have clinical manifestations, the connection of which with the reactivation of EBV is established, but they are non-specific, often they are non-specific, often detected against the background of other pathology.

Almost all patients experience General weakness, rapid fatigue, poor sleep, headache, muscle pain, in some cases a moderate increase in body temperature, an increase in lymph nodes, i.e. symptoms characteristic of CFS, as well as pneumonia, uveitis, pharyngitis, nausea, abdominal pain, diarrhea, sometimes vomiting, in some cases an increase in the liver and spleen. Sometimes exanthema occurs, and oral and genital herpes are more often observed. In blood tests, leukopenia and thrombocytopenia are detected.

Diagnostics.

The diagnosis is made based on the characteristic clinical picture of the blood.

In this case, atypical mononuclears should be at least 10% and detected in two blood tests taken at intervals of 5-7 days.

The diagnosis of MI can be confirmed by detecting specific antibodies by ELISA and NRIF. Ig M - antibodies to capsid antigen (VCA) are detected with the greatest constancy from the first days of the disease VCA, later antibodies to the early antigen complex (EA) are determined, and in the same sequence, Ig – antibodies to these antigens, as well as to nuclear antigen (NA), appear.

Immunodiagnostics allows to differentiate between reactivation of EBV infection, latent infection from THCM. The latter is characterized by the appearance of Ig M-antibodies, for latent infection-Ig G-antibodies. Reactivation reveals both classes of antibodies. Immunodiagnostic meth-

ods can be used to diagnose EBV-lymphoma. PCR can detect the presence of EBV DNA, but PCR does not differentiate between primary infection and latent reactivation.

In practice, until now, the definition of heterologous antibodies is widely used for the diagnosis of IM. These methods are simple and quite sensitive, although they are not completely specific. Of these, the most effective reaction is agglutination of the patient's blood serum with formalized horse erythrocytes (Goff-Bauer reaction). The reaction is performed on the glass, and the results are taken into account after 2-3 minutes. The Paul-Bunnell reaction used in the past (agglutination of sheep red blood cells) is not specific and is not recommended for practical use.

Differential diagnosis

The differential diagnosis of MI is carried out with infectious diseases occurring with fever, polyadenopathy and hepatolienal syndrome (adenovirus infection, benign lymphadenopathy, HIV infection, rubella in adults, typhoid – paratyphoid diseases), with angina, localized and toxic diphtheria, CMV, leukemias, lymphomas (lymphogranulomatosis), and in the presence of jaundice with viral hepatitis, with yersiniosis. In the present era, the most relevant differential diagnosis of MI with mononucleosis – like syndrome in HIV infection is the differential diagnosis of MI with mononucleosis-like syndrome. If the latter is available asymmetrically an increase in various lymph nodes (2 – 4), atypical mononuclear cells appear on the background radiation. In all cases, THEY must be tested for HIV infection.

Adenovirus infection is characterized by a lower degree of polyadenopathy, the frequent presence of conjunctivitis, and the absence of atypical mononuclears. In benign lymphoedema, the febrile reaction is weakly expressed, there is a primary affect, lymphadenitis with a tendency to suppuration, secondary lymphadenitis, usually asymmetric; there are no tonsillitis and pharyngitis, atypical mononuclears in the blood.

In rubella, mainly the posterior neck lymph nodes increase. Fever, enlargement of the spleen and liver are short-lived, single mononuclears are rarely detected.

For typho-paratyphoid diseases uncharacteristic polideepcia, tonsillitis, pharyngitis.

Streptococcal sore throat is characterized by a rapid onset, intense pain in the throat, the appearance of plaque on the 1st – 2nd day of the disease, an increase and soreness of the submandibular lymph nodes, island-inflammatory changes in the blood. The duration of the febrile period does not exceed 3-4 days, there is no increase in the spleen, nasal breathing is not changed.

Differential diagnostic analysis of suspected diphtheria should be carried out depending on what form (localized or toxic) is suspected by changes in the pharynx. The difference in the clinical picture of these forms is the main point of differential diagnosis of MI with pharyngeal diphtheria. Localized insular or membranous diphtheria of the pharynx can be excluded if the patient has a rough change in the contours of the neck due to an increase in the lymph nodes with small plaque

corresponding to this form. With localized diphtheria, the reaction of the lymph nodes is relatively small and the swelling on the neck is unusual. A sharp difficulty in nasal breathing and nasal nasopharyngitis are not observed. These symptoms speak in favor of IM. The latter is also characterized by a longer and less high fever than in diphtheria. When differentiating IT from the toxic form of diphtheria, it is important to take into account the nature of the temperature reaction. The resemblance to it in such cases is determined by the extensive deposits that completely cover the tonsils. The duration of temperature in MI with large tonsillitis is most often 8-10 or more days, with diphtheria, such a long fever is extremely rare due to the addition of other diseases. The onset of the disease and the formation of a temperature reaction in MI is most often gradual, within 2-4 days, while the toxic form of diphtheria is characterized by an acute onset, a high temperature in the first 2-3 days of the disease and its rapid reduction to normal or subfebrile numbers. Suspicion of a toxic form of diphtheria occurs with MI usually not in the first days of the disease, but in the 2nd week of the disease, when all the symptoms gradually increase to the maximum, and the attacks increase. In the toxic form of diphtheria without serum treatment, paleness, symptoms of polyneuritis and cardiovascular disorders appear in such late stages of the disease, with IM, the patient's condition is relatively light, the color of the skin is pink. Diphtheria plaque is characterized by a high density, smooth surface, grayish-white color, when they are applied loose, curd-like, smeared.

Significantly different in these diseases, the change in the contours of the neck. In diphtheria, the swelling is most pronounced in the submandibular area. In contrast to toxic diphtheria with MI, even with sharp edema of the tonsils, the plaque is absent or has a localized character. The swelling is localized around the enlarged lateral cervical lymph nodes.

In diphtheria, difficulty in nasal breathing is caused by a sharp edema of the pharyngeal soft tissues or films on the nasal septum. The nasal discharge in diphtheria is scanty, sucrovichnoe, with infectious mononucleosis, the nasal mucosa appears swollen, has a plentiful discharge.

In cmvi, the appearance of atypical mononuclears in the blood is possible for a short time and in small quantities; in doubtful cases, serological studies are used.

Differential diagnosis with leukemia is based on the results of hematological research, in rare cases, bone marrow punctate is examined. With lymphogranulomatosis, there is an asymmetric non-simultaneous increase in various groups of lymph nodes. With lymphomas, the lymph nodes are dense, painless, and sedentary. In difficult cases, histological examination of the lymph node biopsy is performed.

In viral hepatitis, jaundice appears after the pre-jaundice period. Uncharacteristic polideeprcia, tonsillitis, pharyngitis, the presence of atypical mononuclear cells in the blood.

Generalized yersiniosis is characterized by the presence of a peculiar rash (a symptom of gloves, socks), polyarthrititis, dyspeptic and abdominal syndromes, and kidney damage. There are no atypical mononuclears in the blood.

Complications of MI occurring against the background of immunodeficiency can be by their nature: hematological (autoimmune hematological anemia, thrombocytopenia, granulocytopenia, spleen rupture), cardiological (pericarditis, myocarditis) and neurological (meningitis, meningoencephalitis, myelitis, neuropathies).

Treatment

Patients are hospitalized for clinical indications. A special diet (table #5) is indicated for 6 months after the patient has suffered ONLY in the presence of hepatitis with jaundice. In the presence of fever, bed rest is prescribed. Detoxification therapy is performed. It is recommended to take antihistamines. Corticosteroids are effective, however, given their immunosuppressive effect and the lymphoproliferative properties of EBV, they are indicated (in a short course of 3-5 days) only for severe intoxication, the threat of asphyxia, CNS lesions, thrombocytopenia and hemolysis. In high fever with severe intoxication, antipyretic drugs (Panadol, paracetamol, etc.) are prescribed. Antibiotics even in the presence of angina with plaque are ineffective. In these cases, macrolides (erythromycin, Sumamed), tetracycline preparations, and fluoroquinolones are mainly used. Ampicillin is contraindicated, since its administration often causes the appearance of a toxic-allergic rash in patients with mononucleosis.

Patients who are on outpatient treatment are recommended a semi-bed regime, a sparing diet. Care for the oral cavity (rinse solution furatsilina. sodium bicarbonate, etc.), multivitamins with trace elements, phytomedicines with antioxidant and immunostimulating effects (Echinacea, licorice root).

After the disease has passed, physical activity should be restricted for 3 months. Observation by an infectious disease specialist or a hematologist for 3 months is indicated. All patients diagnosed with MI and suspected of it should be tested for HIV in the acute period of the disease, after 1, 3 and 6 months during the convalescence period.

The forecast is favorable. Fatal outcomes are observed rarely, recovery in the vast majority of cases is complete. However, the lifelong persistence of EBV in the body is fraught with a threat of its reactivation in the development of immunodeficiency. In rare cases, the infection may become chronic.

Specific prevention measures have not been developed. Anti-epidemic measures are not carried out in the hearth.

Differential diagnosis of MI in the peak period with diseases accompanied by the development of mononucleosis-like syndrome

Clinical signs	of IM	HIV infection	Yersiniosis	Chlamydia
Catarrhal and respiratory syndrome	nasal Congestion without discharge, sore throat, often with overlays	Hyperemia of the posterior pharyngeal wall, candidal stomatitis	Moderate pharyngitis, angina	Not typical
Rash	Occasionally spotted, spotty-papular, on the trunk, limbs, without itching	Occasionally, spotty-papular on the trunk, limbs, without itching	, small-Point, spotty-papular, petechial, appears in a short time, expressed exudative component, rash merges on the feet, hands, around the joints. Skin itch.	There may be ephemeral rashes of various types, without a specific localization. Skin itch.
Lymph nodes	The front and rear necklines are enlarged. generalized lymphadenopathy. Painful, mobile	Enlargement of various groups, tight-elastic, up to 1 cm, painless, persist for a long time	Microlithography, sometimes enlarged cervical lymph nodes	Generalized lymphadenopathy persists for a long time. The lymph nodes are enlarged, dense, painless, and soldered to the skin
An increase in the liver and spleen	is Characteristic. Possible jaundice of the mucous membranes and skin	Not typical	Increased moderately, jaundice often	The spleen is enlarged, the edge is tight, painless
Some features that are important in diagnostics	Young age. The symptoms are clearly cyclical. Fever for more than 2 weeks. Benign nature of the disease. The outcome is favorable.	Features of behavior, appearance, lifestyle that indicate belonging to a certain risk group	of Arthralgia, myalgia, dyspepsia, polymorphism of clinical symptoms, multi-organ damage from the first days of the disease	There is no stage in the development of symptoms, they appear gradually against the background of fever

Differential diagnosis of MI with streptococcal tonsillitis, diphtheria, and adenovirus infection

Clinical sign	of MI	Streptococcal tonsillitis (sore throat)	Diphtheria (toxic form)	Adenovirus infection
The beginning	Both acute and gradual	Acute	Acute	Often there is an acute, more often gradual
Fever	It can be different. The rise in temperature is associated with the development of angina	Up to 7 days. The height of fever is associated with the severity of changes in the oropharynx	High, lasts up to 7 days. Extinction is normalized to the changes in the oropharynx	Sometimes high. Often the disease for 5-7 days proceeds without fever
Sore throat	Moderate	Strong	Strong in toxic forms	Moderate
The overall condition	Changes little	Chills, joint and muscle pain, body aches, headache	Lethargy, adynamia, pallor, lack of appetite	Changes slightly
, the oropharyngeal Mucosa Changes slightly	Bright hyperemia, granulose pharyngitis, there may be hemorrhages. There is no edema	Bright spilled hyperemia. There is no edema	Dim congestion, cyanotic mucous membranes. Severe edema	Mild spilled hyperemia, granularity can persist for up to 3 weeks. No edema of
the Mucous nasopharynx	nasal Congestion. There are no selections	Does not change	In diphtheria of the nose film, sukrovichnye detachable	nasal Congestion. Profuse mucosal or serous discharge
Palatine tonsils	Increased, sometimes to the third degree, due to hypertrophy, hyperemic	Increased to III degree due to hypertrophy, vividly hyperemic	Increased significantly due to pronounced edema, cyanotic	Enlarged, in lacunae effusion
character of overlays	In the course of gaps, yellow, yellow-green color. They may be partially fibrinous in nature and spread beyond the tonsils	Purulent, yellow-green along the course of lacunae. Do not go beyond the tonsils. putrid breath	The film on the surface of the tonsils is gray, dull, dense, when removed, the surface bleeds. It is removed with difficulty	In the lacunae of effusion
, the Lymph nodes	are Isolated (cervical) or generalized lymphadenopathy	Increase, soreness of submandibular lymph nodes (can be only on one side)	Increase, soreness of submandibular lymph nodes	Increase in the cervical group of occipital, submandibular
the configuration of the neck	is Changed due to sharply enlarged cervical lymph nodes and edema of subcutaneous fat around them	Does not change	Edema of the subcutaneous tissue of the neck is both unilateral and bilateral	Does not change
Changes in oth-	Significant in-	It doesn't happen	Complications	Catarrhal, follicular,

er organs do not change (in the acute period)	crease in spleen and (less) liver		in the early and late periods: myocarditis, nephrozo-nephritis, poly-neuropathy	film conjunctivitis, pharyngitis, tracheitis, tracheobronchitis, hepatolienal syndrome
hemogram Change	Moderate leukocytosis, significant lymphocytosis (60% or more), atypical mononuclear cells (more than 12%). ESR - n	Leucocytosis, neutrophilosis, P _I shift. Erythrocyte sedimentation rate increased	Leukocytosis, neutrophilia. P _I shift. Increase of ESR	Moderate leukocytosis, lymphomonocytosis. Atypical mononuclears in the amount of up to 12%. ESR has not been changed

Questions for self-monitoring

1. The concept of "mononucleosis-like syndrome" and diseases accompanied by the development of the syndrome.
2. Brief information about the Epstein-Barr virus as the causative agent of infectious mononucleosis.
3. Epidemiology of EBV infection.
4. Pathogenesis of infectious mononucleosis.
5. Clinical characteristics of MI and its main periods: initial, peak, reconvalescence.
6. The picture of peripheral blood in patients with MI in different periods of the disease.
7. Specific and non-specific serological methods of MI diagnosis and evaluation of their results depending on the duration of the disease.
8. Possible complications of MI and the causes of their development.
9. Differential diagnosis of MI in the initial period of the disease.
10. Differential diagnosis of MI in the period of the height of the disease.
11. Features of the course of the convalescence period in MI.
12. Principles of therapy for THEM.
13. Contents of the work on rehabilitation of convalescents who have suffered MI.

Task #1

Patient N. 19 years old went to the polyclinic on the 10th day of the disease with complaints of weakness, yellowing, difficulty breathing through the nose, severe pain in the throat when swallowing, pain in the neck when moving.

According to the patient, the disease began gradually, with an increase in temperature to 37.6° C, weakness. On the 3rd day of the disease, pain appeared in the neck when turning the head, the cervical lymph nodes increased to the size of a pea, were painful when touched. My

state of health did not significantly deteriorate. The temperature remained subfebrile. On the 4th day of the disease, the patient noticed a swollen eyelid, puffiness of the upper half of the face, there was a pronounced nasal congestion, "nasality" of the voice. She was treated independently with symptomatic means. The cervical lymph nodes continued to swell and become painful. On the 7th day of the illness, the condition worsened: the temperature rose to 38.7° C, then to 39.5° C, there was a chill, there was a strong pain in the throat when swallowing, nasal congestion increased. I took erythromycin, antipyretics. Because of the stuffy nose and pronounced soreness of the cervical lymph nodes, I did not sleep well. A significant deterioration of the condition was noted by the end of the 9th day of the disease: chills increased, headache, temperature rose to 39.6° C, due to sore throat, I could not open my mouth, there was difficulty breathing, a sense of lack of air.

On examination: the patient is active, contactable. The face is pasty, the eyelids are puffy. The Voice Is "Nasal". Nasal breathing is absent, there is no discharge from the nose. The skin is physiologically colored, there is no rash. Clearly visible enlarged to the size of beans, sharply painful on palpation anterior, middle, posterior neck lymph nodes. The mucous membrane of the oropharynx is vividly hyperemic, the follicles of the posterior wall of the pharynx are enlarged, juicy, and thick mucus flows down the posterior wall of the pharynx. Tonsils are enlarged to the third degree, in lacunae-abundant overlays of yellowish-green color. On the mucosa of the soft palate, there are single petechiae. The spleen is enlarged, protruding from the hypochondrium by 1.5 cm, its edge is dense, smooth, elastic, slightly painful on palpation. The size of the liver is increased, it is palpated 3 cm below the costal arch, its edge is dense, elastic, slightly painful. The urine is light. There are no meningeal phenomena, no focal symptoms were detected.

1. Establish and justify the diagnosis .
2. Suggest a plan of examination and treatment of the patient.

Answer:

1. Based on the patient's complaints, the duration of fever for more than 7 days, the characteristic development of the disease, temperature rise to 39.5° C, the appearance of sore throat in the second week, swelling of the eyelids and face at an early stage of the disease, visible increase and soreness on palpation of the cervical lymph nodes, hepatolental syndrome, you can make a diagnosis of "Infectious mononucleosis".
2. General blood and urine analysis; biochemical blood analysis; Goff-Bauer serological reactions. In terms of treatment-detoxifying, desensitizing, fermentative, symptomatic drugs, rinsing the oropharynx with antiseptic solutions.

Task # 2.

Patient L., 20 years old, went to the doctor of the clinic on the 9th day of the illness with complaints of chills, fever rising above 39 ° C, pronounced weakness, severe pain in the throat when swallowing, difficulty in nasal breathing. She fell ill acutely, with an increase in temperature to 38° C, then there were pains in the neck when moving her head. After 2 days, I noticed an increase in the cervical lymph nodes, their soreness. The face, eyelids slightly swollen, there was a stuffy nose. On the 8th day, the temperature rose to 39.2° C, there was a strong pain in the throat when swallowing, aching in the body, joint pain. Independently from the 1st day of the disease, I took oletetrin, but without any effect. The condition worsened on the 9th day of the illness: fever up to 39.5° C, could not swallow saliva, it became difficult to breathe, neck pain increased. I called a doctor, who assessed the patient's condition as serious and sent her to an infectious disease hospital with a diagnosis of "diphtheria".

When examined in the emergency room: skin is pale, breathing is difficult, breathing with an open mouth, the position is forced, sitting in bed. BH-20 per minute The voice is "nasal". The neck configuration has been changed due to sharply enlarged Antero - and posterolateral lymph nodes (larger on the right) (up to 2.5 cm). There is no swelling of the neck. The lymph nodes are sharply painful, elastic, and mobile. Trismus of the chewing muscles. The oropharyngeal mucosa is vividly hyperemic, and the tonsils are enlarged to II grade II. There is no edema of the mucous membrane. There are purulent overlays in the lacunae of the tonsils. The palatal curtain is movable. Pulse 110 BPM, blood PRESSURE 120/80 mm Hg. u. Abdomen on palpation is soft, slightly painful in the right hypochondrium. The liver and spleen are enlarged, palpable. There are no meningeal symptoms.

1. Make a tentative diagnosis.
2. What made the local doctor consider the patient's condition as serious?
3. What treatment measures does the patient need?

Answer

1. The diagnosis is "Infectious mononucleosis, the peak period, severe course". Justification of the diagnosis: the duration of fever for more than 7 days in a young patient, lymphadenopathy, angina, hepatolyenal syndrome, deterioration of the condition by the end of the first week with the appearance of sore throat and other clinical signs of acute tonsillitis.

2. The district doctor assessed the patient's condition as severe based on the appearance of clinical signs of asphyxia with the course of the disease: complete absence of nasal breathing, difficulty in inhaling, forced position of the patient, "grabbing" air with an open mouth. The appearance of symptoms of asphyxia can also be explained by swelling of the nasopharyngeal tonsil.
3. Treatment is complex: detoxification therapy, antipyretic, desensitizing drugs, antibiotics.