ФГБОУ ВО «Казанский государственный медицинский университет» МЗ РФ
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Erysipelas

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Definition of the Disease

- Erysipelas – human infectious disease of streptococcal etiology, with acute and chronic forms and is characterised by intoxication syndrome and local changes looking like circumscribed locus of serous hemorrhagic inflammation of skin (rarely mucosa)

Etiologic Peculiarities

- 1. In primary and recurring erysipelas with exogenic route of transmission the cause of infection is beta-haemolytic streptococcus group A with its aggression factors:

  = antigenous substrates T-, R-, M- proteins (M-protein and associated antigens – lipoteichoic acid, opalescence factor lipoproteinase,polysaccaride peptidoglicane etc)

  = extracellular substrates (exotoxins or locally applicable toxins: erythrogenous toxin A, B, C, streptolysine O, streptolysine S, streptokinase, hyaluronidase, proteinase)

Etiologic Peculiarities (continued)

- 2. In relapsing and particularly frequently relapsing erysipelas the etiological cause are the L-forms of streptococcus, which form after primary erysipelas as a result of inadequate etiotropic therapy.

- 3. At the high point of disease from the local focus of inflammation different opportunistic species are released (staphylococcus, hemolysing E.coli, proteus, Ps.aeruginosa etc). This indicates the opportunistic flora activation in patients with immunodeficiency (often secondary).

Laboratory diagnosis

- 1. Detection of antigenemia:
  = A-polysaccaride (A-PSC)
  = protein-ribosomal antigens (PR-Ag)
  = L-form antigens

2. Detection of antibodies:
= AB to A-PSC in ELISA
= AB to O-SL (ASL-O)
= AB to DNA-ase

Epidemiological peculiarities

- 1. In primary, recurrent, rarely relapsing erysipelas the source of infection are patients with different forms of streptococcal infection: tonsillitis, scarlet fever, streptodermia; healthy carriers. Patients with erysipelas are not very contagious, but theoretically the transmission is possible.
- The main route of transmission – percutaneous (through the defects in skin and mucosa). Aerogenic mechanism of streptococcus transmission has a certain significance with primary infection of nasopharynx and subsequent lymphogenous and hematogenous dissemination to the local focus (mostly in erysipelas of the skin).
- So, these forms are acute cyclic infectious process developing as a result of exogenous infection and the incubation period can be counted about 24-48 hours.

Epidemiological peculiarities (continued)

- 2. In frequently relapsing erysipelas - only endogenous mechanism of infection – reversion of the L-forms of streptococcus, that persist in the scar tissue, along small blood and lymphatic vessels, lymph nodes, bone marrow. That's why FRE is considered as chronic form. In this form of erysipelas the incubation period can be counted according to provocative factors: hypothermia, hyperthermia, insolation, emotional stress, hurts, blunt trauma etc.

Epidemiological peculiarities (continued)

- 3. Vulnerability depends on basic immune status condition and the virulence of the bacteria.
- 4. Season is generally summer-autumn, but relapsing forms have no typical season.
- 5. After infection with streptococcus the disease develops only in those who have congenital or acquired predilection. Women get sick more often than men, especially the relapsing form.
- 6. Most of the patients are 40-60 years old and older.

Pathogenesis

- 1. Permeation of the streptococcus into the skin.
- 2. Reproduction of bacteria in the lymphatic capillaries of derma.
- 3.The toxins of the streptococcus get into the blood stream (toxemia).
- 4. Forming of the inflammatory locus.
- 5. Forming of the locuses of chronic streptococcal infection (providing relapses of the disease)

OR

Elimination of vegetative forms of streptococcus with phagocytosis and other immune mechanisms (recovery).

Pathogenic features of frequently relapsing erysipelas:

- 1. Forming of the resistant locus of streptococcal infection in the body (L-forms)
- 2. Dramatic decrease of phagocytosis and bactericidic activity of the skin
- 3. Depression of cellular immunity: decrease of the T-cells, CD4+, CD8+ subpopulations
- 4. Decrease of humoral immunity: low level of immunoglobulines class A and anti-streptococcal antibodies (ASL-O, ASG, ASK) in the serum
- 5. Extremely high degree of allergisation to streptococcus
- 6. Autoimmune reactions against skin and thymus antigens
- 7. Disbalance in hormonal regulation: deficiency of glucocorticoids and redundancy of mineralcorticoids (increase of edema)
- 8. Stable alterations of lymph- and blood circulation with the development of disseminated microthrombosis (DIC syndrome)

Predisposing factors

- 1. Concomitant diseases – plantar mycosis, diabetes mellitus, obesity, chronic venous insufficiency, lymphostasis, trophic ulcers, eczema etc.
- 2. Professional factor – jobs connected with constant dirtying and microtraumatization of the skin, wearing rubber shoes etc.
- 3. Locuses of chronic streptococcal infection as tonsillitis, sinusitis, caries (erysipelas of the face), osteomielitis, thrombophlebitis, ulcers (erysipelas of lower extremities) etc.

There are two main components in the pathogenesis of erysipelas:

- 1. Infectious-toxic (toxins, transient bacteriemia, secretion of biologically active substances), causing fever and intoxication;

- 2. Infectious-allergic, responsible for the local inflammation

Clinical classification of erysipelas

- 1. By frequency:
  = Primary
  = Recurrent
  = Relapsing

  2. By the character of local changes:
  = Erythematous
  = Erythematous-bullous
  = Erythematous-hemorrhagic
  = Bullous-hemorrhagic

Clinical classification of erysipelas (continued)

- 3. By severity:
  = mild
  = moderate
  = severe

  4. By localisation:
  = lower extremities (55-60% - PE, 75-80% - RR)
  = face (25-30%)
  = upper extremities, trunk (5-12%)

Examples of the clinical diagnosis

- 1. Primary erysipelas of the left shank erythematous form moderate severity.

- 2. Relapsing erysipelas (1st early relapse) of the right shank and foot bullous-hemorrhagic severe form

  Complication: phlegmon of the right shank soft tissues.

Evolution of the erysipelas clinical features

- 1. More older people (60 year old and older) – 55,8%
- 2. More lower extremities involvement – 66,5% less frequent face involvement – 25%
- 3. More relapsing forms – up to 45-50% of all cases
- 4. More patients with hemorrhagic manifestations from 10-12% to 43,8%
- 5.More frequent allergic reactions on antibiotics, sulphanilamids and other drugs, especially among patients with relapsing form.

Clinical features of erysipelas

- 1. Acute onset of the disease.
- 2. Intoxication syndrome is usually ahead of other symptoms for practically 18-24 hours and is characterised by high fever, chills, headache, sometimes nausea, vomiting, myalgias, arthralgias.

  Patients complain of malaise, weakness, body pain, sleep problem, loss of appetite.

Clinical features of erysipelas (continued)

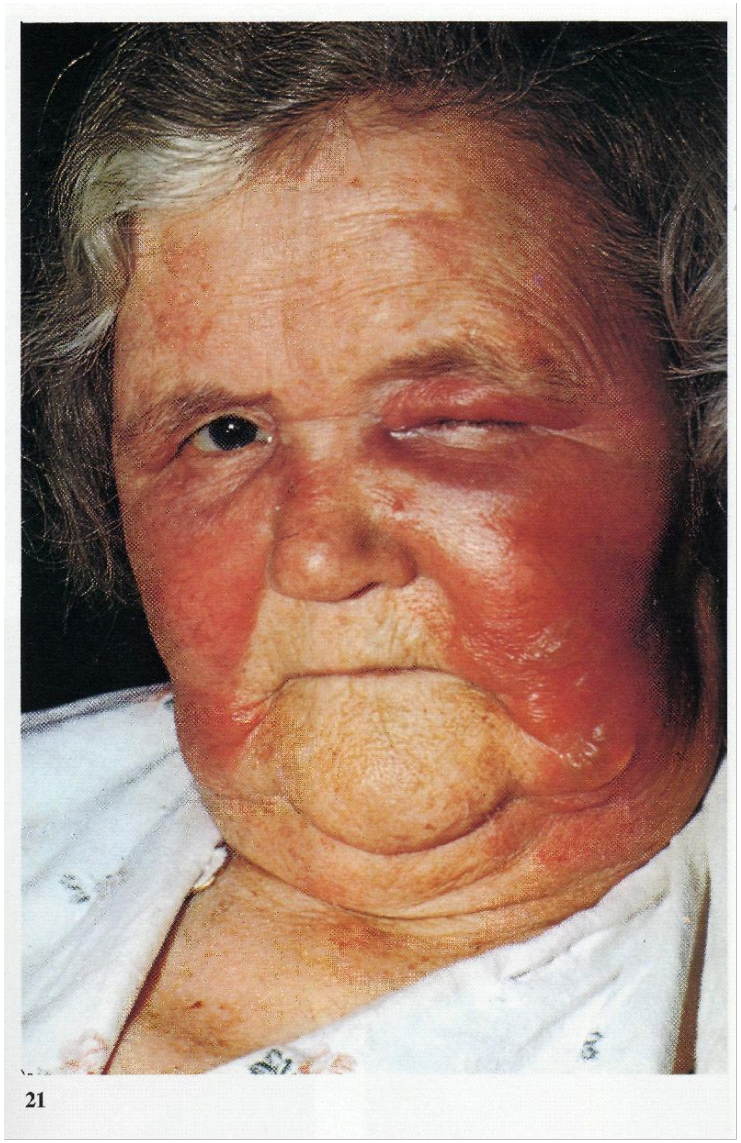
- 3. Early signs of the disease before the local changes can be:
- a) regional lymphadenitis and lymphangitis (in lower extremities erysipelas) characterized by pains in the projection of regional lymphnodes (especially inguinal) and along the lymphatic vessels (medial side of the thigh),
- b) burning pain in erysipelas of the face that starts 5-6 hours before the local inflammatory focus forms.

Clinical features of erysipelas (continued)

- 3. Local process is characterised by sharply circumscribed hyperemia with the peripheral inflammatory wall, edge painfullness, local temperature reaction (erythematous form)
- On the background of hyperemia appearance of other elements is possible:
- = hemorrhagias (erythematous-hemorrhagic form),
- = bullas – bubbles filled with serous fluid (erythematous-bullous form),
- = bubbles filled with serous-hemorrhagic fluid (bullous-hemorrhagic form).

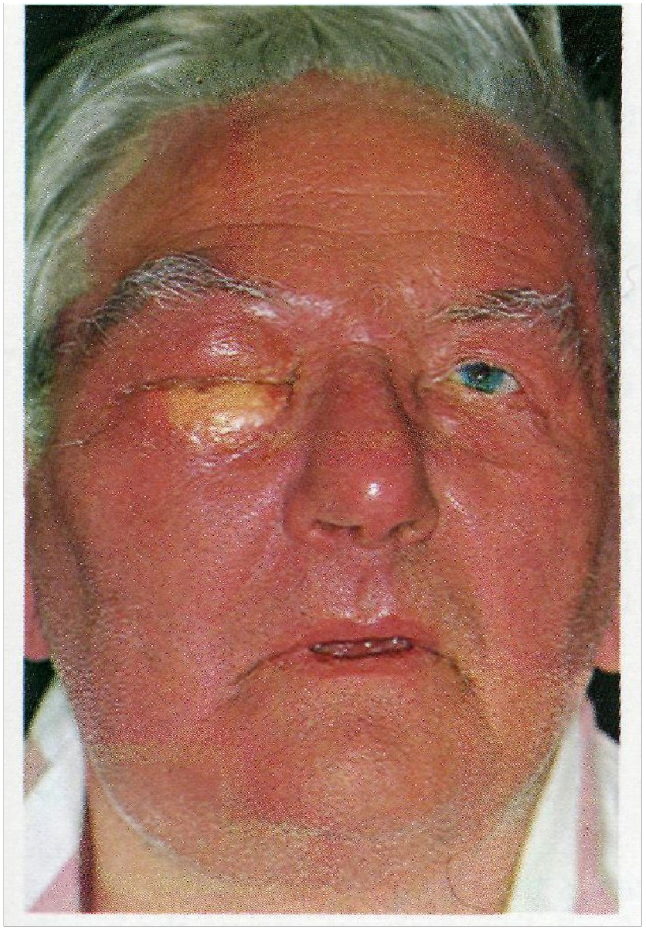
Clinical features of erysipelas (continued)

- 4. Local process is associated with lymphatic edema of various degree depending on the character of local process.
- 5. In case of paired organ involvement usually unilateral process.
- 6. On the face is typically limited on the border of hairy part of the head.

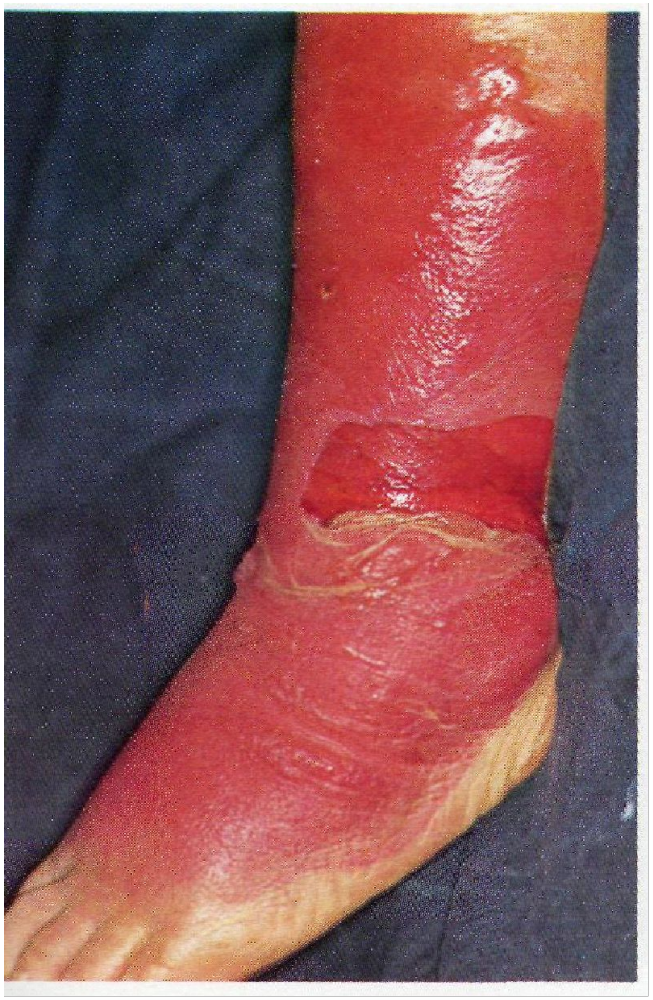


Erysipelas of the face (EB form)

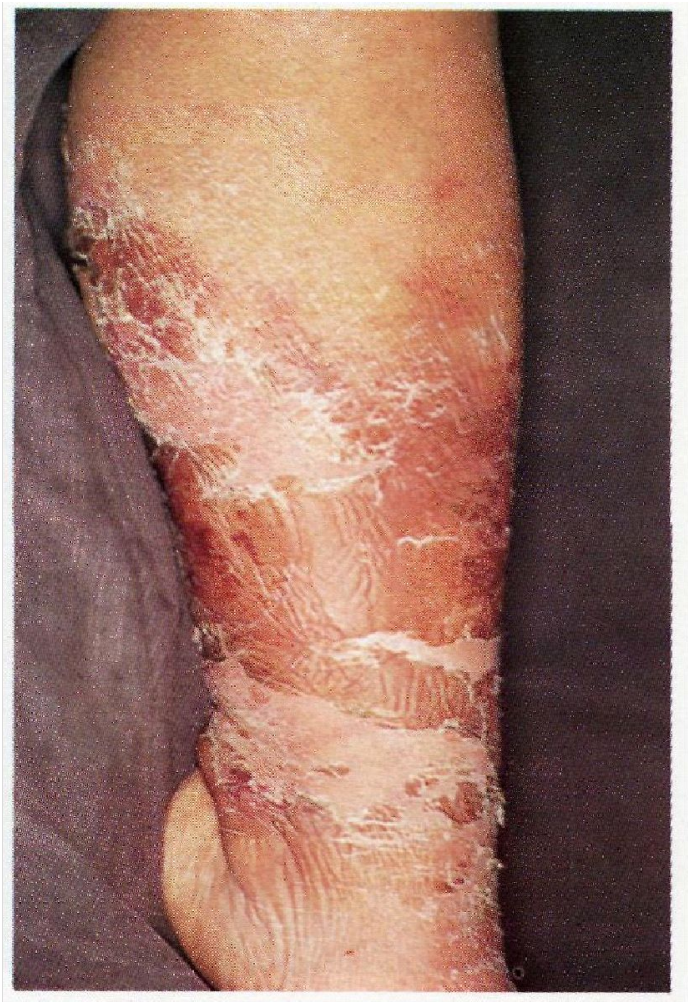
After recovery (peeling)



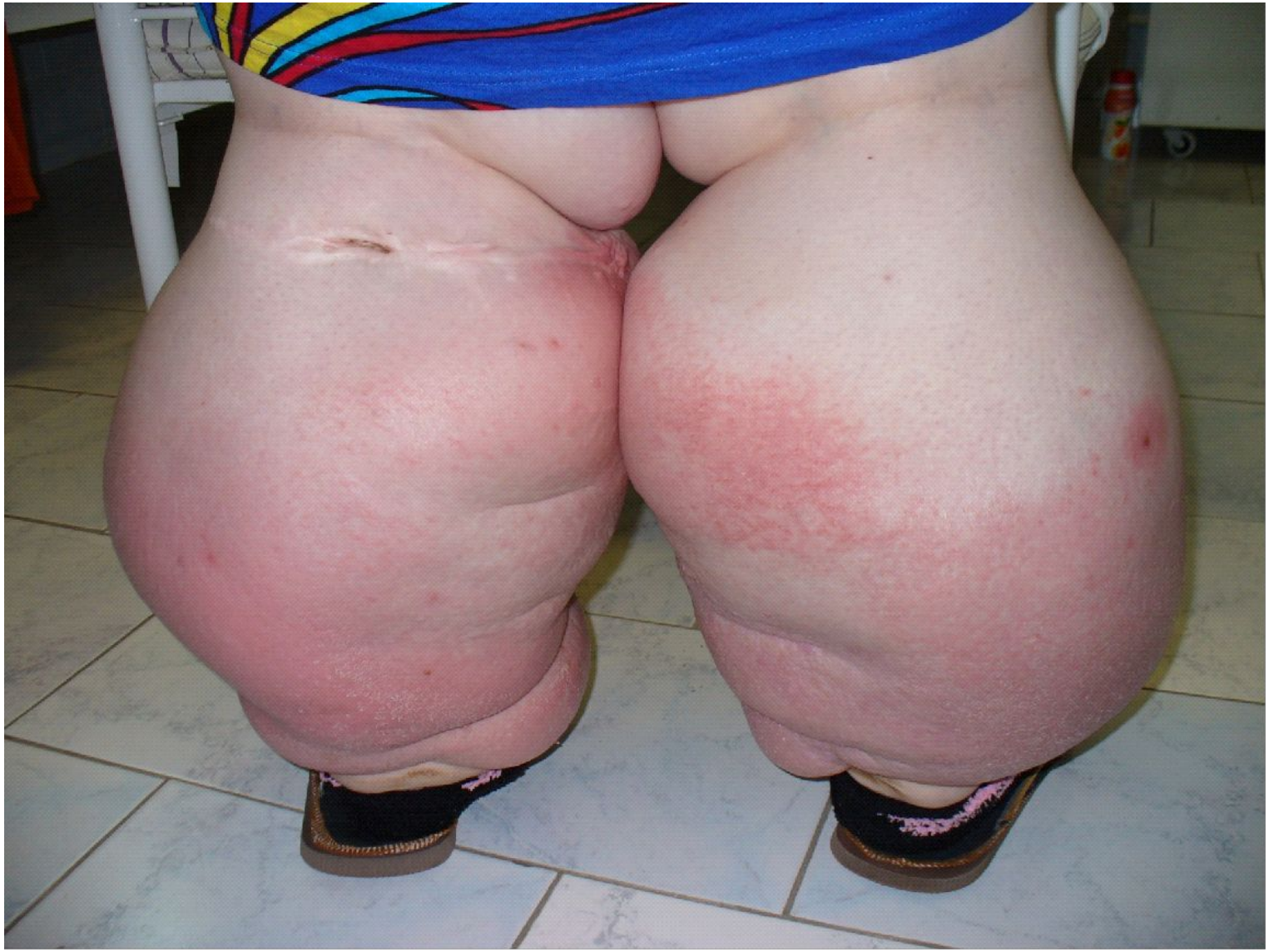
Erysipelas of the face (Erythem. form)



Erythematous-bullous form



Pigmentation and peeling (recovery)



Elephantiasis (outcome)

Diagnosis

- Main method – clinical and anamnesis.
- Differential diagnosis:
- With infectious diseases (skin form of anthrax, erysipeloid etc)
- With dermatologic diseases (streptodermia, staphylodermia, allergic dermatitis etc)
- With surgical diseases (abscess, phlegmon, acute and relapse of chronic thrombophlebitis etc)

Ethiotropic therapy

- 1. In primary and recurrent erysipelas penicillin is the antibiotic of choice – 5-6 mln Un in 24 hours IM, mild forms – 7 days, moderate forms – 10 days, severe forms – 12-14 days.
- 2. In relapsing erysipelas – semisynthetic penicillins (ampicillin, oxacillin, ampiox, amoxicillin, augmentin etc) - 4 g in 24 hours.
- 3. In frequently relapsing erysipelas – antibiotics of choice are cephalosporines (1-4 generations) 2-4 g in 24 hours; linkomicin 1,2-2,4 g in 24 hours.
- (in persistent relapses – 2 course treatment)
- 4. For out-patients – macrolides (spiramicin 6 mln IU/24 hours), tetracyclines (doxycycline 0,2 g./24 hours)

Pathogenetic therapy

- 1. Detoxication therapy – oral (enterodez, regidron etc.); parenteral – crystalloids (polyionic solutions: trisol, acesol, chlosol, kvartasol, 5% glucose etc.), low- and medimolecular colloids (reopolyglukine, reogluman, reomakrodex etc) counted 1:1
- 2. Desensibilisation therapy – antihistamine drugs (dimedrol, suprastin, pipolfen, tavegil, claritin etc)
- 3. Correction of the hemostasis alterations according to the coagulogram control : desaggregants – dicinon, trental, kurantil; dimephosphone etc.; direct acting anticoagulants - heparin, fraxiparin, calciparin etc, indirect acting – sinkumar, kumadin, pelentan etc.

Pathogenetic therapy (continued)

- 4. Immunocorrection with the control of the immune status (immunoglobulines IV and IM, interferones, thymal drugs – timalin, timogen; pirimidines – methyluracil, natrium nucleinate, ksimedon; dimephosphone; bacterial polysaccarides – pirogenal, prodigiosan; herbal adaptogens – eleuterococcus, ginseng, aralia and others.
- 5. Physiotherapy in acute period - UV in suberythemal doses №5 and UHF №5; projectional distant exposure of low intensive laser, in reconvalescence period – potassium iodide, lidase, ronidase electrophoresis; indirect action – laser puncture

Local treatment

- 1. Do not touch (!) erythematous forms.
- 2. Vishnevsky ointment and ichtiolic ointment are strictly forbidden.

- 3. Is only recommended for bullous forms in 2 steps:
- 1 step – applications with antiseptic solutions (furacillin 1:1000, rivanol 1:1000, chlorfillipt, dimexid, 15% dimephosphone etc.)
- 2 step – emulsions and ointments (lanolin cream, sea buckthorn oil, 10% methyluracilic ointment, aecol etc.)

Ambulatory monitoring

- 1. Finishing treatment
- 2. Sanation of the chronic focuses of infection
- 3. Relapse prophylaxis:
- Bicillin-5 1,5 mln. Un IM once a month –
- In relapsing forms – every month for at least 2 years since the last relapse;
- In primary, recurrent forms – for 6 months after the disease.

Thank you for attention!