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Department of Infectious diseases

METHODOLOGICAL GUIDE

ERYSIPELAS

for students studying in the specialty
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Historical information

Erysipelas were known to ancient physicians of India, Egypt, and other countries. In the famous papyrus of ebers, written in 3730-3710 BC, provides information about the face. Celsius (25-30 ad) distinguishes erysipelas as an independent disease and as a complication of wounds. Galen (131-21 BC) distinguished between erysipelas and phlegmon. Abu Ali Ibn Sina (Avicenna) described erysipelas in detail in the "Canon of medical science", paid attention to localized and creeping erysipelas, and dealt with issues of treatment. Of toas Sydenham described the General symptoms of erysipelas in patients with erysipelas, conducted a differential diagnosis between erysipelas and other exanthemas. At the end of the 18th century, assumptions about the contagiousness of erysipelas began to be made. Our compatriot N. I. Pirogov and an Austrian obstetrician I. Semmelweis described erysipelas in hospitals for the wounded and in maternity homes. During this period, attentionмis paid to the return of the disease - relapses. N. I. Pirogov distinguishes phlegmonous and gangrenous erysipelas. Later, the assumption of a contagious onset of the disease becomes widespread. In the last century, a number of works by Russian doctors dedicated to erysipelas were published. Cheslav Penchkovsky (1863) in his dissertation "on erysipelas in General" identifies external and internal causes of erysipelas, draws attention to the General symptoms. IvanGenisht(1864) in his dissertation "on the true erysipelas" discusses the predisposition to the disease, attaches importance to the defeat of the lymphatic system, the age of patients, resolving factors, including psychological trauma. In the 70s of the 19th century.Lukoski first drew attention to the presence of microorganisms in the skin and internal organs of patients with erysipelas. Chain cocci, isolated by L.Basterot and A.F., были названы Ф. Billroth called streptococci, he pointed out the endogenous and exogenous ways of their penetration into the body. Feleisen(1883) isolated a pure culture of streptococci from erysipelas patients, reproduced erysipelas in an experiment by injecting streptococci into 3 people and 3 rabbits. Like R. Koch, and later schotmuller, N. Koch was a great writer. According to V. V. Napalkov(1933), Yu.E. Berezov and A. I. Kulikova(1946), Feleisen believed that there was a specific erysipelas Streptococcus. Along with this, the scientists of the past century(A. See, And.eerovich et al.), as well as modern authors believe that any strain of beta-hemolytic Streptococcus from group A is capable of causing erysipelas, as well as other streptococcal infections. Data In.Lukomski and Felleisen were confirmed by Nabiqasim in thesis "Etiological studies of erysipelas". In the 80-ies of the last centuryпереоде, a monograph by G. Thielmans was published in Russian pereod, which summarizes information about the causes, mechanisms of occurrence, ways of spreading and clinical manifestations of the disease.

A great contribution to the study of the problem of faces in our time has made E. A. Gal'perin and L. V. Cherkasov.

SIGNIFICANCE OF THE TOPIC

Erysipelas is an acute infectious disease caused by hemolytic streptococci, characterized by fever, intoxication and inflammatory lesions of any serological types. These are groups of clearly defined areas of skin. The disease is also known as"St. Anthony's fire".

In recent years, erysipelas as an infectious disease has again attracted the attention of researchers and practitioners with its wide prevalence and tendency to relapse, occurring in 35-45% of patients. It causes the formation of persistent lymphostasis and acquired elephantiasis, which in turn leads to ivalidization of patients, often still at a working age. For many years erysipelas was considered a severe epidemic disease, which occurred with various and dangerous complications. The success of antibiotic therapy has led to a change in our understanding of this disease. Currently, erysipelas are considered to be one of the sporadic, low-contagious infections with a low mortality rate.

The current state of the erysipelas problem and its relevance are characterized by:

1. Significant, not significantly reduced morbidity(12-20 per 10,000 population);
2. a Pronounced tendency to develop frequent and persistent relapses;
3. The greater significance of erysipelas in the formation of secondary elephantiasis;
4. Low anti-relapse effectiveness of traditional treatment methods;
5. Serious shortcomings in the organization of medical care for this infection.

In the absence of effective methods of primary immunoprophylaxis of streptococcal infections, including erysipelas, reducing the incidence of this disease can only be achieved by reducing the frequency of its relapses. However, the development of the principles of rational anti-relapse therapy and erysipelas prevention is impossible without studying the characteristics of the course of streptococcal infection in various clinical forms of this disease.

Currently, the point of view about the streptococcal etiology of erysipelas is generally accepted, although against the background of antibiotic treatment, it is extremely rare to isolate hemolytic Streptococcus from the blood and the focus of inflammation in patients. In this connection, the hypothesis of polyethologicity of erysipelas with the leading role of Staphylococcus in its occurrence was again put forward (V. A. Proskurov). This has led to widespread and unsuccessful prescribing of anti-staphylococcal immunopreparations for the prevention and treatment of erysipelas relapses. However, from the point of view of clinicians, it seems unlikely that Staphylococcus had any serious significance in the etiology of uncomplicated forms of erysipelas. Rapid relief of acute manifestations of the disease with penicillin used in medium therapeutic doses, the extreme rarity of complications in the form of sepsis and thromboembolic processes, serous and serous-hemorrhagic nature of inflammation are absolutely uncharacteristic for extensive and deep staphylococcal skin lesions. Very rarely in recent years, the isolation of hemolytic Streptococcus in erysipelas patients is explained by both the high sensitivity of the microbe to the prescribed antibiotics, and the imperfection of the bacteriological methods used. At the same time, the use

of a complex of modern immunological studies allows not only to confirm the streptococcal etiology of erysipelas, but also to detect differences in the immunogenesis and pathogenesis of its main clinical forms.

As a result of the conducted clinical and immunological observations, it was established. That erysipelas is one of the nosological varieties of streptococcal infection, characterized by both acute and chronic course. Each of the specific clinical erysipelas is characterized by certain manifestations of immunity and allergies. Clinical and immunological characteristics of primary and repeated erysipelas, as well as late relapses of the disease, allow us to attribute these forms of the disease to time-limited acute streptococcal infections. Their occurrence occurs due to primary exogenous infection or reinfection with other serotypes of Streptococcus. Recurrent erysipelas with frequent and early relapses refers to a chronically ongoing endogenous streptococcal infection with a pronounced allergic component in the pathogenesis.

The other side of the problem is the leading role of erysipelas in the formation of persistent lymphostasis and elephantiasis, which often lead to disability of patients. In patients, a dissociated violation of the function of the adrenal cortex was detected, manifested in a change in the ratio between glucocorticoid hormones and mineralocorticoids towards the relative predominance of the latter. A simultaneous decrease in the glucocorticoid function of the adrenal cortex and an increase in the excretion of aldosterone in combination with impaired histamine and serotonin metabolism were also mainly associated with recurrent erysipelas with deep disorders of lymphcirculation . This suggests a significant significance of the identified biochemical disorders in the pathogenesis of persistent lymphostasis. The clinical effect of prednisone in the case of emerging lymphostasis in relapses of the disease was accompanied by the elimination of pronounced hyperhystaminemia in these patients. These observations are a prerequisite for the development of therapeutic methods that prevent the development of elephantiasis.

As it is known, erysipelas is an infectious-allergic disease with the presence of infectious-toxic and allergic components in its pathogenesis. The detected violations of the pituitary-adrenal system function were determined by the degree of sensitization of the patients ' body to hemolytic Streptococcus and did not depend much on the severity of intoxication in erysipelas. According to modern concepts, the pituitary-adrenal system is an important factor determining the non-specific reactivity of the body. Non-specific reactivity largely determines the outcome of the disease, including the formation of recurrent and chronic forms. In erysipelas, these phenomena are closely related; the main causal role belongs to chronic-recurrent streptococcal infection with pronounced sensitization of the body to hemolytic Streptococcus. This provision is of fundamental importance. It indicates the need for complete elimination of foci of chronic streptococcal infection in the body of patients and subsequent reinfection during the period of not yet recovered reactivity of the body, which is observed for 1-1. 5 years after recovery from erysipelas. Naturally, the traditional methods of erysipelas treatment

(sulfonamides, penicillin, physiotherapy), eliminating acute manifestations of the disease, do not prevent its subsequent relapses.

Based on the obtained results of clinical observations, immunological and biochemical studies, the infectious diseases clinic of medical faculties 1 of the Moscow medical Institute suggests two stages of measures to prevent frequent and persistent erysipelas relapses. At the first stage, complex treatment of patients during relapse of the disease is provided with the use of two-course antibiotic therapy with reserve antibiotics, biogenic stimulation and glucocorticoids (for the formation of lymphostasis) to suppress foci of chronic streptococcal infection in the skin. The second stage consists of systematic monthly administration of prolonged-acting antibiotics (bicillin-5) for 2-3 years to prevent reinfection and the development of late relapses. Billingparadise is advantageously carried out simultaneously with dispensary observation of patients and treatment of opportunistic diseases of the skin and peripheral vessels. A sharp decrease in the number of relapses of the disease in patients who received phased anti-relapse treatment allows us to recommend it for introduction into General practice. The pronounced anti-relapsing effect of the domestic drug bicillin-5, its low toxicity, and the absence of serious adverse allergic reactions make it indispensable for preventing erysipelas relapses in patients suffering from various concomitant diseases, including elephantiasis. Effective and safe means of immunoprophylaxis of erysipelas and other streptococcal infections are not expected to appear in the foreseeable future. The introduction of long-acting antibiotics will remain the only possible way to prevent their relapses for a long time to come.

Etiology

The causative agent of erysipelas is beta-hemolytic Streptococcus of group A of any serological types circulating in this area. It is a G+ microorganism that can exist in bacterial, transitional forms, and L-form. Pure culture of it was first isolated byFeleisen (882) from a patient with erysipelas. This gave rise to talk about a specific erysipelas Streptococcus. Later it became clear that similar serological types are isolated in all streptococcal infections and there is no specific erysipelas Streptococcus. Against the background of widespread use of antibiotics, it became difficult to get Streptococcus from the blood and the focus of infection. Along with this, due to the fixing role of the local inflammatory focus and secondary infection, Staphylococcus, Escherichia coli and other microorganisms began to be isolated.The idea of viral etiology of erysipelas was expressed.Despite this, streptococcal etiology is confirmed by the production of streptococcal antigens, L-forms, serological reactions. In reconvalescents -, handicaps on the site of the former focus are found both in free form and in macrophages that purify cells from bacteria. In some cases, as a result of treatment with antibiotics, as well as under the action of antibodies , lysozyme, complement component C3 and other echanises of anti - infective protection, bacterial forms of Streptococcus can be transformed into L-forms and eliminated.

Epidemiology.

Erysipelas have long ceased to be an epidemic contagious disease. There are almost no outbreaks of this infection observed in the past in maternity hospitals and surgical departments. With the exception of occasional outbreaks in psychiatric hospitals. Erysipelas can be either an exogenous or endogenous infection.

Although sporadic , erysipelas are a common disease with an incidence rate of 120-240 per 100,000 population. The source of infection is most often healthy bacterial carriers of Streptococcus , less often-individuals with various streptococcal diseases and very rarely-patients with erysipelas. The latter become a source of infection in the broken hours and days of the disease for persons with a genetically determined predisposition against the background of immunodeficiency of various nature. Newborns, pregnant women, women in labor, persons who have undergone oral surgery, patients with various variants of immunodeficiency, patients with HIV infection, hematological and oncological patients should not have contact with erysipelas in the acute period of the disease, as they are at high risk of infection.

The transmission mechanism is carried out by contact and air-drop routes. The epidemiological history of erysipelas patients differs significantly from that of other acute infections. When collecting it, it is necessary to take into account contact with patients, including any streptococcal infection(angina, scarlet fever, rheumatism, erysipelas, acute respiratory infections); genetic predisposition (erysipelas, lymph circulation disorders in close relatives); professional factors (working conditions). A General and local predisposition should be determined. The first category includes chronic tonsillitis and other streptococcal diseases. In which erysipelas occur 5-6 times more often. Local predisposition for faces represent chronic diseases of the oral cavity, carious teeth, diseases of ENT-organs in the face of the legs- lymphedema, lymphatic-venous insufficiency, edema of various origins, athlete's foot, trophic disorders; for the faces of the thorax and upper limb - surgery on mammary glands with subsequent post-operational scars, lymphedema with elephantiasis, Liferea, fistulas, non-healing wounds, etc.; for erysipelas of the genital organs - congenital or acquired elephantiasis naruchnik genitals.

Post-traumatic and postoperative scars predispose to the localization of the focus precisely in the location of their location. It should be clarified whether the patient has had erysipelas in the past, when, how many times, what is the frequency of relapses, when was the last one, where and how was he treated.

Increased susceptibility to erysipelas can be caused by prolonged use of steroid hormones. In the mechanism of erysipelas development, resolving factors are also taken into account: microtrauma, trauma, cooling, a sharp change in temperature, Psychotrauma. Working women of older age groups are more susceptible to the disease, men get sick 2 times less often, and children are very rare.

Erysipelas affects people of different professions, often builders, workers of " hot " workshops working in cold rooms, and for workers of metallurgical and coking plants, streptococcal infection becomes an occupational disease. The seasonality of

erysipelas does not coincide with the spring-autumn one, as in patients with other streptococcal infections. Patients with primary erysipelas, in which the face is more often affected, get sick in the cold season - in autumn and winter. Patients with recurrent erysipelas, which is more often localized in the area of the lower extremities, are sick mainly in warm and humid times – in summer, warm spring and autumn.

Pathogenesis

General patterns of disease development in exogenous pathways of infection in patients with primary, repeated and rarely recurrent erysipelas differ from the pathogenesis of chronic, persistently recurrent erysipelas in endogenous
way of infection. The introduction of betahemolytic Streptococcus into the skin occurs due to its damage in patients with primary erysipelas or infection from the focus of latent infection in often recurrent cases. Then the pathogen multiplies in the lymphatic capillaries of the dermis. Exo - and endotoxins enter the bloodstream, and this leads to the appearance of chills, high fever and other symptoms of intoxication. Bacteremia is short-term in nature and its significance in the development of the disease is not fully understood. The local excitatory focus is infectious and allergic in nature, it is associated with the formation of immune complexes (IR) in the dermis and papillary layer of the skin with simultaneous infiltration by lymphocytes. At периваскулярномthe perivascular location of ICS containing the third fraction of complement, they act as a trigger that causes intravascular blood clotting with violation of the integrity of the vascular wall, microthrombosis and micro-hemorrhage. This is how a local hemorrhagic syndrome is formed. In rare cases, hemorrhage in the deep layers of the skin can lead to the development of necrosis, necrotic fasciitis.

Capillaries of blood and lymphatic vessels are affected. Violation of capillary lymph circulation in the skin leads to the formation of hemorrhages and blisters with serous and hemorrhagic contents

The formation of foci of chronic streptococcal infection in the skin and regional lymph nodes with the presence of bacterial and L-forms of Streptococcus leads to the development of early erysipelas in a number of patients.

Elimination of bacterial forms of Streptococcus on the background of phagocytosis, IR formation and other protective mechanisms can end in recovery.

Erysipelas disease is caused by allergization of the body to Streptococcus and its toxins and a decrease in the level of natural resistance factors. Transferred even once, erysipelas increase the predisposition to relapse. Without an individual predisposition with autoimmune and immunocomplex reactions, the characteristic clinical picture with a non-purulent inflammatory focus will not develop. Infectious and toxic manifestations are accompanied by the action of toxins and biologically active substances, and allergic manifestations determine the development of the focus. With persistent recurrent erysipelas, a persistent focus of streptococcal infection is formed, often with L-forms of Streptococcus that persist in macrophages. They can undergo reversion to bacterial forms, while reducing the

bactericidal activity of the skin, phagocytosis and other factors of non-specific anti-infective protection. Suppression of cellular immunity is accompanied by a decrease in the total number of T –cells, subpopulations of T-lymphocytes. The decrease in humoral immunity activity indicators is characterized by a drop in the level of IgA, anti - streptococcal antibodies(ASL-O,ASG,ask), and an increase in autoimmune reactions to skin and thymus antigens.

Infectious-allergic and immunocomplex mechanisms of local inflammatory focus formation explain its non-purulent-serous or serous-hemorrhagic character. Damage to blood and lymphatic capillaries with previous inferiority of the lymph nodes can lead to sclerosis of their walls with subsequent violation of the outflow of lymph.

Metabolic disorders in the skin contribute to an increase in the activity of fibroblasts, the formation of connective tissue and lymphedema.

With repeated recurrences and subsequent violations of lymphatic drainage the process of progressive fibrosis, is formed fibregum. The functional activity of neutrophils decreases in patients with recurrent erysipelas.

Increasing the activity of cellular and humoral immunity contributes to sanogenesis. Sanogenesis is associated with the neutralization of toxins and the formation of an active phagocytic reaction in relation to Streptococcus.

Pathomorphological changes in erysipelas patients are characterized by thickening of the epidermis and dermis due to edema, infiltration of lymphoid, histiocytic elements; hemorrhagic manifestations are characterized by hemorrhagic exudate with an admixture of fibrin. When histological examination of the skin on the site of the former focus, signs of serous or serous-hemorrhagic inflammation with small-cell leukocyte infiltration of the dermis, mainly around the capillaries, are visible. In the exudate, Streptococcus and blood cell elements are detected. There are no specific morphological changes in the internal organs. Dystrophic changes are expressed moderately.

Clinical picture.

In recent years, there has been an evolution of the clinical course of erysipelas, which is characterized by an increase in the number of patients with a severe course of the disease, a sharp increase in the number of hemorrhagic forms of erysipelas, and the predominant localization of a local inflammatory focus in the lower extremities.

For correct diagnosis of erysipelas, it is necessary to use the most rational classification of the disease.

Clinical classification of erysipelas

1.*By severity:*

1) easy;
2) medium weight;
3) heavy.

2. *By the nature of the local hearth:*

1) erythematous;
2) erythematous-bullous;
3) erythematous-hemorrhagic;
4) bullous-hemorrhagic.

3.*By brevity*

1) primary;
2) repeat;
3) recurrent(early, late relapses, persistent relapsing, number of relapses)

4. *By the prevalence of local manifestations:*

1) localizable;
2) common;
   a) migrating;
   b) metastatic.

5.*Complications:*

1) local;
2) general.

6. *Effects:*

1) lymphedema;
2) secondary elephantiasis(vibraderm).

Erysipelas are characterized by the sharpest beginning, regardless of the brevity. Usually, patients indicate not only the date, but also the hour of onset of the disease. The incubation period is established only in posttraumatic erysipelas, it is 2-3 days. The initial period is accompanied by rapid development of intoxication, high fever, repeated chills, headache and muscle pain, weakness, a drop in blood PRESSURE . It is not surprising that in the first hours of the disease often are diagnosed with the flu, food poisoning, meningitis. After a few hours, and with erysipelas of the lower extremities more often on the 2nd day of the disease, a local inflammatory focus begins to form and the period of the height of the disease begins. Simultaneously with paresthesias, pain in the region of regional lymph nodes appears in the focus. Due to the irradiation of pain in the iliac region, some patients are mistakenly diagnosed with acute appendicitis and inguinal hernia.

Erythematous form is the initial stage for other forms, and as an independent form is currently rare. Erysipelas are always bright, hot and have irregular outlines . Pain in the focus is insignificant, may appear on palpation or change of position. Erythematous-hemorrhagic erysipelas are transformed from erythematous erysipelas from the end of the 2nd-beginning of the 3rd day of the disease. The transition to a bullous-hemorrhagic focus occurs from the previous ones as a result of deeper damage to the blood vessels of the retinal and papillary layers of the dermis.

In 80-90% of patients, the focus has an erythematous-hemorrhagic and bullous-hemorrhagic character. Erysipelas of the lower limb are localized in the lower leg and foot. On the inner surface of the femur extends painful levangie; isolated lesion of the thigh does not happen. The spread of the focus up, on the

torso, buttocks is observed in people with в прошлоinguinal-femoral lymph nodes removed in the body.

When the focus is localized on the face, the eye slits narrow, the focus spreads to the cheeks, forehead, eyelids, often to the auricles, the scalp.

Erysipelas of the upper extremities and chest are observed mainly in women who have undergone mastectomy, against the background of post-mastectomy lymphedema of various degrees. The focus appears on the background of intoxication, and the pore simultaneously with it, can be spotty, but always has irregular outlines. Due to the absence of axillary lymph nodes, the focus extends to the chest, torso and is hot and bright. In men, this form of erysipelas can occur as exogenous post-traumatic if safety precautions are not followed, as well as against the background of lymphedema of various origins.In rare cases in women, erysipelas of the upper limb and chest develop without a previous mastectomy, against the background неоперированнойof an unperfected breast tumor after chemotherapy or radiotherapy иерапии.

Erysipelas of the external genitalia are rare against the background of lymphedema of this area, more often in men, it occurs heavily and can be accompanied by purulent-necrotic complications. The spread of the focus on the torso is observed against the background of any forms of erysipelas. When the focus is localized in the lower and upper extremities, usually one limb is affected and there is never an isolated lesion of the hands and feet.

Complications

In erysipelas, complications occur in 3-8% of patients. Local complications may represent an abscess, phlegmon, festering hematoma, superficial necrosis, postalization bull, phlebitis, thrombophlebitis, ITS.

At the height of the disease, erysipelas in more than half of patients is accompanied by neutrophilic leukocytosis, accelerated ESR, toxic proteinuria, changes in immune status, the formation of immune complexes , hypercoagulation, impaired hemostasis , fibrinolysis, various variants of intravascular coagulation. All this matters for the development of relapses, prognosis, and differentiated treatment, and not for the diagnosis of the disease.

Differential diagnosis of erysipelas.

The widespread spread of erysipelas, the presence in its clinical course of symptoms similar to the manifestation of many other diseases, require differential diagnosis of erysipelas with more than 50 nosological forms. At the same time, the diagnosis of erysipelas is often mistaken. The greatest number of diagnostic errors is observed in the differentiation of erysipelas with surgical and skin diseases, which is explained by the lack of familiarity of doctors with the features of the clinical course of erysipelas. Every year, 40-50% of patients mistakenly referred to the infectious Department for erysipelas are diagnosed with various purulent processes (abscess, phlegmon, panaritium).

Diseases of peripheral vessels (thrombophlebitis, phlebitis, obliterating atherosclerosis) and their complications are the cause of an incorrect diagnosis of

erysipelas in 28-36% of cases. In 18-20% of patients who are not referred to the erysipelas Department, various skin diseases are detected, including dermatitis , toxicodermin, eczema, shingles and others. Significantly less often (2-3%), there is a need to differentiate erysipelas from acute arthritis of different etiologies, skin manifestations of collagenoses and diseases of the hematopoietic system.

To make a correct diagnosis of erysipelas, it is necessary to know the main diagnostic criteria for this disease. As a result of studying and analyzing case histories 3100 patients with different forms of erysipelas it was found that as the clinical diagnostic criteria ogut to be used in the following typical symptoms of the disease: acute onset of the disease (93%) with pronounced symptoms of intoxication, amid which the most constant headache (96.2 percent), General weakness (85,6%), chills(78,5%), often nausea(34,8%) and vomiting(26%); fever with a rapid temperature rise to 38.0-39,0 and above; earlier development of symptoms of intoxication and fever(for a few hours or even 1-2 days) compared to the emergence of local manifestations of the disease (62%);the preferential localization of the local process in the lower extremities(60,8%) and face (31,5%); the development characteristic of erysipelas erythema with a clear limitation from the healthy skin, rapid proliferation, infiltration, edema; regional lymphadenitis (67%);the absence of pronounced pain in inflammation alone; moderate neutrophilic leukocyte with a neutrophilic shift to the left and a moderately elevated ESR(56%)

As a result of research, it was found that in the acute period of the disease. Regardless of the severity and handicap of the local process, similar changes in the immunogram developed. The concentration of circulating immune complexes (CIC) increased by 4-6 times compared to the indicator in healthy subjects. In this period, the increase in the CEC level was mainly due to large-scale complexes. Changes in the immunoglobulin spectrum of blood did not significantly differ from the indicators of healthy people. The total and relative number of T-lymphocytes decreased. The level of B-lymphocytes changed slightly. The subpopular composition of T-lymphocytes was characterized in the acute period by a decrease in the content of" active " rosette-forming cells-ROCS and the ratio of theophylline-resistant(TFR) and theophylline-sensitive (TPH) ROCS. According to the analysis of TTMM results, suppression of the inhibitory ability of t-lymphocytes of the dermis in relation to the migration activity of macrophages has been established.

Thus, in primary erysipelas, regardless of the form and course of the disease, an immunodeficiency of various degrees of severity develops. The formation of an immunodeficiency of 2-3 degrees is of great importance in the development of relapses of the disease. Based on these data, we can conclude that it is necessary to search for adequate immunocorrective therapy schemes.

Among purulent diseases most often mistaken for erysipelas, abscesses of various localization should be indicated, especially in the period preceding the occurrence of fluctuations, in contrast to erysipelas, when the proper layer of skin (dermis) is affected, the abscess develops in the subcutaneous fat tissue, where the

cavity of the abscess is formed. The disease develops gradually. In the area of an impending abscess, the skin turns red, there is infiltration and edema. However, the site of hyperemia of the skin does not have причущихclear boundaries inherent in the erysipelas and trends to significant spread. There is a pronounced soreness on palpation of the infiltrate, especially in its center. When feeling erysipelas, soreness is usually noted on the periphery of the affected area of the skin. Pain in the lesion is observed with an abscess and at rest, especially increasing with active and passive movements of the affected limb. The rise in temperature and symptoms of intoxication are increasing, these patients gradually with the progression of suppurative inflammation.

In the initial period of phlegmon disease, difficulties may arise in the differential diagnosis of it with erysipelas. As with erysipelas, the onset of phlegmon is acute, sometimes violent, with an increase in temperature to 39-40. In the area of localization of phlegmon, there is a strong pulsating pain. There is a swelling, edema, redness of the skin. When touching soft objects
extremely dense infiltration of a spilled character is determined in the tissues, later it is softened and there is a fluctuation. Severe pain at the site of phlegmon localization on palpation and at rest, the excessively dense nature of the infiltrate allow, despite the bright erythema, to exclude the diagnosis of erysipelas.

Thrombophlebitis of subcutaneous veins begins with pain along the course of the vessels of the affected, in the limb itself, its edema. There is a limited hyperemia of the skin in the form of spots and stripes only over the affected veins. The temperature is usually subfebrile, intoxication and the phenomenon of regional lymphadenitis are absent.

Skin changes in obliterating atherosclerosis are localized in the area of the feet and distal parts of the legs, where there is a congestive hyperemia with a characteristic cyanotic hue or necrosis phenomena.

Among skin diseases, dermatitis is especially often diagnosed as erysipelas. Contact dermatitis, along with itching, redness and edema of the erysipelas, is characterized by a fairly clear restriction of the inflammatory focus. In contrast to erysipelas, on the background of an erythematous surface, patients with dermatitis have various elements-nodules, vesicles, scales, crusts. Skin infiltration is expressed slightly, there is no intoxication, regional lymphadenitis.

In toxicoderma, as in erysipelas, the occurrence of local manifestations of the disease is accompanied by a high temperature and symptoms of intoxication. The difference is that local changes in toxicoderma are very diverse. Erythema is combined with other rashes, the multiplicity of which, along with the data of the anamnesis, allows you to make a correct diagnosis.

Acute eczema with localization on the face at the first glance at the patient resembles erysipelas. With a more thorough examination, polymorphism of the lesion is revealed: against the background of hyperemia of the skin, small bubbles, eroded areas , мокнутияwetness, dry crusts are visible. There is no characteristic skin infiltration and regional lymphadenitis, temperature and intoxication for erysipelas.

Also, erysipelas often have to be differentiated from shingles. A clear limitation of the hyperemic skin area, the presence of vesicular elements, temperature and intoxication give these two diseases similar features. In contrast to erysipelas, shingles begins with pain or a burning sensation along the course of the nerve trunks, followed by the appearance of erythema and localization of it along the branches of a particular nerve.

Among the patients referred to the erysipelas Department, patients suffering from various forms of deep vasculitis are periodically detected, most often with acute nodular erythema.

Among the rarer diseases that are important for the differential diagnosis of facial erysipelas and its consequences, we should include the Rossolimo - Rosenthal syndrome.

Of the infectious diseases that occur with erythemas, only erysipelodon is misdiagnosed as erysipelas. It is usually mistaken for the skin form of the disease.

According to the literature, of other infectious diseasesдиагностики c, only the cutaneous form of anthrax requires differential diagnosis with erysipelas.

In the initial period of erysipelas, before local changes occur, but already with the developed temperature reaction and intoxication, the disease must be differentiated with influenza, food toxicoinfection, meningitis.

In patients with recurrent erysipelas, the anamnesis data and the presence of residual phenomena of previous relapses help determine the further direction of diagnostic search. The analysis of diagnostic errors showed that the reason for them in the vast majority of cases was not fully collected anamnesis. Wider acquaintance with the peculiarities of the course of erysipelas will improve the quality of differential diagnosis of these diseases, especially in the early stages of development.

Mode

In the first five days, and in the case of damage to the lower extremities - during the entire period of the disease, it is recommended to observe bed rest until the complete elimination of signs of inflammation, including regional lymphadenitis. An important component of complex therapy in patients with erysipelas is the detection of microtrauma or the determination and treatment of their disinfecting solutions -5% iodine tincture or alcohol 1% solution of diamond green. It is unacceptable to find patients in maternity hospitals and clean surgical departments.

Treatment

Most erysipelas patients are treated on an outpatient basis. Indications for hospitalization are a severe course of the disease with severe intoxication, a common focus, hemorrhagic forms, recurrent nature of the disease, childhood and senile age, the presence of severe concomitant diseases.

Treatment of patients with primary, recurrent erysipelas and rare relapses is also carried out in hospital settings. In primary erysipelas, penicillin is prescribed for 1,000,000 UNITS 6 times a day intramuscularly for 7-10 days, at the same time-desensitizing agents, ascorutin, physiotherapy. In case of recurrent erysipelas, two courses of antibiotics are indicated. Firstцефалоспоринового, a first-or

second-generation cephalosporin antibiotic is prescribed at a minimum dose of 1 g 3 times a day intramuscularly for 7-10 days against the background of desensitizing drugs , askarutin, physiotherapy and other pathogenetic agents. For the second course, lincomycin is used 0.5 g 5 times a day orally or 0.6 g 3 times a day intramuscularly for 7 days.

When relapsing erysipelas in parallel with etiotropic therapy, non-specific non-steroidal anti-inflammatory drugs are used: chlotazole, butadion, metindol in normal doses. In case of recurrent erysipelas, non-specific stimulators and immunomodulators are also indicated иммуноодуляторы: T-activin, нуклеинатsodium nucleinate, splenin, glyceram, methyluracil, interferon preparations.

Reasonable appointment of immunomodulators for persistent recurrent erysipelas is desirable after determining the immune status of each patient.

Due to the predominance of hemorrhagic forms of erysipelas with the activation of the procoagulant link of hemostasis, the use of direct-acting anticoagulant heparinis indicated - its introduction by electrophoresis from the cathode of 5000 UNITS in 15-20 ml of isotonic sodium chloride solution at a current of 10-15 A; the duration of the procedure is 15-20 minutes daily for 5-6 days. At the same time, trental is prescribed for 0.2-0.4 g 3 times a day.

Taking into account that staphylococci can play a certain role in the pathogenesis of erysipelas. As well as mixed strepto-staphylococcal microflora, all reconvalescents after the end of antibiotictherapy should be injected subcutaneously with staphylococcal anatoxin 0.5 ml or staphylococcal antifagin.

ДизентоксикационнаяDysentic erysipelas therapy is aimed at reducing the toxic effecton the body of products of vital activity and the breakdown of microbes, as well as tissues caused by beta-hemolytic streptococci of group A. in severe erysipelas, parenteral dysentic therapy is carried out using an infusion of crystalloid solutionsRinger, Labori, glucose(5% solution) with the addition of 5-10 ml of 5% ascorbic acid solution, 60-90 mg of prednisone. Given the increased hydrophilicity of tissues in the focus of inflammation and the phenomenon of secondary hyperaldosteronism in the acute period of the disease, it is necessary to strictly monitor and keep track of the amount of injected fluid and indicators of daily diuresis. In severe edematous syndrome and hyperexudation, diuretics are prescribed (2 ml of 1% solution of lasix)

Substances that reduce vascular wall permeability. If there is a local hemorrhagic syndrome, measures are taken taking into account the state of hemostasis and fibrinolysis . With clearly expressed hypercoagulation phenomena, the use of direct-acting anticoagulants heparin 5000 U is indicated. The drug is administered subcutaneously or by electrophoresis. The duration of the procedure is 15-20 ins daily for 5-6 days. In the absence of pronounced hypercoagulation, it is recommended to introduce protease inhibitors -kontrikala and gordoxa to the focus of inflammation by 100,000 UNITS, the course of treatment is 5-6 days. If there is a pronounced activation of fibrinolysis in the early stages of the disease, it

is advisable to treat with a fibrinolysis inhibitor amben at a dose of 0.25 g 3 times a day for 5-6 days.

Infectious-toxic shock occurs in response to a massive intake of microbial toxins into the blood against the background of a decrease or perversion of humoral and cellular protective reactions of the body. This phenomenon is observed in the case of massive death of pathogens caused by etiotropic therapy with bactericidal drugs. Intensive therapy should be aimed at stabilizing hemodynamic and metabolic disorders. As an infusion agent, crystalloid and colloidal solutions are used alternately. Initially use 0.9% sodium chloride solution, lacteal, kvartasol. Subsequently, colloidal solutions are prescribed that facilitate the movement of fluid from the interstitial to the intravascular space. самыThus, interstitial edema , hypovolemia, blood thickeningкровт, and non-specific detoxification are reduced. In order to reduce metabolic acidosis, administration of 300-400 ml of 5% sodium bicarbonate solution is indicated. To stabilize the cell membranes, reduce the inflammatory response and intoxication, glucocorticoid and antihistamine agents are prescribed.

In the early stages of shock, when disseminated blood clotting prevails, intravenous heparin is administered at first simultaneously, and then dropwise for 5-10 thousand ED under the control of blood clotting time.

When acute heart failure occurs, leading to hemodynamic pulmonary edema, treatment measures are aimed at relieving the small circle of blood circulation by reducing blood flow to the right ventricle. For this purpose, peripheral vasodilators –nitrates, as well as diuretics of rapid action-1% solution of lasix-6 l intravenously jet. Limit the introduction of fluid, the patient is given a semi-sitting position, temporary imposition of flagella on the extremities. To improve the contractile function of the myocardium, cardiac glycosides are prescribed in combination with Panangin (10 ml intravenously). Elimination of hypoxemia is achieved by constant inhalation of moistened oxygen using nasal catheters. With severe vascular insufficiency, large doses of prednisone are prescribed-up to 300-400 mg /day intravenously and rheopolyglucin 200 ml intravenously. When the blood pressure drops, resort to using 1 ml of 1% solution of mezaton with 250 l of 5 % glucose solution intravenously or 0.5 l of mezaton in 20 ml of 20% glucose solution intravenously.

Nonsteroidal anti-inflammatory drugs.

Patients with severe skin infiltration at the site of the lesion, which persists in the period of convalescence, as well as with relapses of the disease, are shown the use of non-steroidal anti-inflammatory drugs: acetylsalicylic acid , indomethacin, butadion, diclofenac. Nonsteroidal anti-inflammatory drugs have a pronounced anti-inflammatory, analgesic and antipyretic effect.

Local therapy

Treatment of local manifestations of erysipelas is carried out only in the bullous form of the disease with localization of the process on the extremities. Patients erythematous form of erysipelas do not require local application of medicines. The use of fat-based dosage forms is contraindicated. In the presence of intact blisters, they are incised at one of the edges and after evacuation of the exudate, a dressing

with a 0.1% solution of rivanol or 0.02% solution of furacilin is applied to the focus of inflammation, changing it several times during the day. The duration of application of bandages - no more than 3-5 days, it is unacceptable to use tight bandaging. With limited erythematous manifestations in patients with a light course of primary erysipelas, treatment with cold is possible. The cryotherapy technique consists of daily short-term freezing of the surface layers of inflamed skin with a jet of chloroethyl until побеленияthe skin whitens.

In the presence of extensive wet erosions on the site of opened blisters, local treatment begins with the use of baths of 0.1-0.5% aqueous solution of potassium permanganate for the extremities, followed by the application of bandages. In local hemorrhagic syndrome (with erythematous-hemorrhagic erysipelas), 5-10% liniment dibunol is prescribed in the form of applications to the area of the focus of inflammation (2 times a day for 5-7 days)

Physiotherapy

Physiotherapy measures are prescribed after normalization of temperature. Erythematous doses of ultraviolet rays are applied to the affected focus and areas of healthy skin. When maintaining a period of convalescence, skin infiltration, edematous syndrome of revionic lymphadenitis, prescribe applications of ozokerite or bandages with warmed naphthalan ointment - for the defeat of the lower extremities and paraffin applications-for the inflammatory process on the face. To improve blood circulation in the lower extremities , lidase electrophoresis, hyaluronlidase(for a course of treatment of 10-120 sessions), magnetotherapy, radon baths are used. These procedures are indicated for patients with the initial stage of formation of elephantiasis.

A relatively high efficiency of low-intensity laser therapy(in the red or infrared range) of the local focus of inflammation, especially in hemorrhagic forms of erysipelas, has been proved. The dose of laser radiation varies depending on the state of the local hemorrhagic focus, the presence of concomitant diseases.

Rules of discharge

Convalescents after erysipelas can be prescribed after full clinical recovery, but not earlier than 7 days of normal temperature

Prophylactic medical examination

Reconstructors after primary erysipelas are registered by the doctor of the office of infectious diseases for 3 months, with the involvement of doctors of other specialties, if necessary, from those who have suffered a relapsing form of the disease –for at least 2 years.

Prevention

One of the main aspects of the problem of erysipelas as a streptococcal infection is the tendency of the disease to chronically relapse - 25-35 % of all cases. In more than 70% of patients, recurrent erysipelas often occur against the background of concomitant conditions. Accompanied by disturbances of trophicity of the skin, reducing its barrier function and exacerbation of chronically occurring

diseases(chronic focal infection of ENT organs, odontogenic infection, and others) prevention faces provides the prevention of minor wounds, fungal infections of the skin, hypothermia, as well as prevention of somatic diseases, contributing to the formation of immunodeficiency.
Billingparadise faces indicated for patients with recurrent erysipelas,and also reconvalescents after erysipelas of the number of people in older age groups with severe diseases of the cardiovascular system and kidneys, metabolism(diabetes, obesity), disorders of blood circulation in the lower limbs(lymphedema, elephantiasis-hybridoma, chronic venous insufficiency), leading to deterioration of trophic tissue, and ulcers.

Bicillinoprophylaxis begins one month before the start of an unfavorable season(1.5 million UNITS of bicillin 5 or retarpen 2.4 million UNITS are administered intramuscularly for 3-4 months each month). With frequent relapses faces that are not associated with the season recommended continuous billingparadise for 2-3 years. In the presence of significant residual phenomena after erysipelas, the drug is administered at intervals of 4 weeks for 4-6 months.

Security questions about the topic

1. Characteristic of the etiological factor-hemolytic Streptococcus
2. Significance of associative microorganisms in the recurrent course of erysipelas
3. Epidemiological significance of various sources of infection
4. Mechanism of development of General toxic syndrome
5. Mechanism of development of local toxicoallergic process
6. Characteristics of various clinical forms
7. Etiotropic treatment
8. Significance of specific and non-specific therapy
9. Possibility of using staphylococcal anatoxin
10. Prevention of erysipelas.

Control tasks

Task #1.

A local doctor was called to a 63-year-old patient. Ill for the second day. The night before, I felt weak, chills, headache, slight pain in the muscles of my arms and legs, and my lower back. At night, these phenomena intensified. Temperature 38.2. I Noticed that in the area of the parvous temple there was a reddening of the skin, which within a few hours spread to the cheek, eyelids,forehead, and ear. At night, I slept poorly, my body temperature rose to 39 C, nausea appeared, and I vomited twice.When examined, the condition is moderate. On the right side of the face, an area of erythema covering the upper part of the cheek, eyelids , forehead,temporal and parietal areas, and the auricle. The right eye slit is closed due to edema of the eyelid. The erythema zone is painful on palpation, hot to the touch. Its edges are not smooth, clear, rise above the surface of the surrounding skin. The nasolabial triangle is pale. Palpated

enlarged, painful lymph nodes, more on the right. Heart tones-rhythmic, pulse 112 BPM, AD110 / 60 mm HG. There is vesicular respiration in the lungs. BH-19 per minute. The tongue is moist, slightly covered with a damp coating. The oropharyngeal mucosa is not changed. The abdomen is soft, painless. The liver and spleen are not enlarged. There is no dysuria. There are no meningeal symptoms.

1. Make a diagnosis
2. Are there indications for hospitalization?
3. Treatment plan.

Response to the control issue:

1. Acute onset of the disease, the presence of symptoms of intoxication and erythema zones that appeared somewhat later on the face with clear edges rising above the surrounding skin, hot to the touch and moderately painful palpation, regional lymphadenitis indicate erythematous facial skin. The severity of symptoms of intoxication corresponds to the average severity of the disease.The erythema zone has a limited spread on the face. Previously, the patient did not have erysipelas. Consequently, the patient has an erythematous erysipelas of the face, moderate severity, primary. Severe edema of the eyelids, narrowing of the eye slits is associated with the spread of inflammation to the eyelid area.
2. Etiotropic therapy: oxacillin 1.0 gram / m, after six hours, amoxicillin 1.0 gram after 8 hours inside for 7 days.Lincomycin 1.0 gram after 8 hours inside up to 7 days. Pathogenetic therapy of excessive drinking, askorutin. Physiotherapy – in the acute period of UF,UHF, subsequently with non-receding edema application of ozokerite, calcium chloride electrophoresis.

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