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"NORTH OSSETIAN STATE MEDICAL ACADEMY»
MINISTRY OF HEALTH OF THE RUSSIAN FEDERATION

Department of Infectious diseases

METHODOLOGICAL GUIDE

DIPHTHERIA

for students studying in the specialty
31.05.01 General medicine (specialty)

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I.Annotation. Purpose of the lesson

The purpose of studying this topic is to develop students ' skills in making a diagnosis of diphtheria and solving questions of therapeutic tactics.

P. Introduction

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

1. Etiology and epidemiology of diphtheria.
2. Pathogenesis and pathological anatomy of diphtheria.
3. Clinical classification and characterization of various forms of diphtheria.
4. Methods of laboratory diagnostics.
5. Methods of etiopathogenetic therapy of diphtheria.
6. Prevention of the disease, measures in the focus of infection.

The student should be able to:

1. Correctly collect an anamnesis and make an examination of the patient, to identify the main signs of diphtheria.
2. Properly perform an examination of the oropharynx.
3. Solve the issue of hospitalization and organize it.
4. Determine the plan of examination of a patient with suspected diphtheria.
5. Make a sampling for bacteriological research.
6. make a differential diagnosis with diseases that occur with symptoms of oropharyngeal damage-angina, infectious mononucleosis, mumps, peritonsillar abscess, blood diseases, etc.
7. Assess the severity of the course, anticipate complications.
8. Make a treatment plan, enter anti-diphtheria serum.
9. Provide for the rehabilitation of convalescents.
10. Make a sanitary and educational conversation on the prevention of diphtheria for work in the hearth.
11. Conduct surveillance of contact persons in the focus of diphtheria.

III. Generalities

Currently, diphtheria is spread mainly among the adult population, often in the form of toxic forms. The growth of diphtheria among adults causes an increase in morbidity among unvaccinated children. Early and correct diagnosis of diphtheria is the keyto its effective treatment and prevention. The severity of the disease, the high percentage of complications andmortality in common throat diphtheria determine the relevance of studying the disease by future doctors.

Diphtheria is an acute infectious disease caused by toxigenic Corynebacterium diphtheria, characterized by local fibrinous inflammation of mainly the mucous membranes of the mouth and nasopharynx, as well as the phenomenaof General intoxication and damage to internal organs.

Today, Russia takes the leading place in Europe in the incidence of diphtheria - it accounts for 97% of all cases.

During the period 1970-1999, there were two major "spikes" of the disease.

The first wave of the disease occurred in the 80 years, i.e. the beginning of the rise is 1977, and the peak incidence was in 1984. At that time, 646 people aged 16 to 61 were under observation, among whom the majority were men. In General, the course of diphtheria in the 80s was relatively favorable, complications were observed in 127 people, and deaths occurred in 7 people. All of them had a toxic form of oropharyngeal diphtheria of various degrees of severity, two patients had severe multiple complications (myocarditis, pneumonia, OHSS), three patients abused alcohol and four were not vaccinated in childhood.

The second wave of the disease began in 1991, when the number of patients increased by about 6 times. During the period from 1990 to 1995, 4,650 people were under observation, among whom the majority were women (2,725 people). More than 55% of patients were aged between 30

and 50 years, while during the first wave, the majority were aged under 30 years. Among the clinical forms, localized (76%) prevailed, and toxic was 15%. The number of combined forms increased by 4 times (4%), and 3-4 organs (oropharynx, larynx, tongue, eyes, genitals) were involved in the process. Significantly often observed croup and diphtheria. Laryngeal lesions were accompanied by the development of stenosis (80%), which often led to a fatal outcome. In the 90 years, the number of severe polyneuritis increased significantly (60%) with damage to the cranial nerves (36%), with damage to the cerebellum (1.5%), gait disorders, dizziness, impaired coordination of movement. And also in recent years, the frequency of complications associated with the addition of secondary bacterial infections, leading to the development of pneumonia, pleurisy, and sepsis, has increased. The death rate in 1993-1994 was 54 people - the highest figures for the last 10 years.

In 1996, the number of diphtheria patients in Russia was 13,604, including 4,417 children. Thus, we can note an increasing trend in the incidence of diphtheria in recent years, which is probably due to the deterioration of the economic and social situation in the country, the influx of refugees, and a decrease in the volume of medical assistance to the population. A significant role was played by the low level of vaccination coverage of young children and the increase in the number of non-immunized adults.

HISTORY

Diphtheria epidemics were known even to Hippocrates. The first reliable description of diphtheria belongs to the medical historian Areteus, who lived in the first century ad. This infection has been described under various names: Egyptian or Syrian disease, pharyngeal plague ulcer, malignant tonsillitis, tracheal tonsillitis,choking disease, respiratory tube disease, and so on. From the XVIII century to the present, the term "croup" is used when the larynx is affected by diphtheria.

There is an opinion that the birthplace of diphtheria is Asia, from where it entered Europe and gradually spread throughoutthe globe. Extensive epidemics of diphtheria in the seventeenth and eighteenth centuries are known to have terrified the populations of Europe, especially Italy and Spain. In the XVIII century, diphtheria appeared in England, Germany, Holland, Switzerland, and North America. Since the first half of the XIX century, diphtheria epidemics have been registered in almost all countries of the world with high child mortality. It is assumed that diphtheria was introduced to Russia from Romania, first in the Northern, then in the southern provinces. Since the second half of the XIX century, the incidence of it in Russia has increased sharply.

Despite the long history and ubiquity of the distributionof diphtheria, it wasonly in the twenties XIXof the XIX century that the French scientist Bretonneau and his student Trousseau (1821-1824) developed it into an independent nosological unit. Bretonneau has established a link between individual process localities and a characteristic feature - the formation of a film. Theidentity of diphtheria and croup films, as well as the Association of suffocation in diphtheria with the narrowness of the child's larynx, were noted. He also developed the tracheotomy operation in detail.

Bretonneau suggested calling the disease "diphtheria", which in Greek means "false skin", "false film". Since 1846, the term "diphtheria" (Trousseau) has been used, emphasizing the importance of General phenomena in the picture of the disease.

The causative agent of diphtheria was discovered in 1883 by Clebs on slices of films taken from the throat of patients. In 1884, these data were confirmed by Loffler by isolating a pure culture of diphtheria bacteria and studying some of their properties.

In 1884-1888, Roux and Lersen obtained a diphtheria toxin, which they studied in an experiment on animals, which allowed us to finally solve the question of the etiological role of this microbe in diphtheria. In 1890, the Russian scientist Orlovsky discovered an antitoxin in the blood. As a result of these studies, the possibility of creating anti-diphtheria serum was presented. This medicinal product, which allowed a sharp reduction in mortality in diphtheria, was obtained independently in 1892-1894 by Roux in France, Behring in Germany, and ya.y. Bardakh in Russia.

Specific prevention of diphtheria was first developed in Russia in 1902 by S. K. Dzerzhikovsky, who conducted an experiment on himself.

In 1912, Schick proposed a skin reaction with a toxin to identify individuals susceptible to

diphtheria.

In 1913, Behring used a diphtheria toxin neutralized with antitoxic serum for prevention; in 1923, Cato recommended immunizing with anatoxin.

For the first time, anti-diphtheria serum was used for medical purposes in 1894 in the children's clinicof the Moscow University by N. F. Filatov, R. N., and Rabrichevsky. K. A. Rauhfus (1897) proved its effectiveness with conviction. V. I. Molchanov and his students made a significant contribution to the development of the doctrine of diphtheria. Some questions of pathogenesis, specific and non-specific treatment of the disease and its complications were carefully developed, and a classification of clinical forms of diphtheria was created. Our scientists have established the law of frequency of diphtheria epidemics, their dependence on the time of year, the influence of age and individual characteristics of the body on the incidence and mortality from diphtheria, improved the method of active immunization against diphtheria (PF Zdrodovsky, 1949), which contributed to its wide introduction into the practice of Soviet health. Due to the implementation of anti-epidemic measures and, first of all, the creation of a high level of antitoxic immunity in the population, this infection has been eliminated as an epidemic disease in our country.

ETIOLOGY

The causative agent of diphtheria is the gram-positive stationary bacterium Corynebacterium diphteriae. Bacteria have bulbous thickening at the ends due to the inclusion of wool-lutin grains (Babesch-Ernst corpuscles). When dividing, the cells diverge at an angle to each other, whichcauses their characteristic arrangement in the form of spread fingers, hieroglyphs, Latin letters V, Y, L, parquet, etc. According to Neisser, bacteria are colored brown-yellow with bluethickened ends. There are two main biovars of the pathogen (gravis and mitis), as well as a number of intermediateintermedius ones (intermediate, etc.). the Bacteria are whimsical and grow on serum and blood-media. The most common medium is tellurite (Klauberg II medium), because the pathogen is resistant to high concentrations of potassium and sodium tellurite, which inhibits the growth of contaminating microflora.

The main pathogenicity factor is diphtheria exotoxin, which is considered to be one of the mostpotent bacterial poisons. It is second only to botulinum toxin and tetanus toxin.The ability to toxin formation is shown only by lysogenic strains of the pathogen infectedwith a bacteriophage carrying the Tox gene, which encodes the structure of the toxin. Nontoxigenic strainsof the pathogen are not able to cause disease and are called diphteroids. Adhesiveness, i.e. the ability to attach to the mucous membranes and multiply, determines the virulence of the strain. The pathogen persists for a long time in the external environment. In diphtheria film, in drops of saliva, on door handles, toys, it persists for up to 15 days. In water and milk, the pathogen persists for 6-20 days. It is adversely affected by direct sunlight, high temperature. When boiling, Corynebacterium diphteriae dies within 1 min. , in 10% hydrogen peroxide solution - after 3 min., in 1% sulema solution - after 1 min., 5% phenol solution and 50-60° ethyl alcohol - after 1 min. Resistant to low temperature. When heated to 60°C, it dies after 10 minutes. UV rays, chlorine-containing preparations, Lysol and other des also have an inactivating effect. tools.

EPIDEMIOLOGY

The main source of diphtheria infection is a personwith diphtheria or a carrier of toxigenic diphtheriamicrobes. In the body of a patient with diphtheria, the pathogen is detected already in the incubation period, it is located during the entire acute stage of the disease and in most individuals continues to be isolated some time after it. Thus, in 98% of cases, diphtheria bacilli are isolated in the first week of convalescence, in 75% - after 2 weeks, in 20% - more than 4, in 6% - more than 5 and in 1% - 6 weeks. and more.

Epidemiologically, the most dangerous individuals are those who are in the incubation period of the disease, patients with erased, atypicalforms of diphtheria, especially rare localities (for example, skin diphtheria in the form of eczema, diaper rash, pustules, etc.), which differ in a longer course compared to diphtheria of the usual localization and typical course, and are diagnosed late.

Koopman, and Campbell (1975) note a special contagiousness of patients with a cutaneous form of diphtheria, which occurs as impetigo, due to the tendency of these forms to significant contamination of the environment.

Bacterial carrier develops after the endureddiphtheria and in healthy individuals, while there may be a carrierof toxigenic, atoxigenic and simultaneously both types of corynebacteria.

With diphtheria, a healthy carrier is widespread, it significantly exceeds the incidence, occurseverywhere and even in places (the Philippines, India, Malaya) where this infection has never been recorded.

The epidemiological importance of carriers of toxigenic bacteria of diphtheria. Reconvalescent carriers, as well as patients in the acute period of the disease, emit the causative agent many timesmore intensively than healthy bacterial carriers. But, despite this, during the period of sporadic morbidity, when manifest forms of diphtheria are rare and in these patients, contacts with healthy persons are very limited due to low mobility due to poor health, a special epiploological significance is acquired, except for patients with erased, atypical forms of diphtheria, healthy bacterial carriers of toxigenic corynebacteria. Currently, the latter are the most widespread and mobile sources of diphtheria spread.

Healthy carrier is considered as an infectious process without clinical manifestations. This is confirmed by the indicators of antitoxic and antibacterial (specific and non-specific) immunity, electrocardiogram data produced in the dynamics of carrier. Pathohistologically, changes of the multilayered flat epithelium, submucosal layer, and lymphoid apparatus of the tonsils, inherent in acute inflammation, were found in the tissues of the tonsils of rabbits-carriersof corynebacteria.

The frequency of the carrier toxigenic Corynebacterium reflects the epidemiological situation for diphtheria. It is minimal or reduced to zero in the absence of morbidity and significant in the absence of diphtheria - 4-40. According to the data, in the centers of diphtheria carrier is 6-20 times higher than among healthy individuals.

Unlike the carrier of toxigenic cultures, the carrier of nontoxigenic corynebacteria strains does not depend on the incidence of diphtheria, it remains more or less constantor even increases.

The level of carrier status in collectives also dependson the condition of the nasopharynx. In foci of diphtheria, carrier among children with a normal condition of the pharynx and nasopharynx mucosa is detected 2 times less often than among children suffering from chronic tonsillitis. The role of chronic tonsillitis in the pathogenesis of long-term diphtheria bacteriocytosis is also evidenced by the research of A. N. Sizemov And T. I. Myasnikova (1974). In addition to the formation of a long carriage of a large knowledgeincrease give concomitant staphylo-, streptococcal microflora, especially in children with chronic pathological changes of the nasopharynx. V. A. Botchkova et al. (1978) consideredmelting away that the presence of chronic foci of infection in the nasopharynx and related infectious diseases to reduce the immunological reactivity of the organism and are the cause of the stress weakly antibacterial immunity, leading to the formation of bacteria.

The degree of danger of carriers of toxigenic corynebacteriais determined by the level of antitoxic immunity in the collective, which affects the process of carrier indirectly, reducing the incidence of diphtheria and thereby dramatically reducing the possibility of contact with the pathogen. With a high level of antitoxic immunity and the presence of a significant number of
carriers of toxigenic bacteria may not develop diphtheria. The carrier becomes dangerous if non-immune individuals appear in the collective.

Many authors (V. A. Yavrumov, 1956; T. G. Filosofova, D. K. Zavoiskaya, 1966, etc.) note (after widespread immunization of the child population against diphtheria) a decrease in the number of carriers among children simultaneously with an increase in their number among adults. The reason for this is a significant percentage (23) of adults who are not immune to diphtheria, which corresponds to the number of all children being immunized. This is the reasonfor the increased role of adults in the epidemic process of diphtheria.

Healthy carrier is usually 2-3 weeks, relatively rarely lasts more than a month, and sometimes up to 6-18 months. According to M. D. Krylova (1969), reinfection of the carrier by a new pathogen phagovariant may be one of the reasons for long-term carrier transmission. Using the phagotyping

method, it is possible to determine the duration of bacterial propagation more accurately. This method is also promising in identifying the source of diphtheria outbreak in the focus.

Both toxigenic and non-toxigenic corynebacteria can simultaneously circulate in different groups. According To G. p. Salnikova (1970), more than half of patients and carriers simultaneously vegetate toxigenic and non-toxigenic corynebacteria.

In 1974, the classification of bacterial carriers was adopted, taking into account the type of pathogen, the state of the nasopharynx and the duration of carrier (order No. 580 of the Ministry of health of the USSR of June 26, 1974):

1.Bacterial carriers of toxigenic diphtheria microbes:
a)with an acute inflammatory process in the nasopharynx, when the diagnosis of diphtheria is excluded on the basis of a comprehensive examination (including quantitative determination of antitoxin in the blood);
b)with chronic inflammatory process in the nasopharynx;
c)with a healthy nasopharynx.

2.Bacterial carriers of atoxigenic diphtheria microbes:
a)with an acute inflammatory process in the nasopharynx;
b)with chronic inflammatory process in the nasopharynx;
c)with a healthy nasopharynx.

3.For the duration of selection of a microbe:
a)transient bacterial carrier (single detection of diphtheria bacilli);
b)short-term carrier (microbes are released within 2 weeks);
c)medium-term carrier (microbes are released within 1 month);
d)prolonged and recurrent carrier (microbes are isolated for more than 1 month).

In addition to humans, the source of diphtheria infection in the wildcan also be domestic animals (cows, horses, sheep, etc.), in which corynebacteria are found on the mucous membranes of the mouth, nose, and vagina. A great epidemiological danger is the presence of pustules on the udder of cows and chronic, untreatable ulcers, the contents of which are determined by diphtheria Bacillus. Carrier and incidence of diphtheria in animals depends on the incidence of diphtheria in humans. During the period of sporadic incidence of diphtheria in humans, the incidence of diphtheria in animals also decreases.

MECHANISM OF TRANSMISSION OF INFECTION:

Transmission of infection occurs mainly by airborne droplets. The infection is dispersed by the patient or carrier when talking, coughing and sneezing. Depending on the specific gravity of the droplet being separated, it can remain in the air for several hours (aerosol mechanism). Infection can occur immediately upon contact or through contaminated air after some time. The possibility of indirect diphtheria infection is not excluded through infected objects: toys, clothing, underwear, dishes, etc. There are known "dairy" outbreaks of diphtheria associated with infection through infected dairy products.

SUSCEPTIBILITY AND IMMUNITY:

Susceptibility to diphtheria is low, with a contagious index ranging from 10-20%. Thus, infants up to 6 months of age are immune to this disease due to the presence of passive immunity transmitted from the mother by placental route. Children aged from 1 year to 5-6 years are most susceptible to diphtheria. By 18-20 years and older, immunity reaches 85%, which is due to the acquisition of active immunity.

But recently, the age composition of diphtheria patients has changed dramatically. The major-

ity of patients are teenagers and adults, and the incidence among preschool children has sharply decreased.

The incidence of diphtheria is influenced by a number of factors, including the state of natural and artificial, i.e. vaccination, immunity. The infection is defeated if 90% of children under 2 years of age and 70% of adults are vaccinated. Socio-ecological factors also occupy a certain place.

FREQUENCY AND SEASONALITY:

B Within a given territory, the incidence of diphtheria periodically increases, which depends on the age composition, immunity and accumulation of diphtheria-susceptible groups of the population, especially children.

The incidence of diphtheria is also characterized by seasonality. During the entire analyzed period, there was a characteristic autumn-winter seasonality for this infection. This period accounts for 60-70 % of the annual incidence.

With poor organization of preventive measures, the incidence of diphtheria in the season increases by 3-4 times.

PATHOGENESIS AND PATHOLOGICAL ANATOMY

Diphtheria is a toxic disease, the development of which is crucial to the macroorganism (his individualdual features, sensitivity to pathogenic factor in the resistance of the tissue in the gate of infection, age, condition of nervous system, immunological properties, etc.). In each case there may be one or a combination of several factors with a predominance of any one contributing to susceptibility to diphtheria infection and disease development.

According to A.D. ADO (1960), with normal function of the nervous system, the body's defenses are mobilized, ensuring the elimination of the disease in its initial period. Functional and organic disorders of the Central nervous system reduce the ability to mobilize the body's defenses, and a person becomes ill when a pathogenic microbe enters.

V. D. Akhnazarova (1959), Tonutti (1950), Frick, Lampl (1952), and Schmid (1957) in animal experiments established thatthe removal of the pituitary gland prevents the development of specific pathohistological changes in the adrenal glands and other internal organs characteristic of diphtheria intoxication. Thisled the authors to suggest that hormonal factors are of great importance in the pathogenesis of the diphtheria process, in particular in the origin of local tissuedisorders, and not the direct action of diphtheria toxin onthe corresponding organs.

An important factor in the formation of diphtheriaprocess, in its localization, and in the severity of the disease isthe age factor and the patient's Constitution. Thus, adults and older children more often develop pharyngeal diphtheria, and infants-diphtheria of the larynx, nose and rare localizations. According To S. N. Rozanov (1948), croup affects children under the age of one year, nasal diphtheria - up to 6 months, and the toxic form of pharyngeal diphtheria - children older than 2 years. V. I. Molchanov (1960) explains the age difference in the formation of diphtheriaof various localization by anatomical and physiological features of the child's body. The rarer disease of infants with pharyngeal diphtheria is associated with underdevelopment of the tonsils, the absence of nerve receptors in the mucous membrane and lymphatic apparatus of the pharynx. Often toxic orhypertoxic forms of diphtheria develop in children with a thymic-lymphatic Constitution, accompanied by thymus dysfunction, exudative-lymphatic diathesis.

The peculiarity of the clinical course of toxic and hypertoxicforms of diphtheria is regarded as a manifestation of an altered reactivity of the body, expressed in anaphylactoid-allergic reaction caused by specific and non-specific sensitization, which either precedes the infectious process, or occurs during it. The sensitized organism reacts strongly and rapidly to the invaded infection: there is a very fast and strong connection of the toxin both at the site of its penetration into the body, and in tissues that are particularly susceptible to it (the nervous system, heart, adrenal glands, kidneys, etc.), which makes it difficult to neutralize the antitoxin.

Confirmation of the allergic theory are similar clinical and pathomorphological shifts observed both in the initial period of diphtheria, and in anaphylactic and allergic conditions. It is the presence of vascular-hemodynamic phenomenon A. A. Koltypin in the form of tachycardia and

lower blood pressure, swelling of the cervical tissue, hemorrhages, large raids with necrotic collapse, thrombocytopenia, small heart, acute emphysematous expansion of the lungs, characterized capillaroscopic picture, a feature of the initial lesions in the lungs in the form of interstitial pneumonia, serous myocarditis, the waviness of the disease, etc.

An important significance in the pathogenesis of the toxic form of diphtheria is attached to the reactive state of the microorganism,which depends on environmental factors and the function of the cortex of the large hemispheres ofthe brain. When the infectious process develops, in addition to specific stimuli, there are also non-specific, additional stimuli that strengthen or weaken the resistance of the macroorganism, contributing to or hindering the development of the disease. In this case, additional stimuli can precede or accompany the action of a specific one. For example, toxic forms of diphtheria are more often observed in unorganized children, since organized children observe a certain regime that supports theiremotional positive tone (S. V. Weiss, 1950; A. A. Markova, 1958). Weakening of the higher nervous activity as a result of its overstrain is accompanied by a decrease in the resistanceof the organism to diphtheria toxin and phagocytic activity of leukocytes. The course of diphtheria intoxication in animals is influenced by the seasonality factor - in the spring-summer period, the disease is more severe than in autumn-winter.

Toxic forms of diphtheria in some cases are the result of a mixed diphtheria-streptococcal infection, as evidenced by bacteriological indicators (studying the microflora of the throat and nose, hemoculture, determination of anti-0-streptolysin, etc.) in patients with diphtheria and in experimental animals. Against the background of streptococcal infection, evensmall subinfectious doses of diphtheria toxin can cause a picture of severe diphtheria intoxication,leading to the death of animals.

Both local changes and General phenomena observed in diphtheria are a consequence of the protective reaction of the macroorganism and the damaging action of the toxin.

The entrance gate is usually the mucous membranes of the oropharynx (microbes use mucus as a habitat), nose, larynx, rarely the eyes, genitals, and skin. Characteristic local changes occur in diphtheria at the site of introduction and localization of infection. The pathogen penetrates deep into the mucous membrane or skin, where it multiplies and produces a toxin. The latter is gradually, in small portions, absorbed into the lymph and bloodstream, acts on the nerve endings embedded in the walls of local vessels. As a result, there are motor disorders in the lymphatic and blood vessels, local congestive hyperemia develops. The intake of arterial blood and nutrients through the lymph flow in this area decreases sharply. Hematopathology damaged barrier increaseis declared as the permeability of the walls. In the intercellular space of this site, exudate is formed. At first, the supply of white blood cells for phagocytosis increases. When exudate reaches the surface of necrotised layers under the influence oftoxin, it turns into fibrin. This is facilitated by tissue thromboplastin, released due to coagulation necrosis of cells of the epidermis or epithelium. On the surface of the affected integument, a fibrinous plaque forms, which, in addition to fibrin and necrotised surface tissues, includes diphtheria microbes, their toxin, any other microflora, and leukocytes. Due to the solderability of fibrin fibers, the plaque becomes elastic in consistency. It is formed during the first days of the disease. When forcibly removed on these days, the plaque is formed again. Its detachment occurs either under the influence ofantitoxic therapeutic serum, or due to the formation ofantitoxic immunity as the macroorganism fightsinfection. Depending on the protective forces of the macroorganism andthe locus of infection, diphtheria, croup or catarrhal inflammation develops. The process involves regional lymph nodes, they increase due to a sharp fullnessof blood, edema and proliferation of cellular elements. Inthe immediate vicinity of the affected lymph nodes, there is an edema of the subcutaneous tissue of the neck. This edema is caused by serous inflammation with numerous cellular infiltrates, and the General toxic effect is caused by a toxin entering the blood.Of great importance in the pathogenesis of toxic and hypertoxic forms is the preliminary sensitization of the body as a result of diseases suffered shortly before diphtheria. Fixation of the toxin in tissues leads to characteristic lesions of the nervous and cardiovascular systems. In the myocardium,-parenchymal degeneration of muscle fibers occurs early, up to complete myolysis and blocky disintegration. It is characterized by fatty degeneration with subsequent destruction of myofibrils andthe

formation of diffuse sclerosis. Changes in the peripheral nervous system occur in the type of parenchymal neuritis. With the development of polyneuritis, especially dangerous is the defeat of the laryngeal, intercostal, and diaphragmatic nerves, leading to paralysis of the respiratory muscles with the development of acute respiratory failure, which is one of the causes of death. Changes in other organs are mainly characterized by a toxic lesion. In the adrenal glandblood circulation disorders, cell destruction up to complete necrosis and disintegration can occur in the kidneys. In the kidneys, a picture of toxic nephrosis often occurs.

CLINIC

Depending on the degree of infection penetration and the response of the macroorganism, various clinical forms of diphtheria developin terms of localization and severity.

The incubation period for diphtheria is from 2 to 10 days.

Clinical forms: due to the different localization of the local process and the severity of General toxic phenomena,diphtheria differs in a variety of clinical forms.

1.Diphtheria localized:

a) the most common (throat, nose, larynx);

b) rare localizations (skin, eyes, mouth, ear, navel wounds in the newborn, external genitalia, etc.).

2. Diphtheria is common. Local changes are present in at least two adjacent or non-contiguous organs (pharynx + larynx, pharynx + nose, pharynx + mucous membrane of the mouth or pharynx, nose + skin, pharynx + external genitalia, etc.).

3. Toxic diphtheria - severe diphtheria, accompanied by significant toxicosis and edema of the subcutaneous tissue (throat, nose and rare localities).

4. Diphtheria-mixed (streptodiphtheria, diphtheria + scarlet fever, diphtheria + measles, diphtheria + chicken pox, etc.).

5. According to the nature and severity of the local process, it is divided into typical and atypical.

6. According to the severity of the disease, it is divided into light, medium-heavy and severe.

The majority of patients (66.7-82%) tolerate a localized form of diphtheria. The second place is taken by toxic diphtheria, then-common and diphtheria of rare localization.

In some years, there have been changes in the ratio of clinical forms of diphtheria, which many authors attributeto changes in the age structure of patients — increasedincidence in older age groups. The increase in toxic forms during the period of sporadic morbidity can probably be explainedby the occurrence of diphtheria in unvaccinated or immunobiologically weakened individuals. Apparently, this is also due to the fact that the calculation of the relative frequency of various forms of diphtheria in recent years is made from small numbers, many times smaller than in 1957-1958. In addition, in previous years, there was a hyperdiagnosis of diphtheria, and now its diagnosis has improved.

Most often, the diphtheria process is localized in the oropharynx. Throat diphtheria accounts for 90% of all cases. There are localized, widespread and toxic forms of throat diphtheria. Localized oropharyngeal diphtheria is the most common form.

With *a localized form* of plaque are located only on the tonsils and do not go beyond their limits. The disease begins acutely, usually with an increase in body temperature to 38-39°C and the appearance of weakly expressed symptoms of intoxication. Children complain of General malaise, headache, minor pain when swallowing,

Upon examination, the General condition of the child is little disturbed,there is a slight increase in regional lymph nodes on both sides, less often on one side. The lymph nodes are slightly painful and mobile.

Depending on the nature of the pharyngeal lesion, there are three types ofpleural, insular and catarrhal forms of localized pharyngeal diphtheria. The most typical filmy (solid) form, in which the film of grayish color, smooth with a pearlescent luster, with fairly well-defined edges, covers the entire tonsil. The film is located on the surface of the tonsils, it is difficult to remove. When it is forcibly rejected, the tonsillar tissue bleeds, and the filmy plaque itself is not rubbed between two slides.

In the insular form of localized pharyngeal diphtheria, the plaque has the form of Islands of various sizes, usually located outside the lacunae on the inner side of the tonsils. The edgesof insular lesions are often uneven, as if they are crawling onto the tissue of the tonsils. In this form of diphtheria, the temperature is usually subfebrile. The General condition of the child is little disturbed.

Catarrhal form refers to atypical forms of throat diphtheria. In this form, the most characteristic sign of diphtheria is absent-fibrinous plaque. The leading symptoms are hyperemia and some swelling of the tonsils. There may be a feeling of scratching or clumsiness when swallowing. The temperature usually does not rise, and there are no symptoms of intoxication. Diagnosis in such cases is possible only on the basisof epidemiological data and the detection of toxigenic diphtheria Bacillus.

Localized forms of pharyngeal diphtheria without specific treatment can progress and become common.

Widespread diphtheria of the throat is less common than Lockerealized. The plaque thus extends beyond the tonsils to the mucous membrane of the Palatine arches, the uvula. Symptoms of intoxication are moderate. The disease begins acutely with an increase in temperature. General weakness, headache, sleep disorders, and sometimes vomiting are characteristic. The patient is pale, listless, complains of sore throat. Regional lymphatic systems . nodes are enlarged to a large bean, are sensitive, but there is no edema of the cervical fiber.

The toxic form is the most severe form of throat diphtheria. The disease begins violently. From the first hours of the disease, thebody temperature rises to 40°C, children become lethargic, sleepy, complain of severe weakness, headache and sore throat, sometimes abdominal pain, in the neck. In the throat from the first hours of the disease, there is a diffuse hyperemia and edema, which oftenprecedes the appearance of plaque. With a pronounced swelling of the tonsils touching. The soft palate, arches and small tongue are swollen. Plaque initially has the form of a delicate web-like mesh or jelly-like film, which is easily removed, but in their place quickly reappear, thicken, compact and spread. Already on the 2-3 - th day of the disease, thick, dirty - gray deposits completely cover the surface of the tonsils, pass to the arches, a small tongue, a soft and hard palate. Pharyngeal hyperemia by this time decreases, has a bluish tint, pharyngeal edema reaches maximum severity. The tongue is coated, the lips are dry, cracked, and the mouth has a peculiar sweet-cloying smell. Breathing through the nose is difficult, snoring, from the nose sucrovichnye discharge that irritates the skin around the nose, sometimes visible films on the septum of the nose. The voice is strangled with a nasal tinge.

It is characterized by damage to the lymph nodes and the appearance of edema of the cervical fiber. All the cervical lymph nodes are enlarged, sometimes they form a large conglomerate, the size of a chicken egg. When palpated, they are elastic andpainful. The skin covering over the edematous tissues is not changed, the pressure is painless and does not leave pits.

Depending on the severity and prevalence of cervical edema, there is a distinction between toxic diphtheria of the first degree-edema of the cervical fiber reaches the middle of the neck; toxic diphtheriатерию of the second degree - edema of the cervical fiber to the clavicle, toxic diphtheria of the third degree - edema of the cervical fiber below the clavicle.

From the toxic form, it is necessary to distinguish *the subtoxic form* of pharyngeal diphtheria, in which there is a slight swelling or pastyness of the cervical tissue in the region of regional lymph nodes. However, pharyngeal edema in these patients is clearly expressed. Subtoxic diseases also include pharyngeal diphtheria, accompanied by unilaterallesions (plaque and edema of the cervical tissue on one side). The most severe forms of diphtheria include hypertoxic and hemorrhagic forms.

The *hypertoxic form* is characterized by pronounced symptoms of intoxication: hyperthermia, convulsions, collapse, unconsciousness, extensive plaque and pharyngeal edema. The course of the disease is lightning-fast. Fatal outcome in this form usually occurs even before the development of complications - on the 2-3 th day of the disease from rapidly progressing cardiovascular insufficiency withthe phenomena of collapse.

In *the hemorrhagic form*, there is a hemorrhagic syndrome in the form of blood soaking of

plaque, multiple bloodspills on the skin, bleeding from the nose, throat, gums, and gastrointestinal tract. The prognosis for this form of diphtheria is also very serious. A more favorable prognosis is for toxic diphtheria with hemorrhagic syndrome, in which thereis a hemorrhagic infiltration of the tonsils, petechiae on the skin, but no bleeding.

The course of toxic pharyngeal diphtheria mainly depends onthe timing of the start of complex and, first of all, specific treatment. With timely administration of anti-diphtheria serum, the symptoms of intoxication quickly disappear, and the attacks are rejected by day 6-8. After the rejection of the plaque, surface necrosis remains for some time. Atthe same time, edema of the cervical tissue decreases and disappears. In the absence of timely specialtreatment, the disease usually progresses and only in cases of localized diphtheria recovery is possible, however, complications in the form of myocarditis orperipheral paralysis may occur.

Diphtheria croup. When the process is localized in the respiratory tract, diphtheria croup occurs-a clinical syndrome accompanied by a hoarse or hoarse voice, a rough barking cough and difficult (stenotic) breathing.

Diphtheria croup can be isolated (only the Airways are affected) or combined (combined-inflammation of the Airways and the pharynx or nose). In most patients, croup is isolated.

Depending on the distribution of the process, there are two main types:

1) localized diphtheria croup (diphtheria of the larynx);
2) diphtheria croup is common:

a) diphtheria of the larynx and trachea,

b) diphtheria of the larynx, trachea and bronchi - laryngotracheobronchitis.

The disease begins with a moderate increase in temperature (up to 38°C), the appearance of mild intoxication (lack of sleep, decreased appetite), a rough, barking cough and hoarseness of voice. These symptoms correspond to the first period ofdiphtheria croup-the stage of croup cough (catarrhal period). The duration of this period in some cases does not exceed a day, in others — 2-3 days. The further course of diphtheria croup is characterized by a steady progression of symptoms and a gradual transition to the second stage — stenotic, which is characterized by stenosis of the Airways, breathing becomes difficult, noisy, there is a retraction of pliable places of the chest (intercostal, supraclavicular and subclavian cavities, the jugular fossa), tension of the auxiliary respiratory muscles (sternoclavicular - mastoid, trapezoid, etc.). the Voice in this period is persistently hoarse or aphonic, cough at first rough, "barking", gradually becomes silent. The stenotic period lasts from a few hours to 2-3 days.

At the end of the stenosis stage, there is respiratoryinsufficiency due to a gas exchange disorder. There is a transition period from the stage of stenosis to the stage of asphyxia. During this period, in addition to breathing with extended breath, deep vtjazhenija compliant places chest, and Athos, a strong anxiety, fear, sweating of the head, cyanosis of the lips and nasolabial triangle, loss of inspiratory pulse ("pulsus paradoxus"). If in this period the patient is not helped, the asphyxic stage occurs. In this period, breathing is frequent, shallow, and arrhythmic. It becomes less noisy, and the retraction of pliable areas of the chest decreases, the child calms down as it were. The condition is extremely serious. The skin is pale gray, cyanosis not only of the nasolabial triangle and lips, but also of the tip of the nose, fingers and toes. Muscle hypotension, cold extremities. The pupils are dilated. There is no reaction to injections. The pulse is frequent and thready, the blood pressure falls. Consciousness is darkened or absent, sometimes there are convulsions. Involuntary passage of feces and urine. Death from asphyxia occurs.

Progression of diphtheria croup with a sequential change of the stages described above, up to asphyxia and death, is observed with late treatment to the doctor or improper treatment. With timely specific therapy, the sequential development of all stages of diphtheria croup is not observed. Under the influence of diphtheria serum after 18-24 hours, the clinical manifestations of the disease do not progress. The child calms down, then gradually the retractions of pliable places of the chest disappear, the breathing becomes even and deep. The cough goes from dry to noiseless and becomes more gentle, moist, and thenstops. The voice remains soundless or hoarse for a long time, and becomes normal only 4-6 days after the stenosis disappears. In some cases, the rejected films can

cause complete obturation of the respiratory tract and sudden asphyxia. Diphtheria croup is most often complicated by pneumonia.

Nasal diphtheria is more common in children from infancy to 3 years of age, sometimes at an older age and even in adults. Inrecent years, it is less common (from 10.9% in 1957-1958, it fell to 2.9 % in 1965-1971).

Distinguish primary and secondary diphtheria of the nose. The latter usually develops as a result of the spread of the process from the pharynx or larynx. In such cases, its clinical picture is obscuredby manifestations of pharyngeal or laryngeal diphtheria. Primary nasal diphtheria is observed in 61 % of cases, and in 39 % of cases it is combined with diphtheria of other localities.

According to the nature of inflammation, there are typical (filmy) and atypical (catarrhal and erosive) forms.

When the filmy form in young children appears snoring due to swelling of the nasal mucosa. After 2-3 days, a mucous or serous fluid begins to be released from one or both nostrils, which soon becomes bloody and purulent. Periodically observed nosebleeds. The skin at the nasal entrances and upper lip is macerated in the form of a furrow. On the nasal septum, films are formed, which can rarely spread to the shells, the bottom of the nose. Sometimes scattered dry crusts appear on the cheeks, forehead, chin, apparently of a specific nature, which disappear soon after the introduction of serum. It is difficult to breathe, especially during sleep, because of this, sleep becomes anxious, the child often wakes up, cries, and breathes with an open mouth. Breast-feeding is difficult. The temperature can be normal, subfebrile, and rarely high. Diphtheria of the nose in children of the first months of life sometimes occurs without discharge from the nose in the form of breathing difficulties.

In the absence of specific treatment, the films can spreadto the mucous membrane of the nasal appendages, through the choanas to the posterior surface of the soft palate and nasopharynx. The introduction of therapeutic serum leads to a rapid reverse development of the process.

The filmy form of nasal diphtheria in older children can occur without disturbing the General well-being in the form of a prolongedrunny nose for 2-3 weeks. with sukrovichnye detachable and often, the diagnosis of diphtheria is established by accident.

The catarrhal form of nasal diphtheria is characterized by persistent bacterial discharge and often chronic recurrentleakage. There is a distinction between wet and dry forms. In the catarrhal-humid form, the mucous membrane of the lower nasal conchs does not swell, thenasal cavity is filled with liquid serous masses that eat away its wings. The dry form is characterized by dryness and fragility of the nasal mucosa with a viscous separable. On the mucous membrane and at the nasal passages, bloody crusts form and persist for a long time. The course of the catarrhal form is favorable.

Diagnosis of catarrhal and erosive forms of nasal diphtheria is difficult, so they are called laboratory forms.

Diphtheria of the nose can be acute, subacute, and chronic, often relapsing course buying. Acute course is usually characteristic of the filmy form, chronic-forthe causative and erosive. Diphtheria of the nose due to the addition of a secondary infection can be complicated by purulent otitis, limfadenitis, pneumonia, etc.

Diphtheria of the throat and nose. On the 3rd-5th day of the disease, films from the tonsils spread into the nasal cavity or, passing the palate and pharynx, into the nasopharynx and the back of the nose. But in some cases, it is possible to transfer plaque from the nasal cavity to the tonsils.

The spread of the process is accompanied by a deterioration in the overall condition. From the nose appear first mucous, and then sucrovichnye discharge, corroding the skin around the nostrils and on the lips, the voice takes a nasal tone, breathing with an open mouth, dryness of its mucous membrane is noted. On the back wall of the pharynx, there are abundant fibrinous overlays, swollen subclavianand anterior anterior neck lymph nodes. Sometimes the process can go to the paranasal nasal cavity and middle ear, then there is swelling of the eyelids and the back of the nose, discharge from the ears.

Diphtheria of the throat and larynx. Usually, the diphtheria process spreadsfrom the pharynx

to the larynx along the walls of the pharynx or, bypassing it (secondary croup), occasionally there is a transition of attacks from the larynx to the pharynx. Clinically, the primary and secondary croup differ little from each other, the difference is only that with secondary croup, its symptoms begin to appear from the 3-4 th day of pharyngeal lesion.

In children with impaired nutrition, croup is more often combined with diphtheriaof the nose, skin, or throat. Symptoms of croup are unclear, not always marked by a rough barking cough. Often the phenomena of stenosis in them are mistaken for pneumonic shortness of breath.

If the croup is combined with a toxic form of pharyngeal diphtheria, then its clinic is expressed dimly, although a careful examination of the patient can reveal hoarseness of voice and stenotic breathing.

Diphtheria of rare localities includes diphtheria of the skin, wounds, eyes, oral mucosa, ear, external genitalia, etc. Isolated primary diphtheria lesions of these organs are extremely rare. It usually occurs as a result of the spread of diphtheria or the introduction of infection from its primary foci.

The frequency of this form in relation to all cases of diphtheria is 0.6-5.3%. According to many Russian authors, in recent years there has been a decrease and even disappearance of these forms of the disease.

According to who data on the frequency of various clinical forms of diphtheria, on the contrary, throat diphtheria is rare in a number of developing countries, since the population develops immunity early as a result of skin diphtheria in childhood. But in the process of urbanization in recent years, there has been a gradual increase in clinically expressed diphtheria ("who Chronicle", 1975, vol. 29, p. 317).

Diphtheria of the skin according to various authors, is between 0.5 and 20% from other sites. Diphtheria of the skin in young children, especially with impaired nutrition, occurs much more often than it is diagnosed (it takes the second place after diphtheria of the nose). The latter is due to anatomical and physiological peculiaritiesof the body of children of this age - insufficient development of the stratum corneum of the skin, its greater susceptibility to infection compared with other integuments.

Skin diphtheria is divided into typical (filmy) and atypical (non - filmy) forms; the filmy form — on lokalizovannuyuand toxic, and non-filmy depending on the nature of the emerging elements on the skin-on pustular impetigo-like, in the form of panaritium and phlegmony of the skin. Dependingon the timing of skin lesions, primary and secondary skin diphtheria are distinguished.

The typical (filmy) form of skin diphtheria in infants is characterized by the appearance of dense fibrinous films on the background of inflamed skin. Usually, the process is localized in the area of diaper rash on the neck, behind the ear, in the inguinal folds. In older children and adults, it occurs in the form of ulcerative-membranous forms, such as localized or toxic diphtheria. A long-term non-healing ulcer is formed with edematous reddish edges, covered with a greyish-dirty film and scanty separable material. In the toxic form with edema of the skin and subcutaneous tissue around the ulcer, symptoms of General intoxication are also observed.

Atypical (non-lamellar) form is diagnosed with difficulty. It occurs in children who are in contact with patients with diphtheria (often suffering from dystrophy, hypovitaminosis). Pustular elements appear on the skin, which do not respond to antibacterial treatment for a long time (weeks or months). Rash elements are usually located near natural openings: the nose, mouth, genitalia, and anus. They are characterized by polymorphism, dense infiltrated edges with a dark red or cyanotic hue. From the contents of the pus of these elements, it is possible to sow diphtheria Bacillus.

In patients with a descending croup who have undergone tracheostomy, it is possible to spread filmy plaque in the areaof the surgical wound.

Diphtheria of the umbilical wound in newborns can occur typically with the formation of a film or atypically in the form of a persistently healing suppurating ulcer with scant discharge.

Diphtheria of the eye. There are croup, diphtheria and catarrhal forms of eye diphtheria. The first place is taken by the croup form, the second - catarrhal and the third - diphtheria. Due to insufficient familiarity of doctors with the clinic of eye diphtheria, despite early treatment to the doctor,

most children are admitted to the hospital on the 4th-5th day of the disease.

The croupous form is characterized by superficial inflammation of the conjunctiva, accompanied by moderate or significant edema of the eyelids, especially the upper ones. The skin of the eyelids becomes hyperemic with a bluish tinge. The crease between the edge of the eye socket and the eyelid is smoothed out. The ocular fissure closes, and when it is opened, a plentiful serous-bloody fluid is released. Because of thedensity of edema, it is much more difficult to turn the upper eyelid out for examination. On the conjunctiva of the eye, grayish-yellow, easily removable overlays are found. Often, the removal of films is accompaniedby bleeding. First, the conjunctiva of one eye is affected, and after 2-3 days - the other. Characterized by low soreness and lack of photophobia. The cornea is not involved in the process, the vision does not suffer. Films and edema disappear 2-4 days after the introduction of serum.

With diphtheria, there is often an unfavorable prognosis (possible loss of vision). Patients develop dense edema of the eyelids, the eyes open with great difficulty, from them appears at first a meagre, and then a plentiful serous-blood discharge. The conjunctiva swells sharply and becomes covered with a dense fibrinous plaque, which often spreads to the cornea. Areas of the conjunctiva that are free of plaque are swollen, hyperemic, and bleed. By the 4th-5th day, the cornea usually becomes cloudy diffusely or in a limited area, its surface is eroded, and an infiltrate is formed in the center, penetrated by vessels with foci of epithelial degeneration. After that, the discharge from the eyes becomes even more abundant, turns into purulent. The edema of the eyelids decreases, the plaque gradually goes away. The films are rejected in the form of plates, after which there are always scars on the conjunctiva. In 3-4 days after the rejection of the films, the cornea brightens. With the restoration of the iris and pupil pattern, the injection of scleral vessels gradually disappears. General disorders in this form of diphtheria are expressed in the form of high temperature, adynamia, pallor.

Under the influence of specific treatment, recovery isaccelerated. Almost always, vision suffers to some extent, up to complete loss of it as a result of panophthalmitis. With a favorable course, clinical recovery occurs by the end of the 2nd week.

The catarrhal form of diphtheria of the eye is difficult to distinguish clinically from other conjunctivitis. It is accompanied by edema of the eyelids, conjunctival hyperemia and abundant purulent discharge. This form is diagnosed only on the basisof positive bacteriological confirmation of diphtheria, epidemiological data and the effectiveness of serum treatment.

Diphtheria of the oral mucosa is an extremely rarephenomenon. Until 1959, in our clinic, it was observed in 0.6-4.7% of children aged 1-4 years, and then it was not registered at all. The diphtheria process can occur on the mucous membrane of the cheeks, lips, gums and tongue usually in the form of a relatively large ulcer of a rounded or oval shape, covered with a fibrinous film with infiltrated edges and edema of the mucous membrane around. Diagnosis of isolated lesions of the oral mucosa is very difficult and it is necessary to conduct a bacteriological study.

Ear diphtheria is much more common than it is diagnosed. In 8% of infants, diphtheria otitis media was detected. Diphtheria otitis can be both primary and secondary as a result of the spread of the process from the nose, throat and larynx. The disease is localized (only the middle ear is affected) and widespread. The common form is severe, sometimes accompanied by repeated vomiting, diarrhea, drowsiness or, conversely, agitation as a result of complications of antritis, mastoiditis, meningitis caused by a secondary infection. Such cases can be fatal.

The disease is characterized by a prolonged and severe course. Otoscopy reveals significant edema, infiltration of the mucous membrane. On the eardrum appear ulcers, necrosis or fibrinous plaque. The same lesions are found on the mucous membrane of the middle ear and on the skin -the external auditory canal. In addition to specific otitis media in diphtheria, latent otitis media are often detected without subjective symptoms, which are detected inspecial targeted studies. A. p. Odoevsky (1958), examining 71 patients with diphtheria, found otitis media in 80.3% of them. The frequency of otitis media is in accordance with the severity of the course of diphtheria and the age of the child. This disease is more often observed in young children and is characterized by an easy course, the absence of spontaneous perforation of the eardrum. The nature of the course of these

aseptic otitis media allows A. p. Odoevsky to consider them the result of local manifestations of infectious allergies. Moreover, the mucous membrane lining the middle ear cavity has a common embryonic origin with the pharyngeal mucosa.

Diphtheria of the external genitals is rare (within 0.1-1.1% of cases), mainly in girls aged 5-8 years as a secondary localization in throat or nose diphtheria. But sometimes there can be a primary localization, usually in young children as a result of ingestion of the pathogen from the bacterial carriers caring for them (during their toilet of the genitals of children).

There are localized, widespread and toxic forms of diphtheria of the genitals. In the localized form, the labia majora, clitoris, or prepuce are locally affected; in the common form, local inflammation spreads to the area betweenand on the skin around the anus or external genitalia. In the toxic form, there is a pronounced edema of the genitals (I degree), subcutaneous tissue of the inguinal regions and thighs (II degree).

A constant symptom for all forms of diphtheria of the genitals is edema with dark cherry hyperemia of the affected mucous membranes. It is caused by paralytic expansionof blood vessels, venous stasis and blood filling. It is characterized by an increase and soreness of the inguinal lymph nodes, as well as a disorder of urination. The disease usually occurs with a slight violation of the General condition.

Lesions can be typical for diphtheria with the presence of filmy plaque on the mucous membrane and atypical (without plaque) in the form of a catarrhal-ulcerative process with a sucrovichnopurulent discharge.

In the typical form, a dirty diphtheria film is formed, tightly embedded in the mucous membrane of the large lips or prepuce. Forcible removal of it is achieved with difficulty and is accompanied by bleeding.

The catarrhal form begins suddenly with a delay inurination, the appearance of pain, abundant fetid blood-purulentdischarge from the vagina or from under the prepuce, in which diphtheria bacilli are found. The inguinal lymph nodes are enlarged and becomesoft and painful.The mucous membrane of the vestibule or prepuce swells, becomes dark cherry-colored. In women, genital diphtheria is most often observed in the form of ulcers located on the labia majora and minora, at the entrance and in the vagina itself. Sometimes the process extends to the skin of the pubis, thighs, buttocks and perineum, it takes the form of pustules, vesicles, eczema, impetigo, ectim.

In the toxic form of diphtheria of the genitals, there can be such severe complications as myocarditis, polyneuritis, nephrosis, with fatal outcomes.

Diphtheria of the gastrointestinal tract is very rare, usually combined with a common or toxic form of diphtheria of other localities. During life, this lesion is not diagnosed, since there is no specific symptom complex characteristic of stomach damage.

According to the literature, with fibrinous gastritis, vomiting of mucus with streaks of blood and sometimes with fragments of films appears, which persistently continues until death. There is noquenchable thirst, frequent paroxysmal pain in the abdomen and flatulence (a constant symptom).

Usually, individual cases of diphtheria of the esophagus and stomachare a pathoanatomic finding in particularly severe, malignantforms of the disease with widespread fibrinous-inflammation.

Diphtheria of the lungs is an extremely rare localization of infection. It is usually combined with the defeat of diphtheria of the upper respiratory tract (larynx, trachea, bronchi).

Diphtheria-mixed. In some cases, diphtheria can be combined with any acute infectious disease (measles, scarlet fever, whooping cough, influenza, chicken pox, etc.) or due to the addition of diphtheria to other infections, or their layering on diphtheria. Diphtheria, which has joined scarlet fever, is characterized by a more pronounced local inflammatory reaction with a slow disappearance of filmy deposits and a more abrupt and prolonged reaction from the side ofregional lymph nodes. Subtoxic and toxicforms of pharyngeal diphtheria occur with prolonged edema and intoxication. The overall reaction is characterized by hyperergic (persistent leukocytosis, fever).

In diagnostic terms, cases of diphtheria joining to scarlet fever in the initial stage, when the initial scarlet tonsillitis is still pronounced, are particularly difficult. However, when dense fibrinous

deposits appear, often spreading beyond the tonsils, the presence of diphtheria should be assumed. Usually, during this period with scarlet fever, the plaque is purulent, loose, and easily removed. In such cases, laboratory methods of investigation (bacteriological, serological) provide significant assistance. At the same time, it should be remembered that diphtheria bacteriocytosis is possible in 2-11% of scarlet fever patients, especially during an outbreak of diphtheria. Joining scarlet fever to diphtheria is usually accompanied by necrotic angina with subsequent purulent complications (lymphadenitis, otitis, mastoiditis, etc.).

According to the observations of T. N. Nikonova, O. D. Ten (1960), the combination of diphtheria and measles is characterized by a decrease in the overall reactivity of the body, patients have poorly expressed inflammatory changes in the throat, minor intoxication, persistent leukopenia with relative lymphocytosis. According to V. M. Molchanov (1960), measles creates an increased predisposition to diphtheria. Most often, the diphtheria process is localized in the larynx and nose. With croup that has developed in the late period of measles, diphtheria should be suspected.

Diphtheria croup occurs in measles with some specialfeatures: a mixed type of dyspnea, persistent aphonia, slow resorption of film deposits, recurrent character of stenosis and almost always complicated by pneumonia, so the prognosis is very serious. Chickenpox, which has joined diphtheria, contributes to a more severe course of the disease. At the same time, toxic forms of pharyngeal diphtheria are more often observed, exacerbation of long-latent nasal diphtheria is provoked, clinical manifestations of diphtheria complications (myocarditis, polyneuritis) worsen, the time of bacterial excretion in convalescents is prolonged.

Streptodiftheria is of particular interest asa result of combined diphtheria-streptococcal infection. Joining streptococcal infection to diphtheria contributesto the more frequent development of severe forms and complications.

In some cases (0,2-2,4%) the clinical picture is onso pronounced that stratolifter easy diagnosisshall be assured. It is characterized by a pronounced temperature andan inflammatory reaction on the part of the pharynx. Develops a bright hyperemia of the pharyngeal mucosa, separated from the solid palate by a demarcation line, with a pronounced pain syndrome along with typical filmy plaque and painful regional lymphadenitis.

Clinic of diphtheria in vaccinated children. The occurrenceof disease in vaccinated people is possible due to a low levelof antitoxic immunity. Insufficient immunity in these patients is explained by violations in the conduct of primary vaccination and revaccination. In addition, it is also possible to reduce the intensityof immunity after infectious diseases. In vaccinated children, toxic forms of the disease and complications are much less common.

The course of diphtheria in vaccinated people is usually smooth. Symptoms of intoxication disappear on the 3-5th day of the disease, the throat is cleared on the 5th-7th day. With a localized form, recovery is possible without the introduction of serum.

However, these features can be traced only in those children who have the disease occurs against the background of residual anti-diphtheria immunity. In cases where vaccinationimmunity is completely absent (refractor children), severe toxic forms can occur with complications and a fatal outcome. The clinic of diphtheria in such patients practically does not differ from that of unvaccinated patients.

COMPLICATIONS

The most characteristic complications of diphtheria occur from the cardiovascular system (myocarditis),peripheral nervous system (neuritis and polyneuritis), and kidneys (nephrotic syndrome). Complications of diphtheria are associated withspecific intoxication and occur, as a rule, in toxic forms, with late treatment with antidiphtheriaserum. In toxic pharyngeal diphtheria of the third degree and especially in hypertoxic forms, massive toxemia can lead to the development of acute cardiovascular insufficiency due to a hemorrhage in the adrenal glands. At the same time, the patient's blood PRESSURE drops, the pulse becomes weak, thread-like. The skin is pale, cyanotic. With increasing phenomena of vascular collapse, death can occur.

Myocarditis occurs at the end of the 1st-beginning of the 2nd week of the disease. There are

muffled tones of the heart and expansion of its borders, systolic noise, sometimes extrasystole. My pulse is racing. Children complain of weakness and poor health. In severe cases, signs of myocarditis develop rapidly. The General condition becomes very severe: increasing pallor, cyanosis of the lips, AdinaMIA. A triad of symptoms is characteristic: acute dilatation of the heart's borders, repeated vomiting and abdominal pain due to an acute increase in the size of the liver. Heart rhythm disorders quickly appear and progress: extrasystole, sinus arrhythmia, gallop rhythm. The blood pressure falls. Patients die with symptoms of progressive cardiac weakness. For the early diagnosis of myocarditis, electrocardiographic examination is of great importance. The ECG showsa decrease in the voltage of the teeth, a shift in the interval S-T,a negative t-wave, and sometimes signs of a block of the GIS beam.

Nephrotic syndrome is noted in the acute period of the disease, at the height of intoxication. In the urine, high proteinuria, hyaline and granular cylinders are detected with a small number of red blood cells and white blood cells. Clinically, the syndrome does not appear, disappears as the symptoms of intoxication decrease and recovery.

A typical complication of diphtheria is peripheral paralysis. Distinguish between early and late diphtheria paralysis. This division is somewhat arbitrary. Early paralysis occurs in the 2nd week of the disease. The cranial nerves are usually affected. Paralysis of the soft palate occurs more often. The voice becomes nasal, the child can not blow out the burning candle, liquid food spills out of the nose, there is no reflex from the soft palate. Palatine ZanaVeska is motionless, drooping or asymmetric (in a unilateral lesion), while the small uvula is deflected to the healthyside. In rare cases, paralysis of accommodation occurs: patients are not able to distinguish small objects, can not read. Even less frequent are external ophthalmoplegia, facial palsy etc.

Late paralysis occurs in the 4th-5th week of the disease,which is characterized by the type of polyradiculoneuritis. They are characterized by all the symptomsof flaccid peripheral paralysis: reduced tendon reflexes (usually in the lower extremities), muscle weakness, impaired coordination, uncertain gait up to complete immobilization in the extremities. In severe cases, themuscles of the neck and torso may be damaged; the patient cannot sit or hold his head.

In addition, paralysis of the larynx (voice and cough become silent), damage to the pharynx (the patient can not swallow food or even saliva), paralysis of the diaphragm (paradoxical-movements of the abdominal wall - retraction of the abdomen when inhaling),and damage to the mechanisms of innervation of the heart can occur. These symptoms occur in isolation or in various combinations and can threaten the patient's life.

For diphtheria polyradiculoneuritis (in the absence of respiratory muscles and diaphragm) are usually favourableing. Paralysis disappears after 1-3 months. with complete restoration of the structure and function of skeletal muscles.

DIAGNOSIS

The diagnosis of diphtheria is based on the detection of characteristic fibrinous, dense whitish-grayish deposits locatedon the surface of the mucous membrane or skin.

Clinical diagnostics is crucial, since it is not possible to delay the introduction of anti-diphtheria serum and waitfor the results of laboratory testing.

Among the methods of laboratory diagnostics, bacteriological research is of the greatest importance. The material collected with a sterilecotton swab from the lesion site is sown on electric media (Leffler, Klauberg, etc.) and after growing in athermostat at 37°C for 24 hours, a bacteriological study is performed. In case of detection of Corynebacterium diphtheria, a preliminary result is given, and the final result is 48-72 hours after studying the biochemical and toxigenic properties of the isolated pure culture. Testing of isolated cultures for toxigenicity is crucial for confirming the diagnosis of diphtheria, especially in dubious and difficult to diagnose cases.

It is possible to determine the toxigenicity of corynebacteria in in vivo - on Guinea pigs, but in practical work the determinationis carried in out in vitro - on dense nutrient media by the methodof precipitation in Ouchterloni agar.

Serological methods of investigation are based on the detection of antimicrobial antibodies in

RPA. The reaction is put with a culture of diphtheria Bacillus. It is considered positive if there is an increase in the titer of antibodies in the dynamics of the disease.

To determine the intensity of antitoxic anti-diphtheria immunity, the Schick reaction and the method of quantitative determination of antitoxin in the blood by Jensen in this infection are important.

DIFFERENTIAL DIAGNOSIS:

Localized diphtheria of the throat often have to differentiate from lacunar, follicular, monoplanes and other angina.

Lacunar angina is characterized by a bright spilled hyperemiaof the mucous membrane of the pharynx, the presence of purulent yellowish-gray overlays in the mouths of the lacunae that do not go beyond the tonsils, they are always easily removed and completely rubbed between slides, i.e. they do not have a fibrinous character.

In follicular angina, the mucous membrane of the pharynx is vividly hyperemic. The tonsils are enlarged and swollen. On the bulging areas of the tonsils, yellowish-gray suppuratedfollicles are visible, located under the mucous membrane. After their opening, small quickly healing defects of the mucusof the standing shell can be detected.

In Simanovsky-Plaut-Vincent angina, yellow-green overlays have a curd-like consistency, located on one tonsil. After rejection of the overlays, a deep ulcer is visible. There is a characteristic putrid smell from the mouth. Submandibular lymph nodes are enlarged on the affected side. In crops of mucus from the pharynx, a spindly stick and spirillae are found.

The common form of throat diphtheria is differentiated from necrotic sore throat in scarlet fever and fungal lesions of the throat.

Necrotic angina is distinguished from pharyngeal diphtheria by a brighthysteria of the mucous membrane, dirty-gray necrotic overlays thatare at the same level with the mucous membrane. In necrotic angina, there is a sharp pain when swallowing, a significant increase and soreness of the regional lymph nodes, a high temperature and pronouncedsymptoms of intoxication. In the blood there is a high leukocytosis, the formula shifts to the left, the ESR is increased.

In fungal angina, overlays are insular or continuous, are located superficially on the tonsils and other areas of the pharynx mucosa, are easily removed with a spatula, have a curd character and are completely rubbed between slides. The mucous membrane of the pharynx is little changed. Regional lymph nodes are not enlarged.

The toxic form of diphtheria throat infections, differencesbut fromoperating mononucleosis, peritonsillar abscess, mumps infection.

Paratonsillary abscess is characterized by an increase in temperature, the appearance of sharp pain in the throat, because of which the patient can not swallow even liquid food and saliva. Opening the mouth is difficult due to a painful trism. The process is usually one-sided. Noted bright redness of the throat on the affected side and bulging almindeligvis tissue. Plaque on the tonsils is usually absent or the remains of purulent overlays are visible along the course of lacunae.

In infectious mononucleosis, there is no pronounced intoxication, and pharyngeal edema is less pronounced. On the tonsils,there are bumpy or continuous false-film overlays, but unlike diphtheria, the overlays are loose, easily removed, crumbled and completely rubbed between slides. Infectiousmononucleosis is characterized by a systemic increase inlymph nodes, an increase in the liver and especially the spleen. A large number of atypical mononuclears are found in the blood.

Toxic pharyngeal diphtheria is sometimes mistaken for a mumps infection (epidemic mumps). The cause of diagnostic errors in these cases is edema of the cervical Cletechutka around the enlarged parotid salivary glands, which is often mistaken for toxic edema in pharyngeal diphtheria. However, with mumps infection, there is no damage to the tonsils and pharyngeal edema.

Differential diagnosis of diphtheria croup. Currently, diphtheria croup is rare, and it is incomparably more common to deal with croup syndrome in SARS.

Diphtheria croup is characterized by a weakly expressed beginning and gradual development of the main symptoms of the disease: dysphonia, reaching aphonia, a rough "barking" cough, which

then acquires a silent character and gradually, but progressively, onthe melting phenomena of stenosis. Croup in acute respiratory viral infections (flu, parainfluenza, etc.) is characterized by a sudden onset, oftenthe disease develops immediately with an attack of suffocation and a rough barking cough. In this case, the voice remains ringing or slightly hoarse, but the ringing notes are always preserved and usually appear during the attack and crying of the child. For acute respiratory infections accompanied by croup syndrome,catarrhal phenomena are characteristic, often high temperature andintoxication.

In rarer cases, diphtheria croup has to be differentiatedfrom croup syndrome, which occurs with measles, chicken pox, aphthous stomatitis and other diseases.

Sometimes there is a need to differentiate the croup from a pharyngeal abscess, laryngeal papillomatosis, foreign bodies in the respiratory tract. In the diagnosis of these conditions, great importance is attached to anamnestic data and the results of laryngo-and tracheobronchoscopy, during which it is possible to detecta foreign body, laryngeal papillomatosis or fibrinous film.

Success in the treatment of diphtheria depends solely on the timely administration ofantidiphtheria serum. The serum dose depends on the form and severity of diphtheria. Early administration of serum provides a favorable outcome even in severe toxic forms. Toprevent anaphylactic shock, 0.1 ml of serum is pre-injected under the skin,after 30 min. 0.2 ml and after another 1-1. 5 h. the remaining amount is intramuscularly administered.

All patients with diphtheria are subject to hospitalization in an infectious hospital. At the prehospital stage, emergency care is provided.

With the development of acute respiratory failure carry out activities on elimination of violations of the external breathing - intubation, tracheostomy.

Acute cardiovascular insufficiency is an indication for prescribing prednisone - 2.5 mg/kg, hydrocortisone - 10-20 mg/kg. In the absence of an effect, this dose is administered againbut after 20-3 Omin.

Detoxification therapy includes intravenous drip administration of hemodesis, rheopolyglucin, 5% glucose solution.

Emergency medical care also includes antibiotic therapy: 2 million UNITS of Benzilpenicillin I / m with repeated administration after 4 hours. Can be used tetracycline, macrolides, etc. antibiotics.

The main treatment of diphtheria celebrate the introduction of the antitoxic serum essence protopteridaerocky. If you suspect a toxic form of diphtheria, or diphtheritic croup serum was administered immediately: first, 0.1 ml of a 1:10 dilution of serum to 20 min again 0.1 ml of undiluted serum and in the absence of allergic reactions, the remaining dose is administered 30 min/m (with sub-form 40 thousand ME, toxic form I article – б0тыс ME, II St. - 80 thousand ED, III St-100 thousand ME). Half of the dose of the drug is administered intravenously, the rest-IV. Antidiphtheria serum in high doses is better administered after an injection of prednisone 120-240 mg.

Convulsive syndrome is stopped with 0.5% seduxen solution (2-4 ml in / m for adults and 1-0. 5 ml for children).

The dose of antitoxic anti-diphtheria serum (PDS) is individual and depends on the form of the disease and the timing of treatment initiation.

Anti-diphtheria serum doses

Clinical forms of diphtheria	Dose of MPD (thousand ME)	
	Dose	Method of administration
Localized diphtheria of the oropharynx, nose, eye, skin, genitals	10-20	V / m
common	20-30	V / m
subtoxic	30-40	V / m
toxic I art.	30-50	V / m, V/V
toxic II art.	50-60	V / m, V/V
toxic III art.	60-80	V / m, V/V
of respiratory Organs	localized 10-20	V / m
of respiratory Organs common, descending	20-30	V/m

Highly purified equine hyperimmune serum is used for treatment. The most pronounced effect is observed with the introduction of serum in the first hours of the disease. If there is no local process, the PDS is not entered.

Initial doses of PDS vary depending on the day of the disease, the prevalence of films in the throat and Airways, their density and the effect of therapy. Serum is administered mainly in/m, less often in / V, and the duration of treatment depends on the localprocess. Currently, for the treatment of patients, a high-titrated specific immunoglobulin, prothyphodifteric plasma, is used, while the dose of PDS can be reduced.

Mandatory is antibacterial therapy with one of the antibiotics: penicillin, gentamicin, rifampicin, etc. In severe cases, antibiotics are used in large doses and in combinations.

Therapeutic measures for diphtheria of the respiratory tract should include:

1. Specific detoxification of PDS;
2. Antibacterial therapy with one of the antibiotics with a bactericidal mechanism of action;
3. Elimination of inflammatory edema of the mucous membrane (prednisone 2-5 mg/kg / day parenterally, hydrocortisone inhalation-125 mg per inhalation after 4 hours, antihistamines);
4. Elimination of bronchospasm (10% solution of euphyllin IV, 0.5% solution of seduxen, lytic mixture);
5. Removing films with an electric pump;
6. Eliminating the phenomena of hypoxia (inhalation of humidified oxygen).

Along with this, the patient is shown diagnostic and sanitization bronchoscopy in order to removeinflammatory products. With the progression of stenosis in the pre-fixed stage, surgery is required. In case of laryngeal diphtheria, extended nasopharyngeal intubation with plastic tubes is performed. With common diphtheria of the respiratory tract-tracheostomy followed by drainage of the tracheobronchial tree.

With the development of ITS complex treatment of patients aimed at stabilizing hamedina-Miki: administered large doses of corticosteroids in accordance with the degree of shock - prednisone 5 - 10 mg/kg and hydrocortisone 20-75 mg/kg deoxycorticosterone acetate (DOX) - 0.5% solution 2.0 ml, for restoration of volume of BCC use of crystalloid solutions (5% p-p glucose p-p

ringer, Laborie - 1,5-2l/day). After that, rheopolyglyukin 400.0 ml, albumin 5-10% 200.0 ml is applied in / in a jet to raise blood PRESSURE, then drip. After the introduction of colloidal solutions, 10% R-R glucose is introduced with vitamins C, WB, KKB. The ratio of colloidal and crystalloid solutions should not exceed 1:2. According to the testimony prescribe anticoagulants, protease inhibitors, vasoactive funds.

Due to the fact that from the 5th-6th day of the disease, early myocarditis can develop, detoxification therapy is combined with the use of dehydrating agents (lasix, mannitol, lespenifril). At low unstable blood PRESSURE, an additional 1% R-R of mesaton 1.0 ml is added. The volume of infusion solutions during this period should not exceed 1 l/day.

To sanitize bacterial separators, clindamycin is used 150 mg 4 times a day, benzyl-pinicillin-novocainic salt according to botys. ED 2 times / day in / m, as well as cephalotin and cephaleandol, parenterally in average therapeutic doses. The duration of the treatment course is 7 days.

After undergoing localized diphtheria, follow-up is carried out within1 month, and after widespread and toxic forms of diphtheria, follow-up is carried out 3-6 months or longer.

PREVENTION

The main importance in the prevention of diphtheria is active immunization. For these purposes, diphtheria toxoid is used as part of the combined DDS vaccine (adsorbed pertussis-diphtheria-tetanus vaccine). Vaccination begins at the age of 3 months. Enter 0.5 ml of the DDS vaccine, three times with an interval of 30-40 days. After 1.5 years, the first revaccination is performed with the same vaccine and at the same dose. The second and third revaccination is carried out at 6 and 11 years of age with ADS-M-anatoxin (adsorbed diphtheria-tetanus toxoid with a reduced amount of anatoxin) at a dose of 0.5 ml.

Children who have relative contraindications to vaccination are vaccinated with ADS-M-anatoxin. According to the indications of ADS-M-anatoxin, adolescents and adults are also vaccinated.

TASKS

1. A.29-year-Old patient, a worker, was taken to the ENT Department in a serious condition with a diagnosis of "Peritonsillar abscess". I fell ill acutely 3 days ago. With a chill, the temperature rose to 38°C. He noted a strong pain in the throat, weakness. I was self-medicating - I used alcohol, gargled, took aspirin, analgin. On the 2nd day of the illness, the temperature increased to 39.5°C, I noticed an increase in the neck, the top button on the shirt was not fastened. Today, he feels even worse, it became difficult to swallow, there was a discharge from the nose, swelling of the neck increased, and his voice became strangled. He became very weak, could not go to the clinic, called a doctor, who sent the patient to the ENT Department.

On examination: General condition is severe, t-39,l°C. Pronounced swelling of the neck on both sides,extending to the ears and down-below the clavicle. The oropharyngeal mucosa is sharply edematous,the tonsils are almost closed, and are completely covered with a gray-white coating that extends to the Palatine arches and the uvula. The mucosa bordering the plaque is hyperemic with cyanotic swelling. The voice is strangled. From the nose Muco-purulent discharge. The skin in the nasal passages is macerated, and there is a film on the nasal septum. Shortness of breath, BDD-26 in min., BP 180/100 mm Hg.

1)Your diagnosis and its justification?

2)clinical management of the patient?

2. Patient C, 20 years old, military serviceman. Turned to the San. part in connection with the increase in temperature, chills, sore throat. Hospitalized 10 hours after the onset of the disease in the ENT Department of the hospital with a diagnosis of "Peritonsillar abscess". When trying to open the abscess, there was no discharge. A day after the onset of the disease, noisy breathing appeared with difficulty in inhaling. The condition is assessed as serious. Pale, cyanosis of the lips, temperature 37.8°C, position in bed forced-sitting with his head thrown back. The voice is hoarse. There is no

cough. Edema of the subcutaneous tissue of the neck to the clavicles. The mucosa of the anterior part of the oropharynx is sharply edematous, vividly hyperemic. The tonsils meet in the middle line. The entire surface of the tonsils, Palatine arches, soft palate and uvula is covered with a solid shiny dense plaque, dirty gray in color. BDD-28 V min., PS-100 beats per minute, rhythmic, AD 90/60 mm Hg.

1)Your diagnosis and its justification?

2)clinical management of the patient?

3. the doctor of the polyclinic is called again to the patient V., 18 years old, a student. Ill on the 7th day. Forthe pain began acutely, with a rise in temperature to 38.3°C, a scratchy throat, coughing, nasal congestion. I went to the doctor, noted an increase in the l / u neck, diagnosed "Adenoviral infection". I took Biseptol, but it did not get better, the temperature held. 2 days ago, there was a sore throat, nausea, noticed the dark color of urine. 2 weeks ago I came from the pioneer-camp, where I was a counselor. The doctor noted a condition of moderate severity. Pale, sclera subactericide. Nasal breathing is difficult. The face is puffy, there is a slight swelling of the eyelids. Oropharynx: hyperemia and swelling of the mucous membranes. The tonsils are enlarged, and they are overlaidwith uneven thickness. The cervical tissue above the enlarged submandibular nodes is pasty.

With a suspicion of diphtheria, he was sent to an infectious disease hospital. 1)Your diagnosis and its justification?

The answers to the test tasks:

1.1) Diagnosis: "Combined oropharyngeal and nasal diphtheria, toxic, III degree»

Grounds for a diagnosis - acute onset, chills, high fever, pain in the throat when GloTanya, swelling of the mucous patches on the tonsils, a challenge to the surrounding tissue, moderate HyperMIA of the mucous membrane cyanotic tinge in the zone bordering the touch, swelling of the cervical tissue to the clavicle, which appeared on the 2nd day, tachycardia, hypertension, of the film on the nasal mucosa, maceration at the nose area.

2) Urgent hospitalization in the infectious Department, administration of anti-diphtheriaserum of 120 thousand IU per week, repeat the dose after 12 hours. Detoxification therapy, corticosteroids, antibiotics.

2. 1) Diagnosis: "Combined oropharyngeal and laryngeal diphtheria, toxic, III degree, laryngeal stenosis 2-3 stages".

The basis for the diagnosis is acute onset, fever, sore throat,edema of the neck to the clavicle, widespread plaque on the oropharyngeal mucosa, edema of the soft palate, tonsils, tongue, arches, the presence of stenotic breathing, with difficulty in breathing, cyanosis, tachycardia, hypotension.

2) Administration of anti-diphtheria serum of 140 thousand IU by Bezredko, detoxification therapy, corticosteroids, antibiotics, oxygen through a nasal catheter, intravenous antispasmodics. Atthe discretion of the ENT doctor, if signs of respiratory failure increase, tracheostomy is performed.

3. 1) there are no Data for diphtheria, there are more reasons to think about infectious mononucleosis, jaundice. In favor of this, they say: the duration of the disease, the late appearance of tonsillitis, pasty face, eyelid edema, difficult nasal breathing, increased l / y, the appearance of dark urine, icteric sclera. It is necessary to re-examine the oropharynx, get the result of the study of the hemogram.

CONTROL QUESTION:

1. Brief description of the pathogen.
2. Epidemiological features of modern diphtheria.
3. What is the role of carrier in the incidence of diphtheria?
4. What pathogenetic features determine the severity of diphtheria?
5. What explains the need for early diagnosis of diphtheria?
6. Rules of examination of the oropharyngeal mucosa.
7. Clinical forms of diphtheria.
8. Early clinical signs of localized and toxic diphtheria of the oropharynx.
9. Clinical signs of diphtheria croup.
10. Complications of diphtheria.
11. Clinic of early and late myocarditis.
12. Causes of fatal outcomes in diphtheria.
13. Diseases with which it is necessary to differentiate diphtheria.
14. Methods of laboratory confirmation of diphtheria.
15. The course of diphtheria in vaccinated people.
16. Specific therapy of the disease.
17. Content of rehabilitation measures.
18. Activities in the focus of infection.

LITERATURE:

1. L. A. Favorova, N. V. Afanasieva and others. Diphtheria , Moscow, 1988.
2. N. I. Nisevich, V. F. Uchaykin. Infectious diseases in children, Moscow, 1985.
3. E. P. Shuvalova. Infectious disease. 3rd edition, Moscow, 1990.