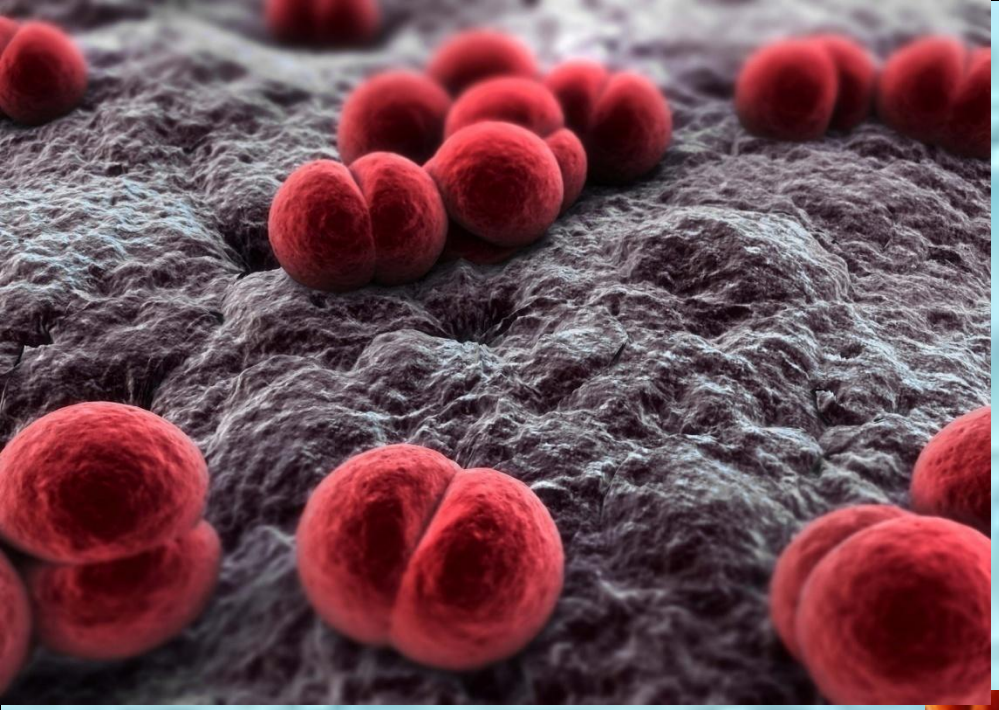
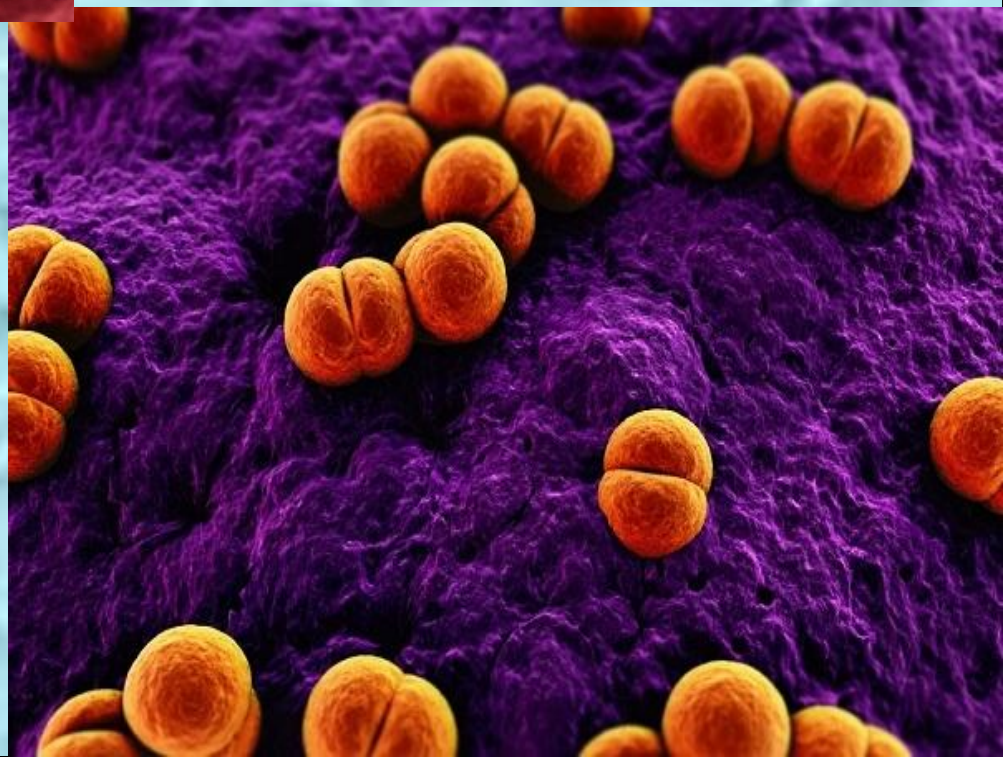


Meningeal syndrome in clinic of infectious diseases

Meningococcal infection



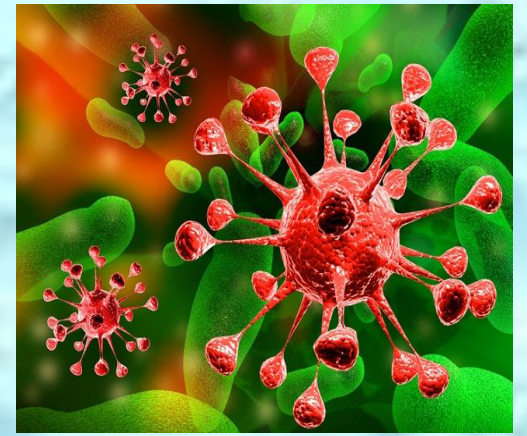
MENINGITIS - inflammation of meninges of brain and spinal cord. Acute meningitis plays a leading role in infectious pathology of the central nervous system. The majority of researchers propose to classify meningitis by the following criteria:

By appearance:

- **Primary** - occurs without any preceding infection or local inflammation as an independent disease
- **Secondary** – occurs as a complication of general or local infectious process;

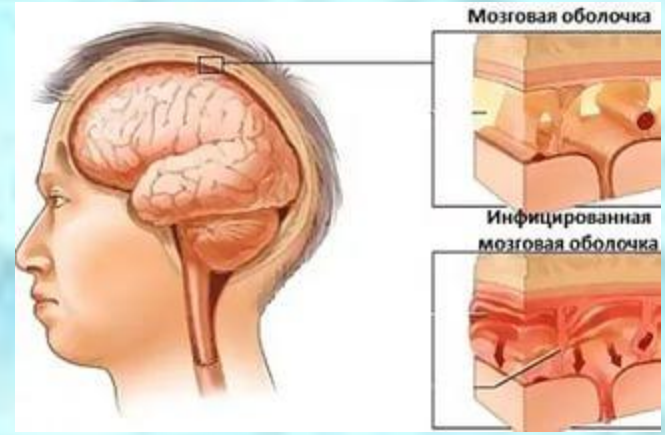
According to etiology:

- Bacterial
- Viral
- Fungal
- Spirochetosis
- Rickettsial
- Protozoal
- Helminthic
- Combined



By character of inflammation and changes in the cerebrospinal fluid:

- Serous;
- Purulent;

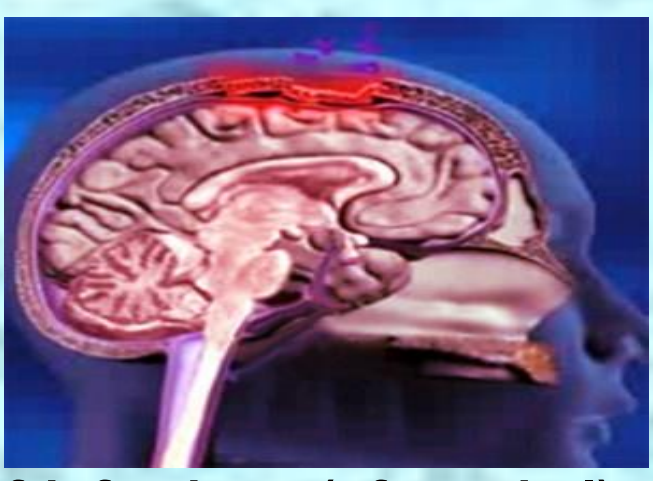


By duration of the course:

- Acute;
- Lingering;
- Chronic;

By severity:

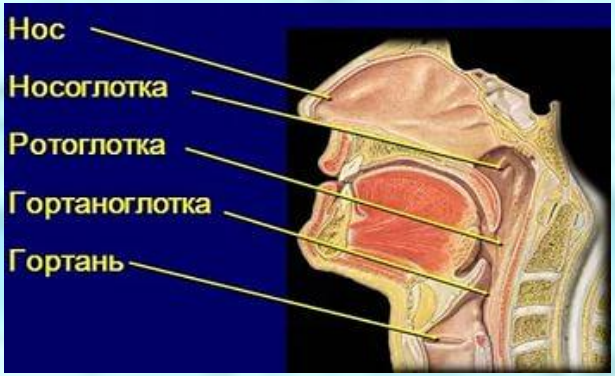
- Mild;
- Moderate;
- Severe;



ENCEPHALITIS – polietiological disease of infectious (often viral) or infectious-allergic genesis, characterized by inflammation of the brain and (or) spinal cord (myelitis), accompanied with cerebral and encephalitic syndrome, increased intracranial pressure and inflammatory changes in CSF.

PATHOGENESIS

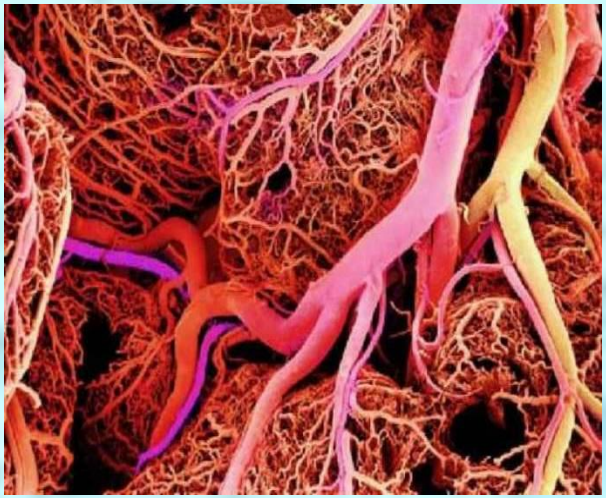
The entrance gates for causative agent can be:



- nasopharynx,
- respiratory tract,
- intestine,
- primary focal inflammation;

Dissemination of the pathogen occurs by:

- hematogenous,
- lymphogenous,
- contact way.



3 pathogenetic factors play an important role in the development of the disease:

- bacteremia,
- toxemia,
- macroorganism (initial immunity)

- Destruction of bacteria,
- releasing of endotoxins,
- development of toxemia,
- increased levels of biologically active substances,
-products of disturbed metabolism

Leads to

- increased permeability of cellular and vascular membranes, blood-brain barrier;
- active penetration of bacteria and toxins in the CNS with development of the pathological process.

- irritation of the vascular plexus of brain ventricles (by bacteria, antigens, toxins and immune complexes);
- increase of cerebrospinal fluid production and intracranial pressure.

Irritation of structures of the diencephalic region (centers of thermoregulation, vomiting and vascular) causes the main clinical manifestations of the disease (headache, repeated vomiting, meningeal symptoms).

The complex of symptoms of acute meningitis consists of 4 major syndromes:

- **general intoxication,**
- **cerebral sd,**
- **meningeal syndrome,**
- **sd of inflammatory changes in the cerebrospinal fluid.**

- General intoxication:

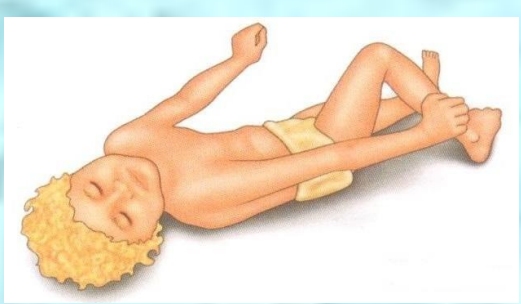
- **acute onset with fever (febrile),**
- **pale skin,**
- **chills, lethargy, weakness,**
- **anorexia,**
- **tachypnea, dyspnea,**
- **tachycardia, muffled tones of the heart, changing heart rate, unstable blood pressure.**

Cerebral syndrome:

- headache – strong, diffused, sometimes local, increases when changing body position (due to high intracranial pressure and irritation of pain receptors by pathogen toxins);

- vomiting - has a central mechanism (due to a direct irritation of the vomiting center), repeated, occurs suddenly, without previous nausea, does not bring relief, increases when change body position;

- possible disturbance of consciousness (from agitation to somnolence, stupor and coma);



- seizures (from twitching of some muscles to generalized convulsions and convulsive status);

- expansion of veins on the head, eyelids and eyes.

Meningeal syndrome - develops due to irritation of cerebral baroreceptors by high liquor pressure or viral/bacterial toxins.
Several clinical signs facilitate the diagnosis of meningitis
There are 4 main groups of symptoms:
1-st group - the symptoms of **tonic muscle tension**:

- **rigidity of occipital muscles** - passive flexing the patient's neck (chin to chest) is difficult or impossible (the severity is estimated by the distance between the head and surface);

- **Kernig's-sign** - flexing the patient's hip 90 degrees then extending the patient's knee causes pain;
- **Brudzinski's-sign** - flexing the patient's neck causes flexion of the patient's hips and knees;

Both of these signs are thought to be caused by the irritation of motor nerve roots passing through inflamed meninges as the roots are brought under tension.

- Brudzinski's contralateral reflex sign – has two components: the identical and reciprocal contralateral reflex.

1)The patient's hip and knee are passively flexed on one side; if the contralateral leg bends in reflex, identical contralateral reflex is demonstrated.

2)Reciprocal contralateral reflex occurs when the leg that has flexed in response to the passive flexion of the contralateral hip and knee begins to extend passively.

- Guillain- symptom;
- Gordon-symptom (when squeezing the gastrocnemius muscle flexes the big toe);

- the position of the patient - head thrown back, torso stretched, hands pressed to his chest, legs bent and given to the abdomen ("meningeal posture", "posture of setter dog", " trigger-pose").

2-d group – symptoms of general hyperesthesia and hypersensitivity of the sensory organs (photophobia, hyperacusia) due to irritation of the posterior roots, cells of the spinal nodes, receptors of meninges, and dicreased level of sensitivity to various stimuli.

3-d group - reactive pain phenomena:

- Bekchterev-symptom;
- Lobzin-symptom (painful grimace when pressing on the eyeballs through closed eyelids);
- Kerer‘s symptom (pain in points of the trigeminal nerve);
- Pulatov-symptom (tenderness to percussion of the skull);
- Mendel's symptom (pain when pressure on the frontal wall of the external ear canal).

4-th group – changed dermal, pariosteal and tendon reflexes:

- Babinsky-reflex;
- Oppenheim-reflex;
- Gordon's reflex (pinching the gastrocnemius muscle);
- Sheffer’s reflex (compression of the Achilles tendon);
- Rossolimo-reflex.

Syndrome of inflammatory changes in the cerebrospinal fluid

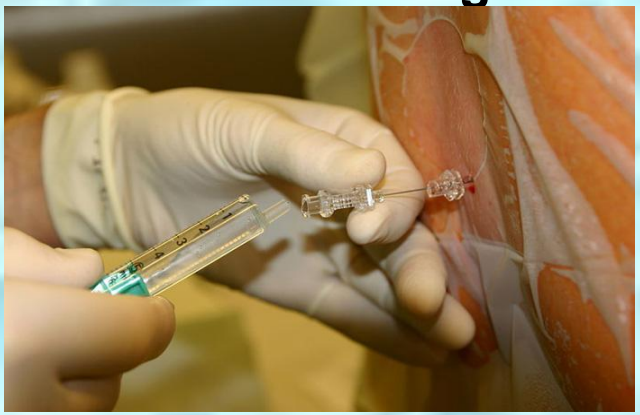
The investigation of CSF can establish the diagnosis of meningitis, its character, intensity and dynamics of the process, the effectiveness of treatment, course of illness and recovery.

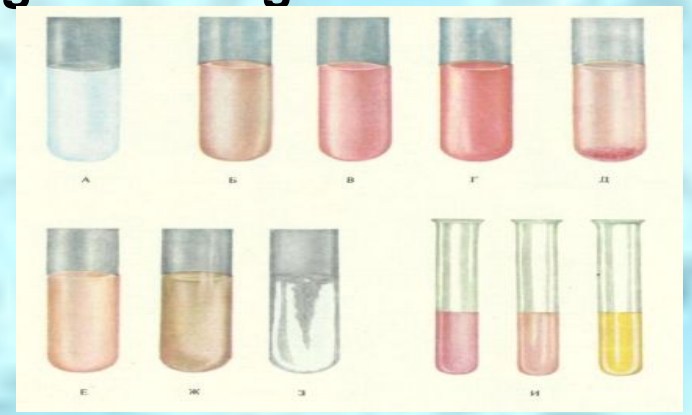
Etiologic diagnosis of CNS infections is very important, because the process is temporary and its effects can be dangerous for the patient's life.

Many meningitis may be treatable due to early diagnostics and adequate treatment.

Leading role in the diagnosis of meningitis belongs to investigation of cerebrospinal fluid.

There are 3 types of liquor - A, B,C
according to the pathological changes





CSF can be purulent, serous, serous-fibrinous, hemorrhagic and xanthochromic.

Inflammation of meninges is accompanied by the following changes of CSF:

- **Increased pressure - cerebrospinal fluid is flowing streamly or by frequent drops.**

- **Change in transparency (cloudy) or color (white, yellow-green etc.) depending on the etiology of the process.**

- **Pleocytosis with a predominance of neutrophils, lymphocytes, or mixed.**

- **Increase the protein content.**

- **Change in the level of glucose and chlorides.**

Type A – turbid, mononuclear cells < 50, polymorphic(90%) - 1000 – 10000/l, protein - 0.5 – 2 g/l, glucose < ½ of glucose in blood.

Characteristic for:

a) bacterial meningitis caused by: Meningicoccus, Haemophilus, Streptococcus pneumonia, Listeria;

b) perforated abscess of the brain;

C) amoebic meningoencephalitis;

Type B – slightly turbid or transparent, sticky, mononuclear cells - 100 – 300/l, polymorphic 0 – 200/l , protein 0.5 – 3 g/l, glucose < 1/2 of the blood glucose;

Typical for:

granulomatous meningitis – tuberculous, mycotic;

Type C - transparent or slightly turbid, mononuclear cells - 10 – 100-1000/l, polymorphic - 0, protein - 0.4 - 0.8 g/l, glucose >1/2 of the blood glucose;

Characteristic for:

a)parameningeal infection;

b)toxic encephalopathy;

c)viral infection;

Differential-diagnostic signs of meningitis by CSF

SIGN	NORMAL	MENINGISM
COLOR TRANSPARENCY	COLORLESS TRANSPARENT	COLORLESS TRANSPARENT
PRESSURE (DROPS/MIN)	40-60	60-90
CYTOSIS (IN MKL)	2-10	2-12
CYTOGRAM (%) RATIO OF LYMPHOCYTES AND NEUTROPHILS	L=80-85 N=3-5	L=80-85 N=3-5
GLUCOSE (MMOL/L)	2,2-3,3	2,2-3,3
PROTEIN (G/L)	0,22-0,33	0,22-0,45
FIBRIN FILM	-	-
REACTION TO PUNCTURE	HEADACHES	RELIEF

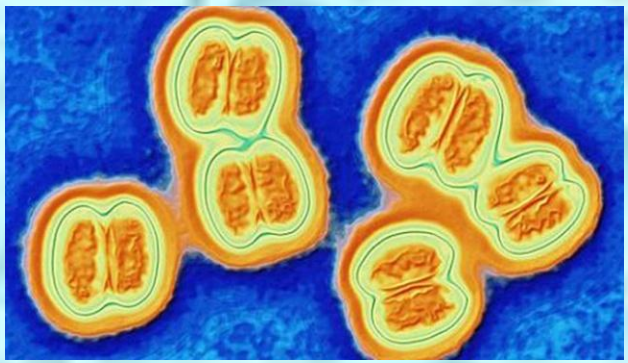
Differential-diagnostic signs of meningitis by CSF

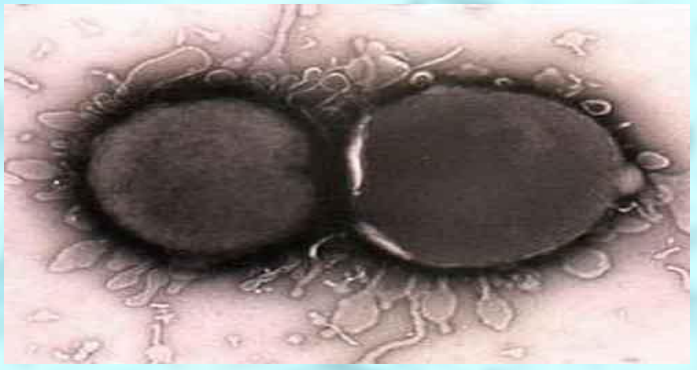
SIGN	SEROUS-VIRAL	SEROUS-BACTERIAL
COLOR TRANSPARENCY	COLORLESS ,TRANSPARENT, OPALESCENT	COLORLESS, OPALESCENT, XANTHOCHROMIC
PRESSURE (DROPS/MIN)	80-120	STREAM
CYTOSIS (IN MKL)	20-800	200-700
CYTOGRAM (%) RATIO OF LYMPHOCYTES AND NEUTROPHILS	L=80-100 N=0-20	L=40-60 N=20-50
GLUCOSE (MMOL/L)	0,55-0,66	DICREASED ON THE 2-d WEEK OF DISEASE
PROTEIN (G/L)	0,16-1,0	1,0-3,3
FIBRIN FILM	IN 3-5%	IN 30-40%
REACTION TO PUNCTURE	EXPRESSED RELIEF, OFTEN THE TURNING POINT OF THE DISEASE	EXPRESSED SHORT-TERM RELIEF

Differential-diagnostic signs of meningitis by CSF

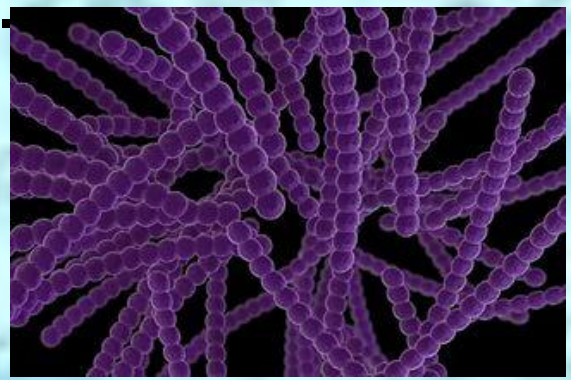
SIGN	PURULENT	SUBARACHNOID HEMORRHAGE
COLOR TRANSPARENCY	WHITISH, YELLOW, GREENISH, TURBID	BLOODY XANTHOCHMORE ROMIC
PRESSURE (DROPS/MIN)	STREAM	MORE 80 OR STREAM
CYTOSIS (IN MKL)	1000-20 000 AND MORE	FROM THE 5-7th DAY100-500 PREVAIL RBC
CYTOGRAM (%) RATIO OF LYMPHOCYTES AND NEUTROPHILS	L=0-40 N=60-100	FROM THE 5-7-th DAY PREVAIL LYMPHOCYTES
GLUCOSE (MMOL/L)	SIGNIFICANTLY DICREASED	NORMAL
PROTEIN (G/L)	0,66-16,0	INCREASED
FIBRIN FILM	ROUGH AS SEDIMENT AT THE BOTTOM OF THE TUBE	SELDOM
REACTION TO PUNCTURE	MODERATE SHORT-TERM RELIF	EXPRESSED RELIF

Primary purulent meningitis is of meningococcal, pneumococcal, Haemophilus influenzae etiology.





Secondary purulent meningitis developed as a complication of sepsis, diseases of ENT-organs (otitis, mastoiditis, mastoiditis, sinusitis), lungs, kidneys and other organs and is most often caused by staphylococci, streptococci, Pseudomonas aeruginosa etc., Fungi, protozoa, helminths are rare pathogens.



Diagnostic criteria of primary purulent meningitis

Meningococcal infection – is acute respiratory infection, antroponosis, with aerogenic mechanism of transmission, pathogenically is characterized by the destruction of respiratory epithelium, clinically - intoxication, rhinopharyngitis, affection of CNS and sepsis-like state.

- **Anamnesis**

– winter-spring seasonality (strong seasonality now is not seen);

- route of transmission is airborne droplets, realized through close contact (within 2 hours at a distance of 30 – 50 cm);

- source of infection - is sick of any form of meningococcal disease patient or carrier;

- risk group - children under 1 year, the elderly , persons with chronic pathology of RT, ENT-organs, immunosuppression;

- frequency of incidence is 8-10 years.

EPIDEMIOLOGY - anthroponosis

Source: Epidemiological danger

- patients with generalised forms of disease
- patients with a nasopharyngitis
- carriers of N.m.

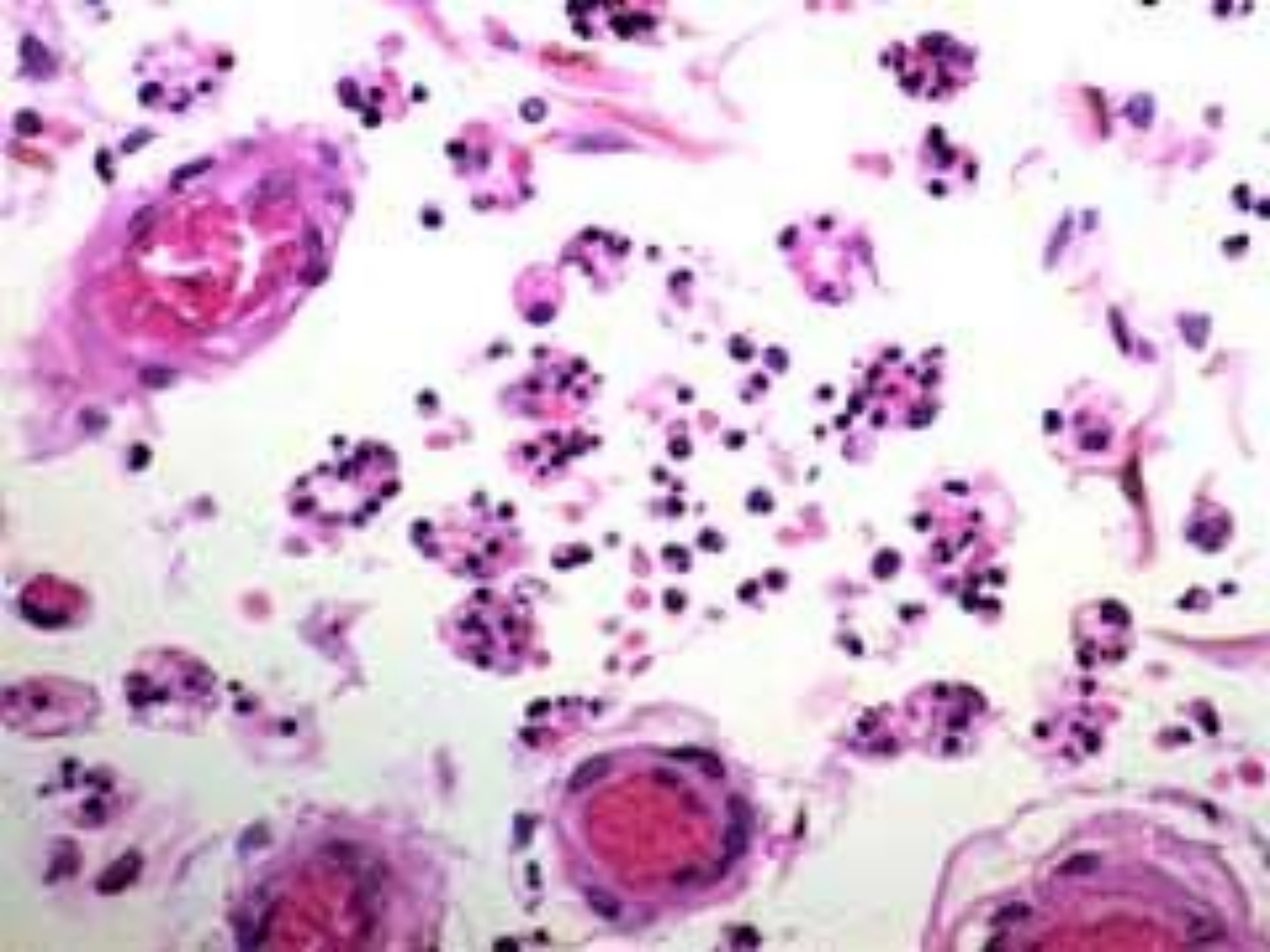
Duration of infectious period 3 - 4 weeks.

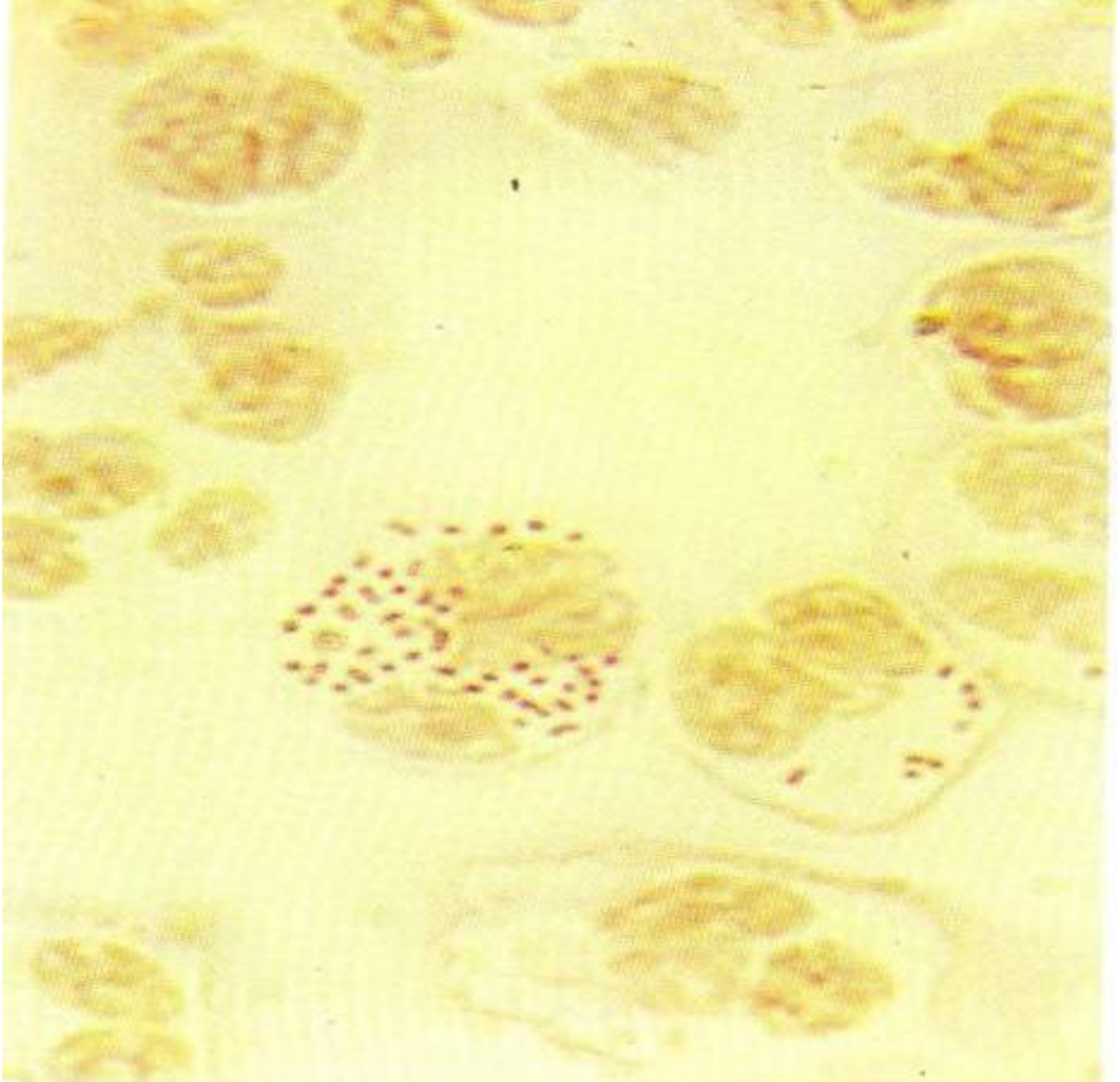
Patients are INFECTIOUS - during a prodromal and acute phases of disease!!!

- **Mortality in meningitis is 3-5%,**
- **meningococcemia - to 20%.**

Causative agent - Neisseria meningitidis

- Small gram-negative diplococci;
- typical location in a pair of coffee beans;
- do not form spores, the capsule is nonconstant;
- nonmotile;
- aerobic, obligate intracellular microbe;
- grows on artificial nutrient media (blood agar);
- for capsular AG allocate nine serogroups (A, B, C, D, X, Y, Z, W135 and E);
- releases endotoxin;
- produces hyaluronidase and neuraminidase;
- secretes IgA-proteases that cleave the IgA molecule that protects the bacteria from Ig;
- nonstable in environment;
- sensitive to drying and cooling, to penicillin, tetracycline, erythromycin and disinfectants





PATHOGENESIS - disease develops in 3 stages:

- PENETRATION - the local forms of the diseases are developed as nasopharyngitis or pneumonia or carrier-state;

- BACTERIEMIA - infiltration N.m. in a blood, following the lysis of bacteria and endotoximia - disease proceeds as acute sepsis with hemorrhagic rush and hemorrhagic sd;

-DISTRIBUTION - into the brain by hematogenous, lymphogenous way, perineuraly and through ethmoidal bone;

Serum antibodies and high concentration of IgA on the mucous of upper RT - play an important role in protection from N.m.!!!

PATHOMORPHOLOGY

- the N.m. causes acute inflammatory response in a place of implantation (statified pavement epithelium).

Endotoximia results of a diffuse vasculitis and DIC - (disseminated intravascular coagulopathy)

Vessels are filled by clots of blood with a major contents of a fibrin and leucocytes, that results in hemorrhages on the body, but on a skin they are accompanied with necrosis at the centre of large eruptions.

The cause of a generalisation of the process is not clear. N.m. “love" well-nourished children and adults »- probably influence of the genetic factors and inadequate response of the organism on implantation of the N.m.!!!

CLASSIFICATION

Localized forms:

- Carriage –without clinical manifestation, can be revealed by detection of N.m. in culture and smears from a nasopharynx;
- Nasopharyngitis;
- Meningococcal pneumonia;

Generalized forms:

- Meningococcal sepsis (acute and chronic)
- Meningitis
- Meningoencephalitis
- Mixed (sepsis + meningitis and etc.)
- Infrequent forms of disease: endocarditis, arthritis, pneumonia, iridocyclitis, otitis etc.)

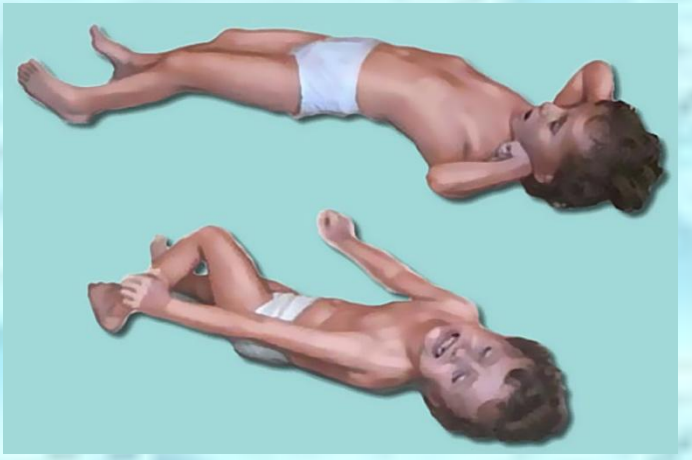
CLINIC

1. Acute onset;
2. Rise of body temperature (to 39-40ºC),
3. Expressed intoxication,
4. Starts with nasopharyngitis,

- Characteristic of cerebral sd:

- headache, repeated vomiting, not bringing relief,

- agitation, insomnia or drowsiness, delirium, hallucinations, in severe cases - generalized convulsions, disturbance of consciousness up to coma.



• **Meningeal syndrome** appears in the first days of the disease of different severity:

- often occurs with development of **edema-swelling** of brain (the attacks of psychomotor agitation);

-1-1,5% of the patients revealed signs of **encephalitis** (paralysis of the facial muscles paraparesis, paralysis, disorder of coordination);

- **affection of cranial nerves**;

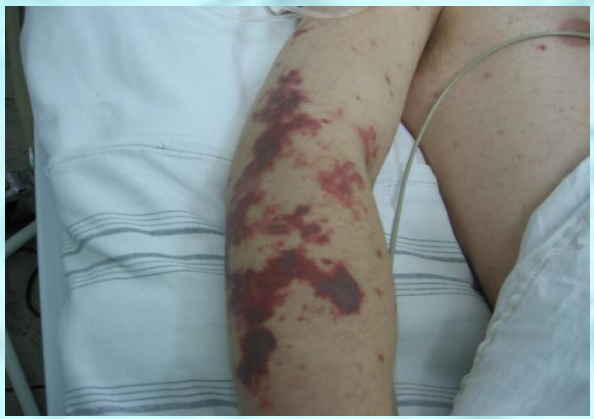
- visual and auditory **hallucinations**, euphoria or depression;

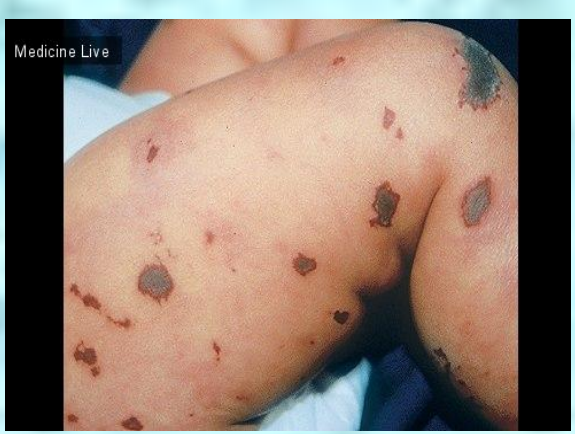
- 30-40% cases of meningitis is combined with **Meningococcemia:**

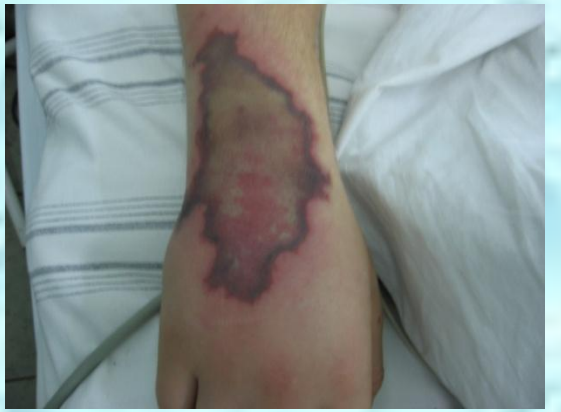
-hemorrhagic rash on the skin with petechial character, sometimes up to 5-15 cm in diameter with necrosis in the center, localized on the buttocks, lower limbs,

-infectious-toxic shock,

- serious complication - is the development of hemorrhages in the adrenal glands (syndrome Waterhouse – Friderichsen),
- heart (endocarditis, pericarditis, myocarditis) and other organs.







• **CBC:** leukocytosis, neutrophilia, shift of the formula to myelocytes, lymphopenia, increased ESR.

• **CSF:**

-is cloudy, yellow-colored,
- flows streamly,
- protein is increased to 1g/l,
- count of neutrophils - more than 1,000 cells in 1 ml,
- in the first hours of the disease can be seen only increased CSF pressure or signs of serous meningitis,

• **Microscopy of CSF,** thick drops of blood or discharging from the nasopharynx can be used to detect gram-negative diplococci, located intracellular.

- **Bacteriological examination** of mucus from the nasopharynx, blood, cerebrospinal fluid is leading in the diagnosis of meningococcal disease.

Material for bac. exam. should be taking before etiotropic therapy;

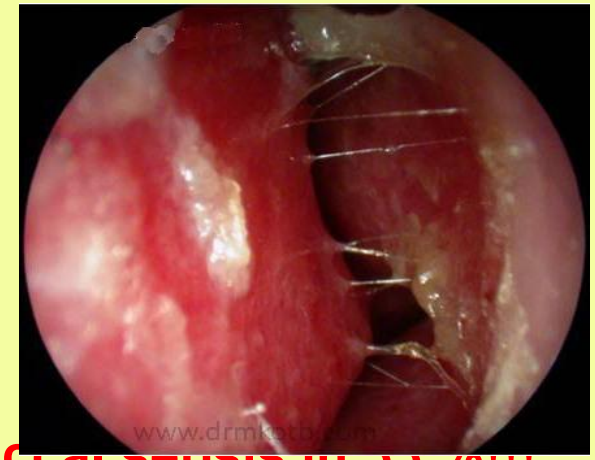
- **Serology** (RIHA) carried out with diagnostics of meningococci of group A and C (conditionally diagnostic titer of antibodies in adults 1:40-1:80).

In severe meningococcal infection and infectious-toxic shock antibodies are detected in low titers, and in some cases not detected at all.

NASOPHARYNGITIS - clinical manifestations:

Moderate parietofrontal headache - 52 %
Malaise/fatigue - 46 %
Dry cough - 66 %
Pharyngalgia - 51 %
Stuffiness of a nose - 68 %

Desorded sleep - 33 %
Fever - 59 % (Subfebrile - 78 % and febrile - 22%)



Mild severity of disease - 88 - 95 %
Moderate current - 5 - 12 %

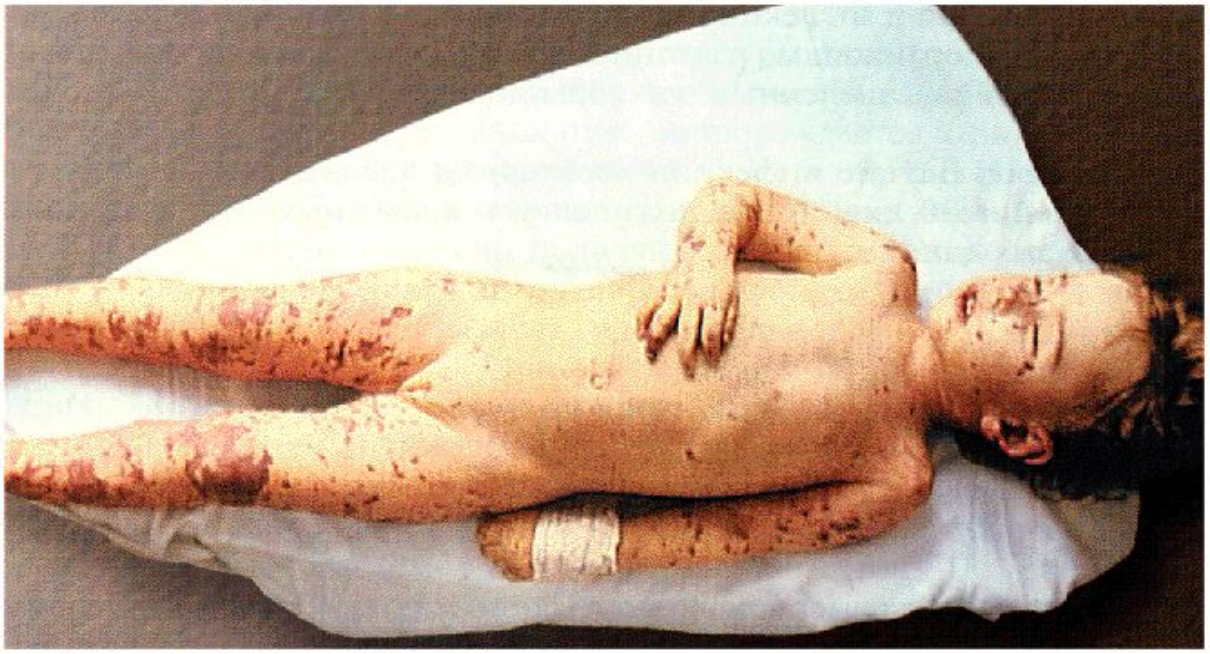
General duration of illness - 5 - 7 days

The nasopharyngitis precedes the meningococcal sepsis in 55 %!!!

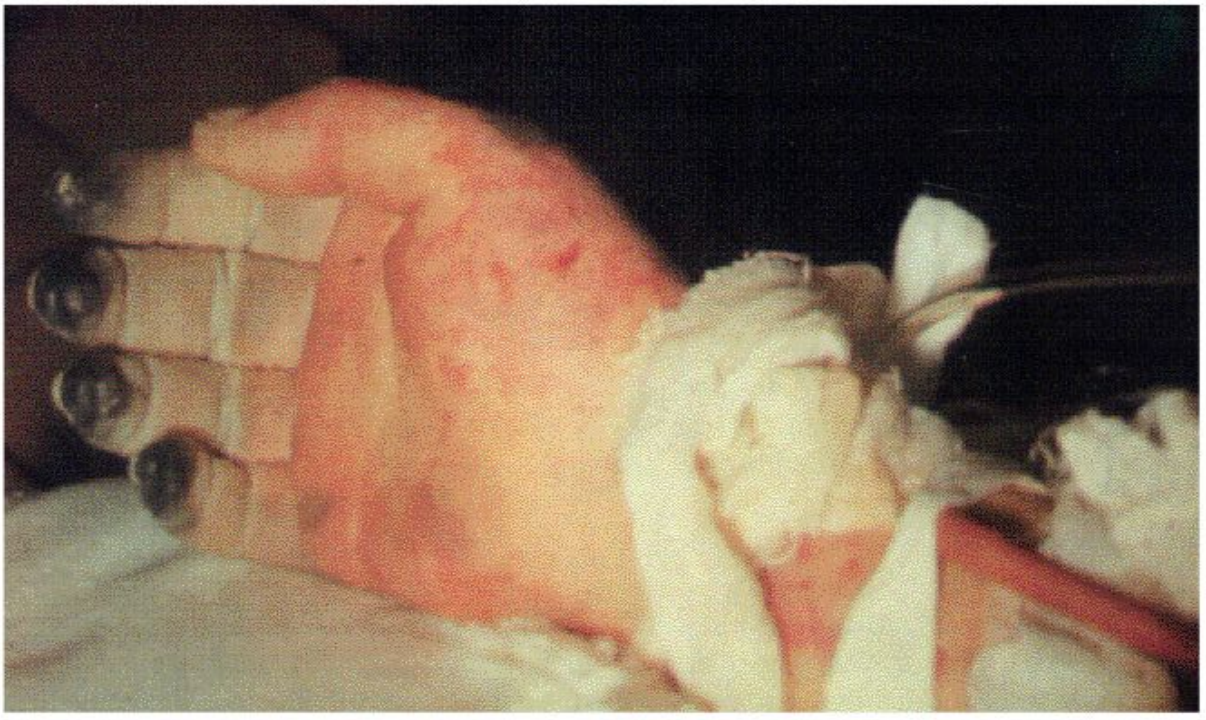
-

MENINGOCOCCAL SEPSIS:

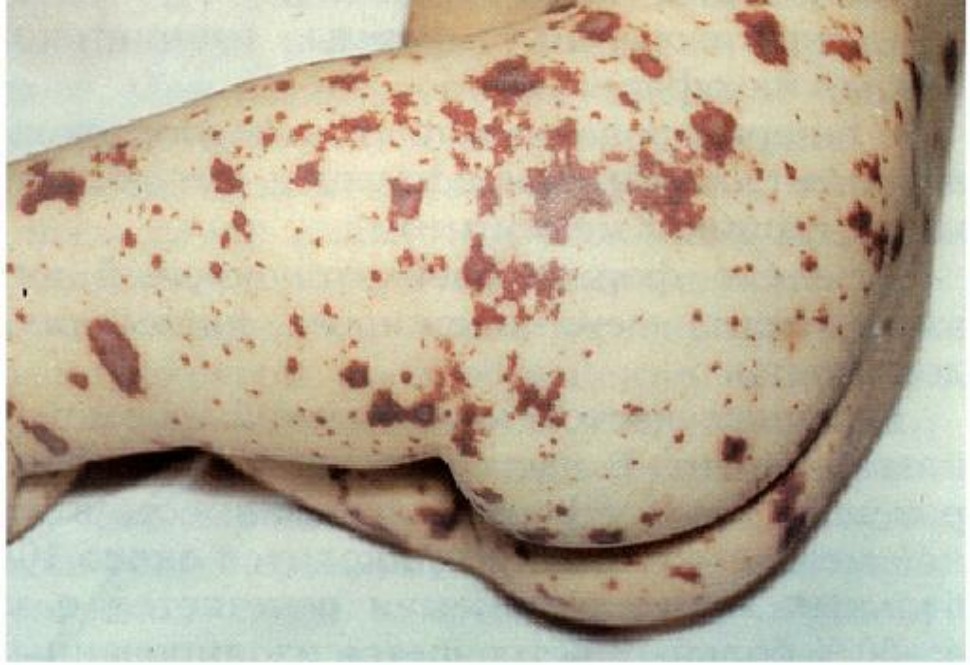
- acute beginning with chill and fast rise of intermittent temperature up to 38 - 40ºC
- expressed intoxication (headache, thirst, weakness, paleness and dryness of a skin)
- appearance of hemorrhagic syndrome: hemorrhagic rush with necrosis on the skin of buttocks, legs, trunk, arms, eyelids (in 2-4-6 hours from the onset of disease!!!)
- enanthema in a transitive folds of conjuctiva, hemorrhages on sclera
- the hemorrhages in joints - on 5 - 13 % are more often than in fingers and legs
- common manifestations of a hemorrhagic syndrome - nasal, uterine, internal bleeding

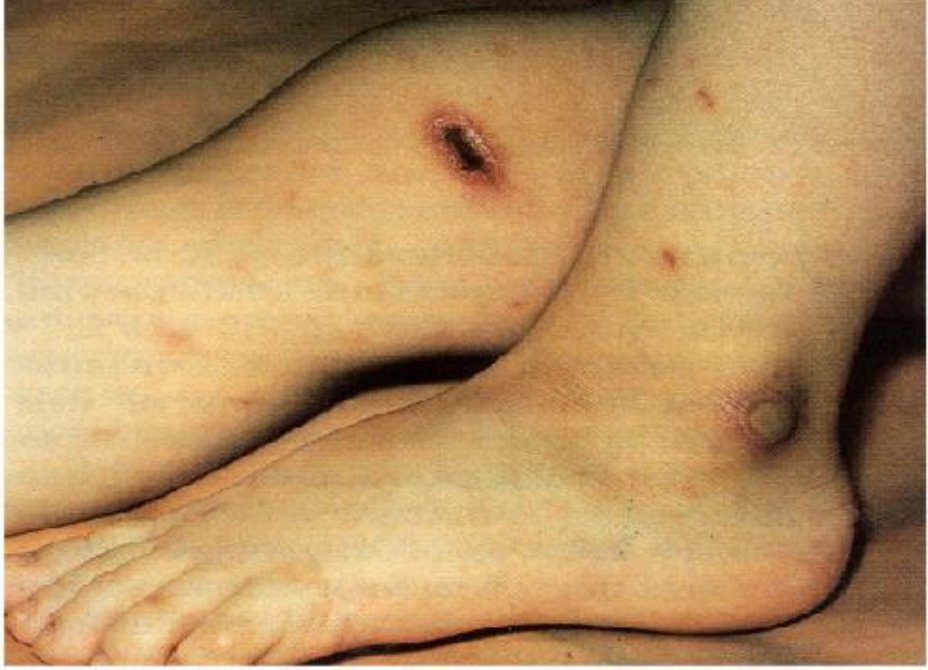


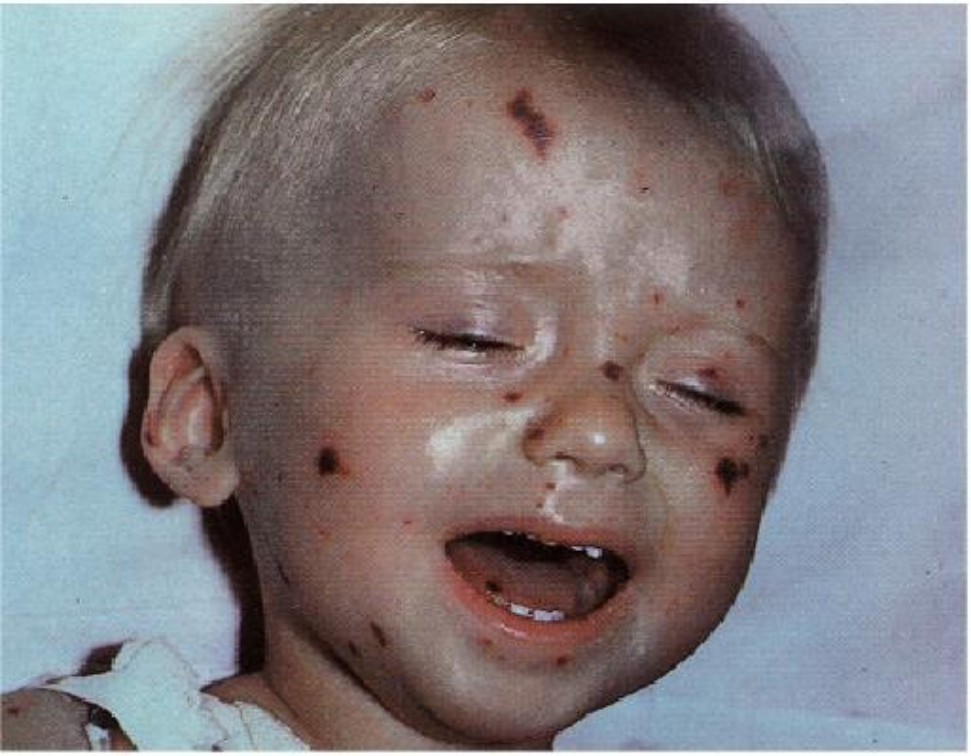
Менингококкемия. Крупные кровоизлияния в кожу рук и ног.

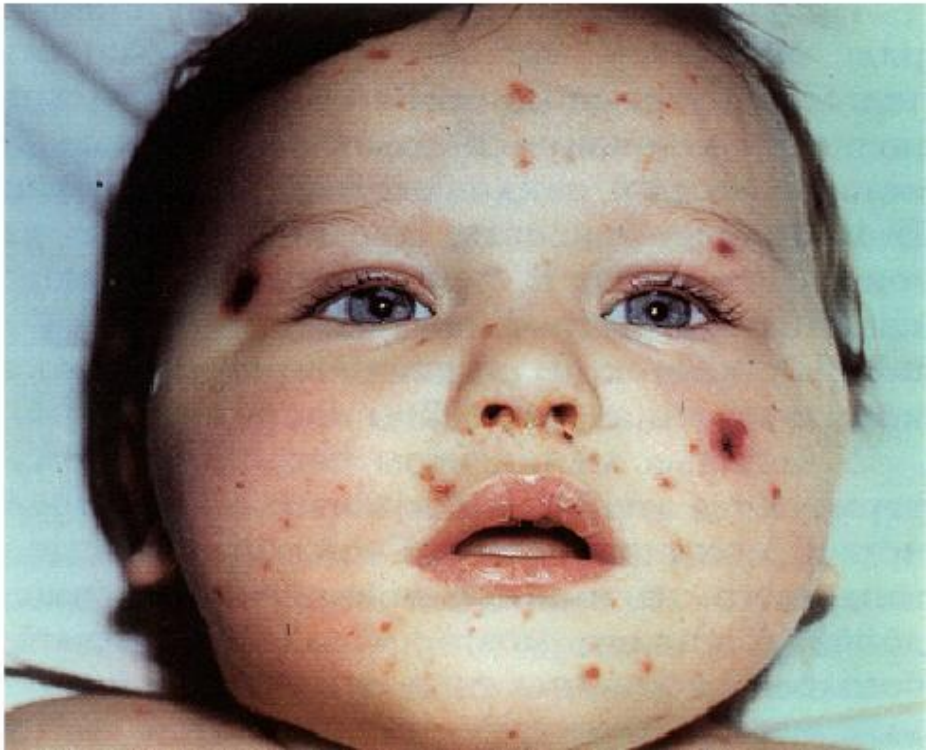


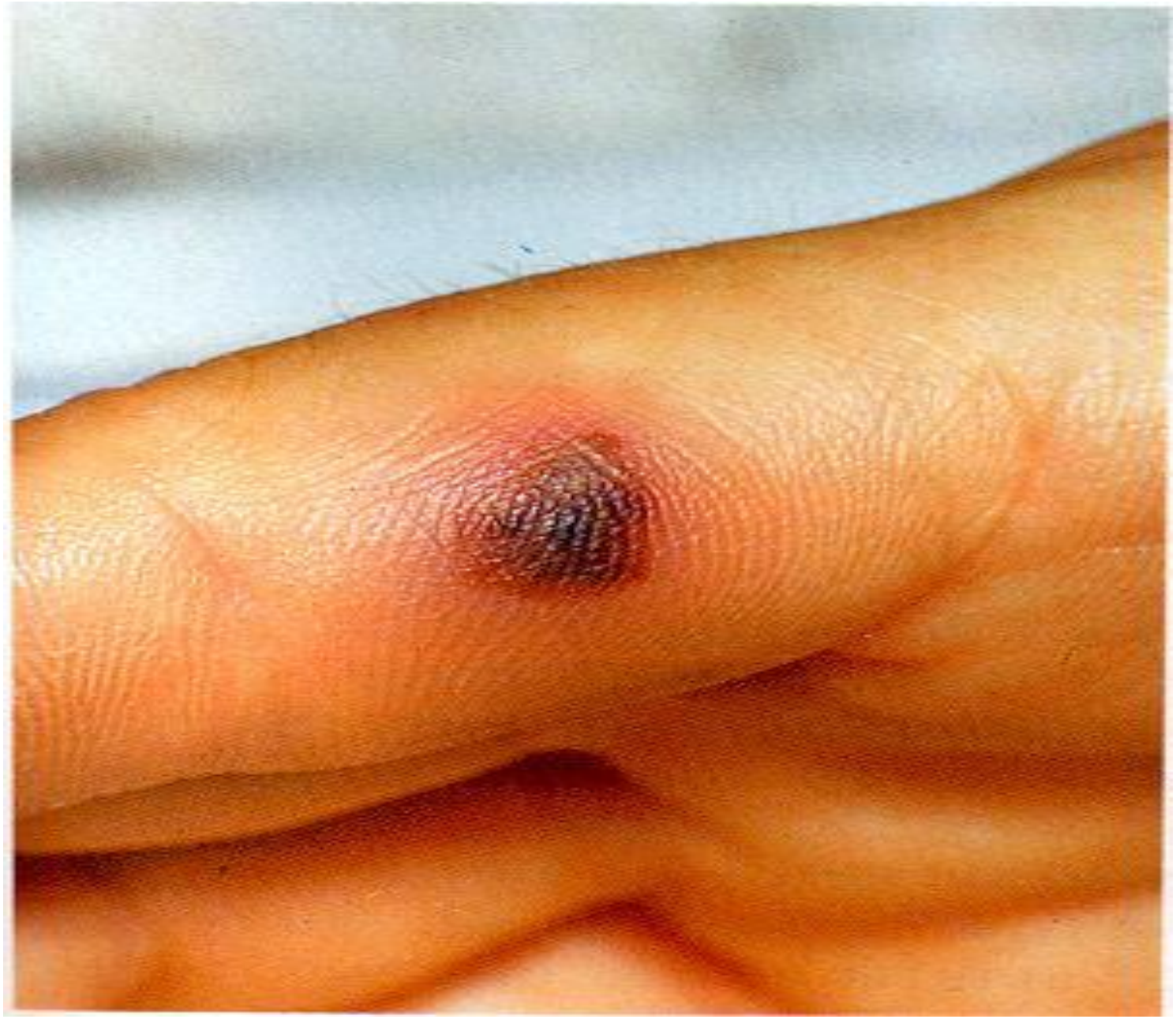
Менингококкемия. Некроз кончиков пальцев (12-й день болезни).

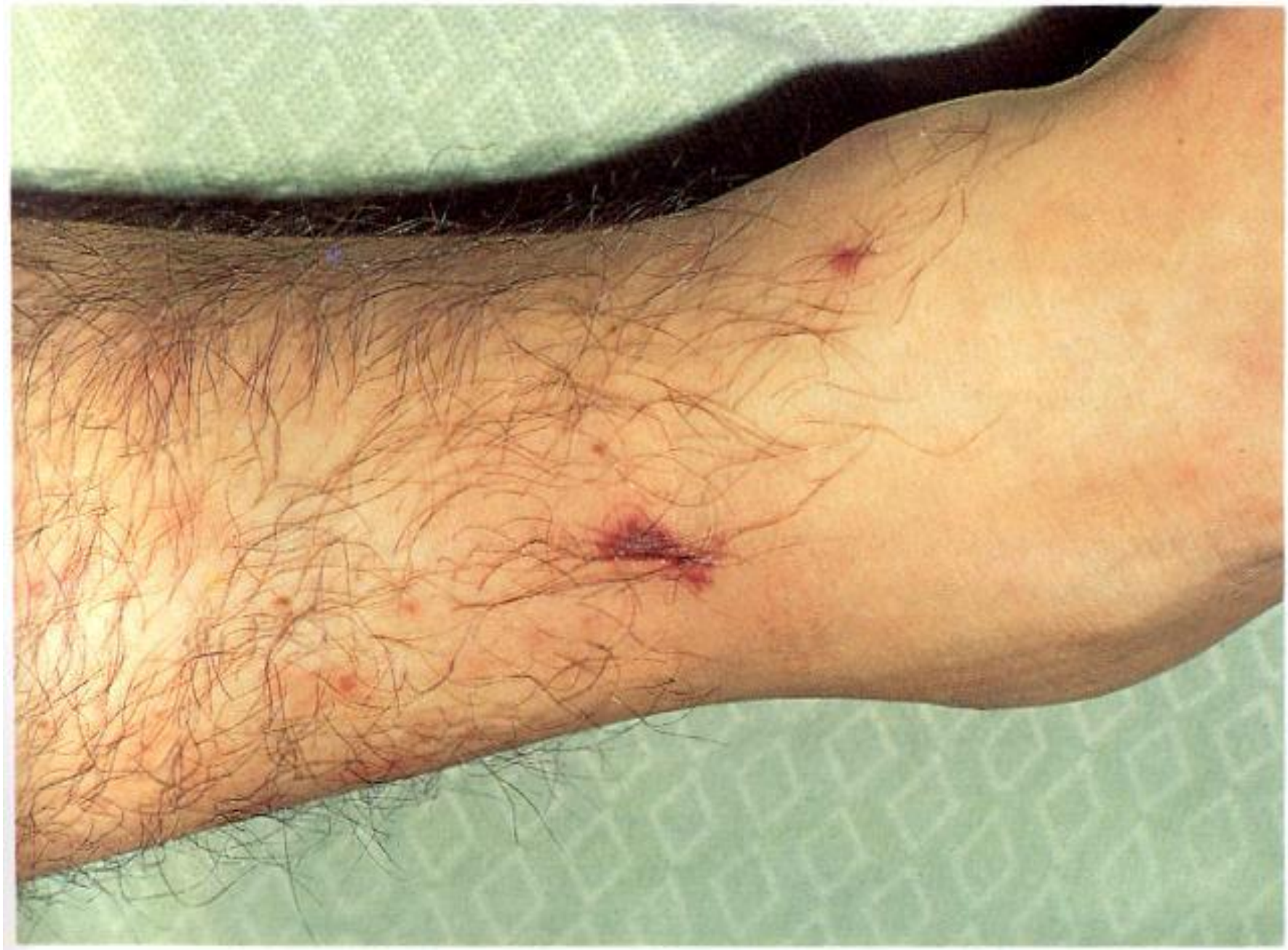


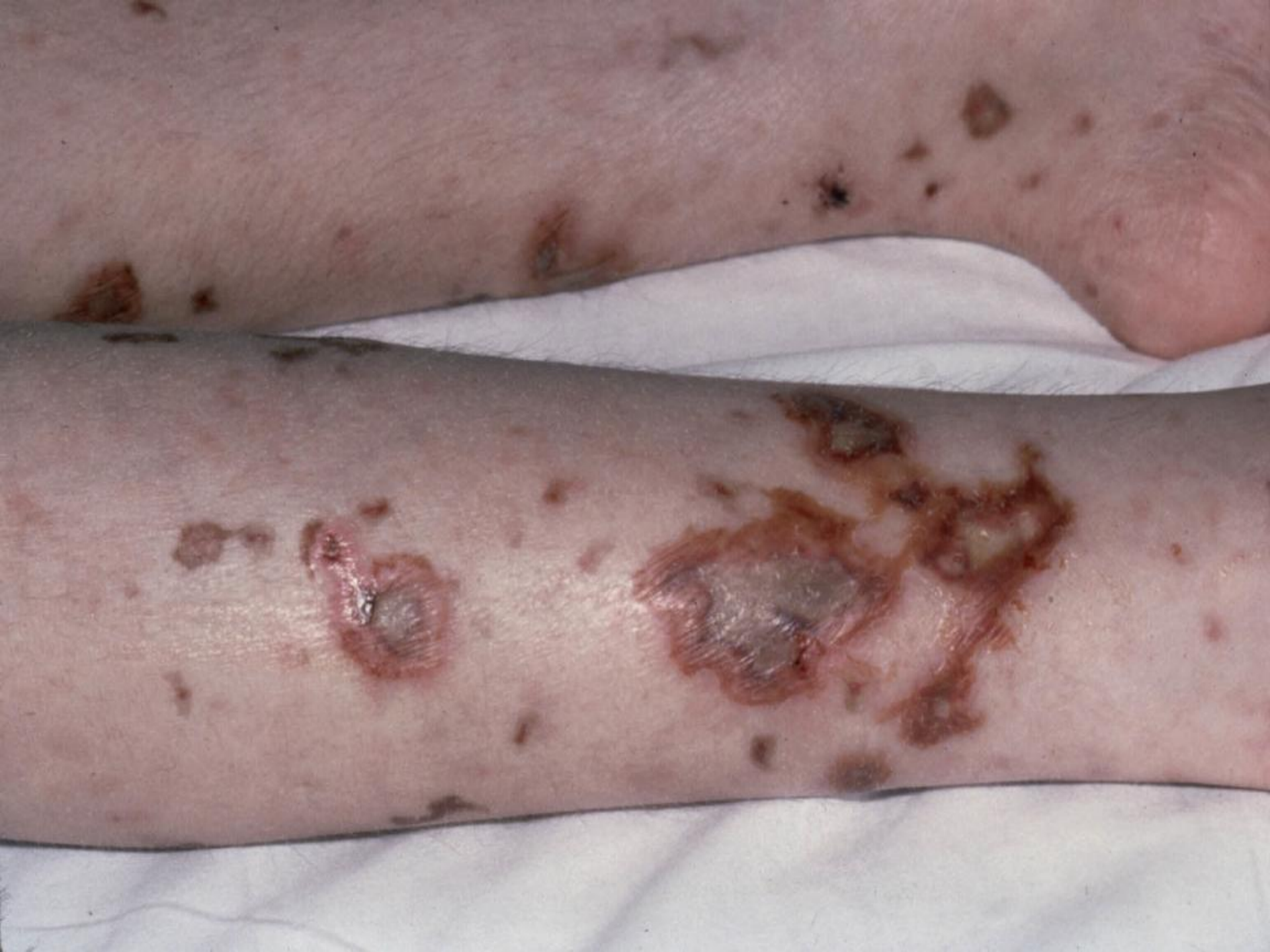


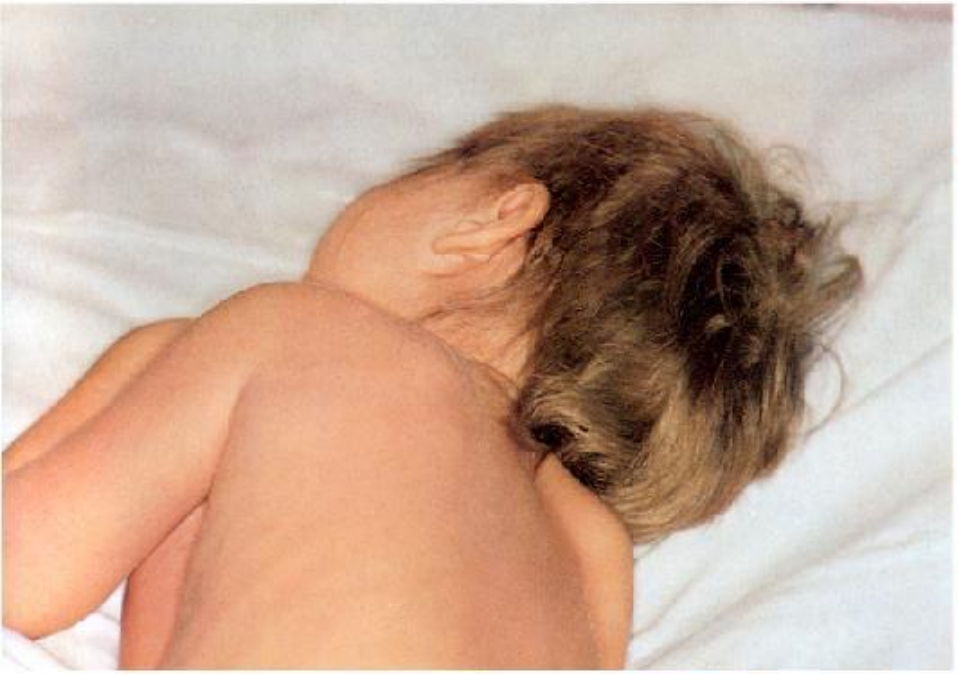


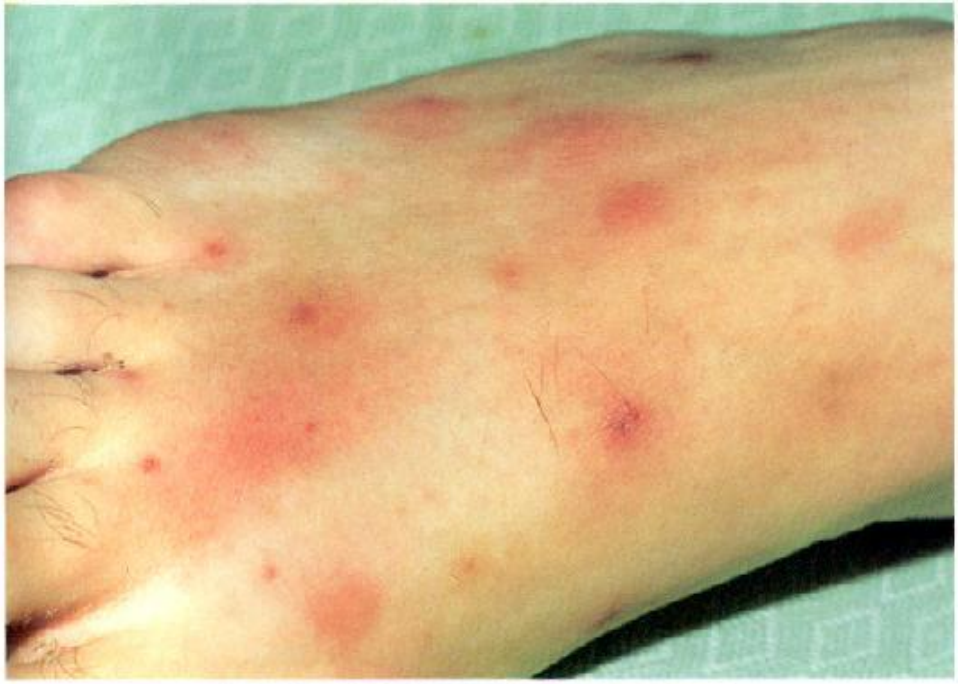


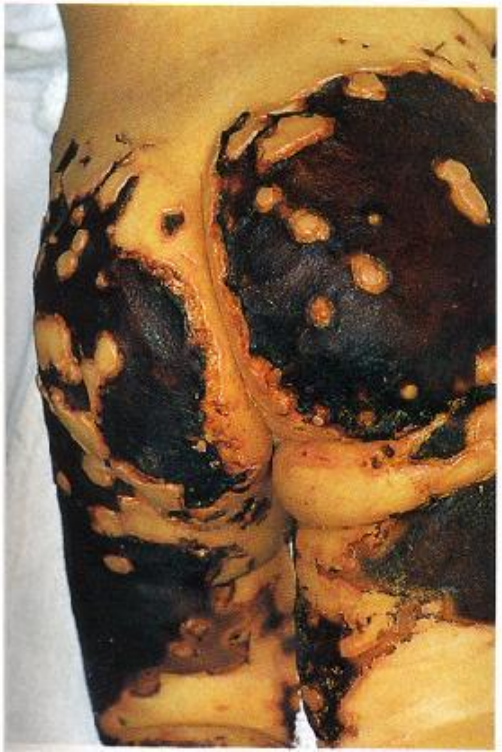


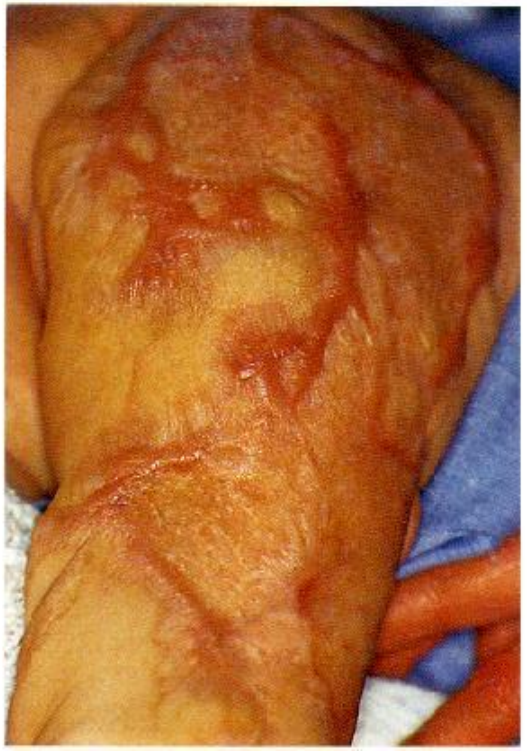


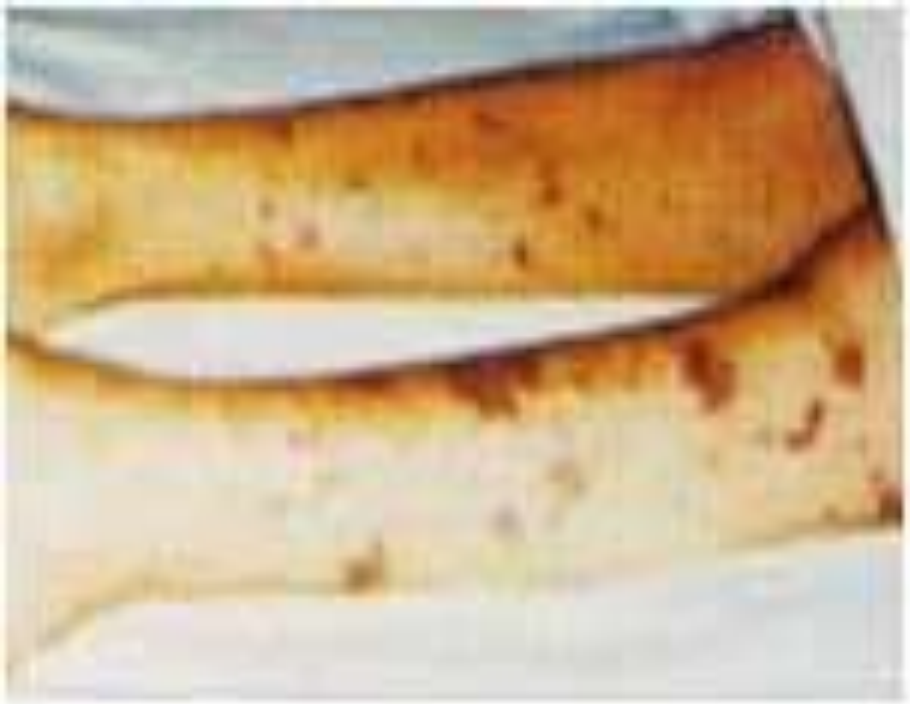


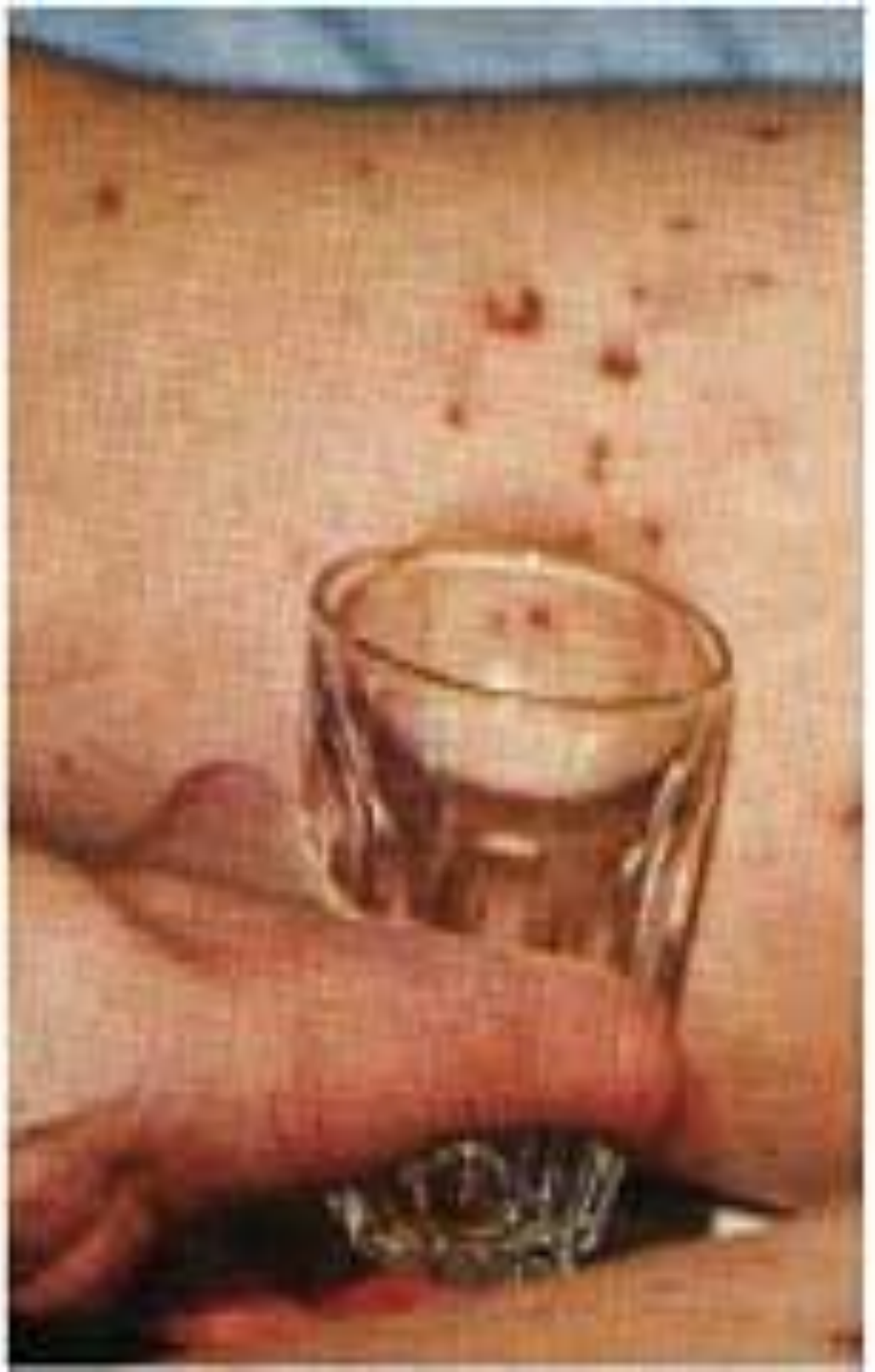


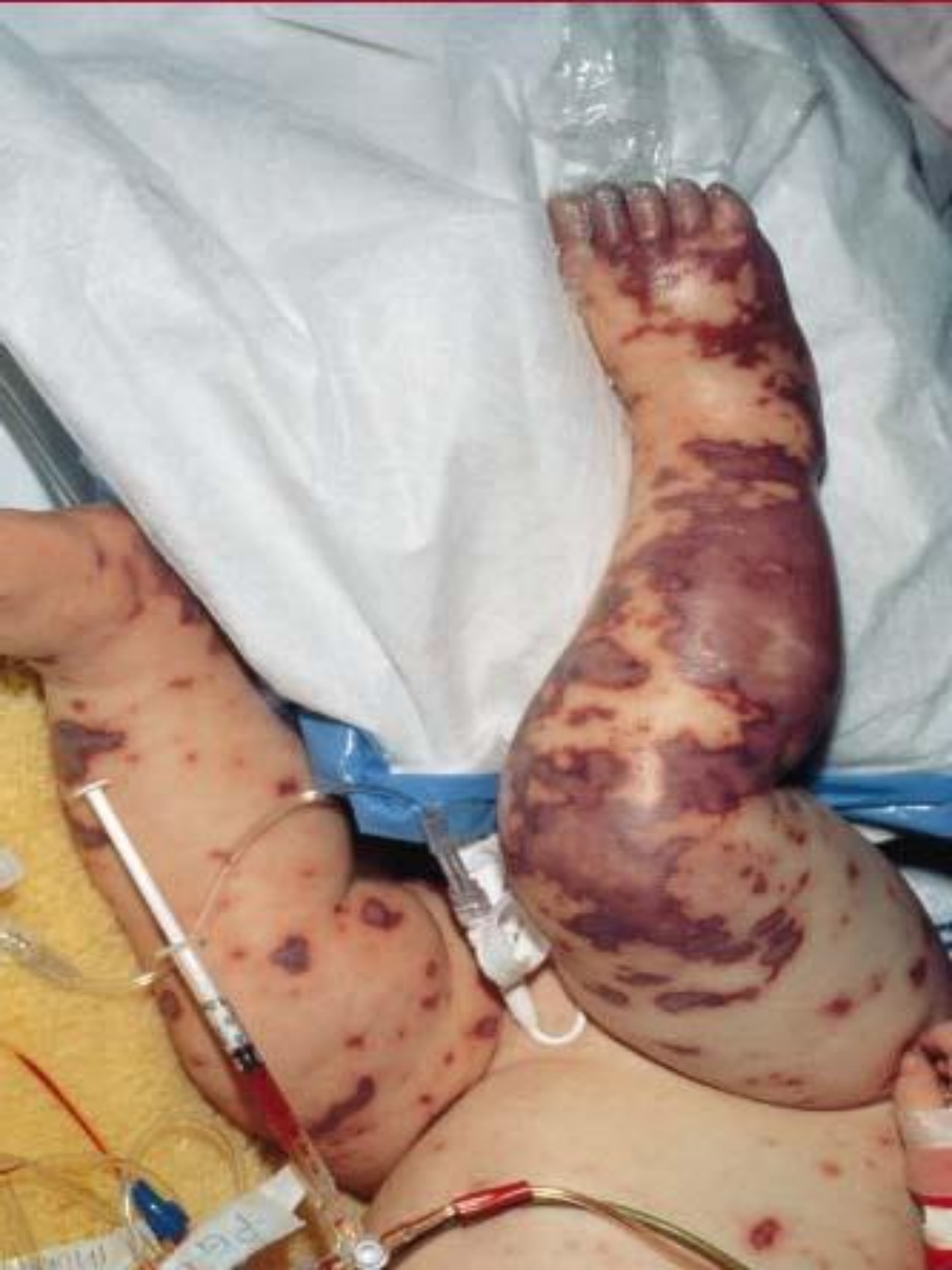


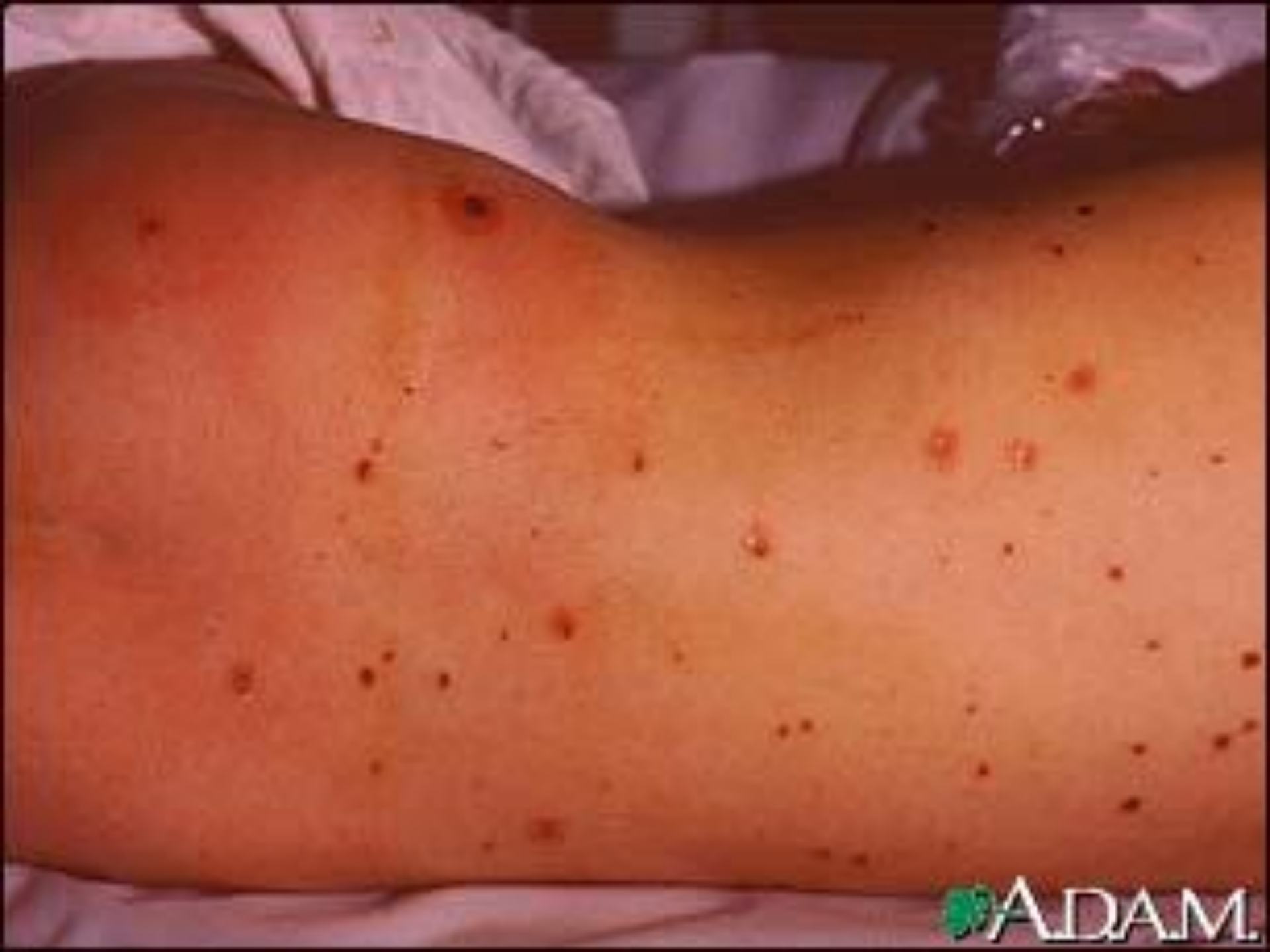











A.D.A.M.

-CVS - dull heart sounds, hypotonia, tachycardia

- RT - dyspnea, cyanosis, superficial breathing, dry rales

- GIT - coated tongue, constipations, increased liver and spleen sizes

- UGT- decreased diuresis, proteinuria, leucocyturia, erythrocyturia, casts

-WBC – hyperleukocytosis, increased ESR (40-65 mm/h)

- Electrolites - hypokalemia, hyponatriumia, hypochloremia

Differential diagnosis will be carried out with

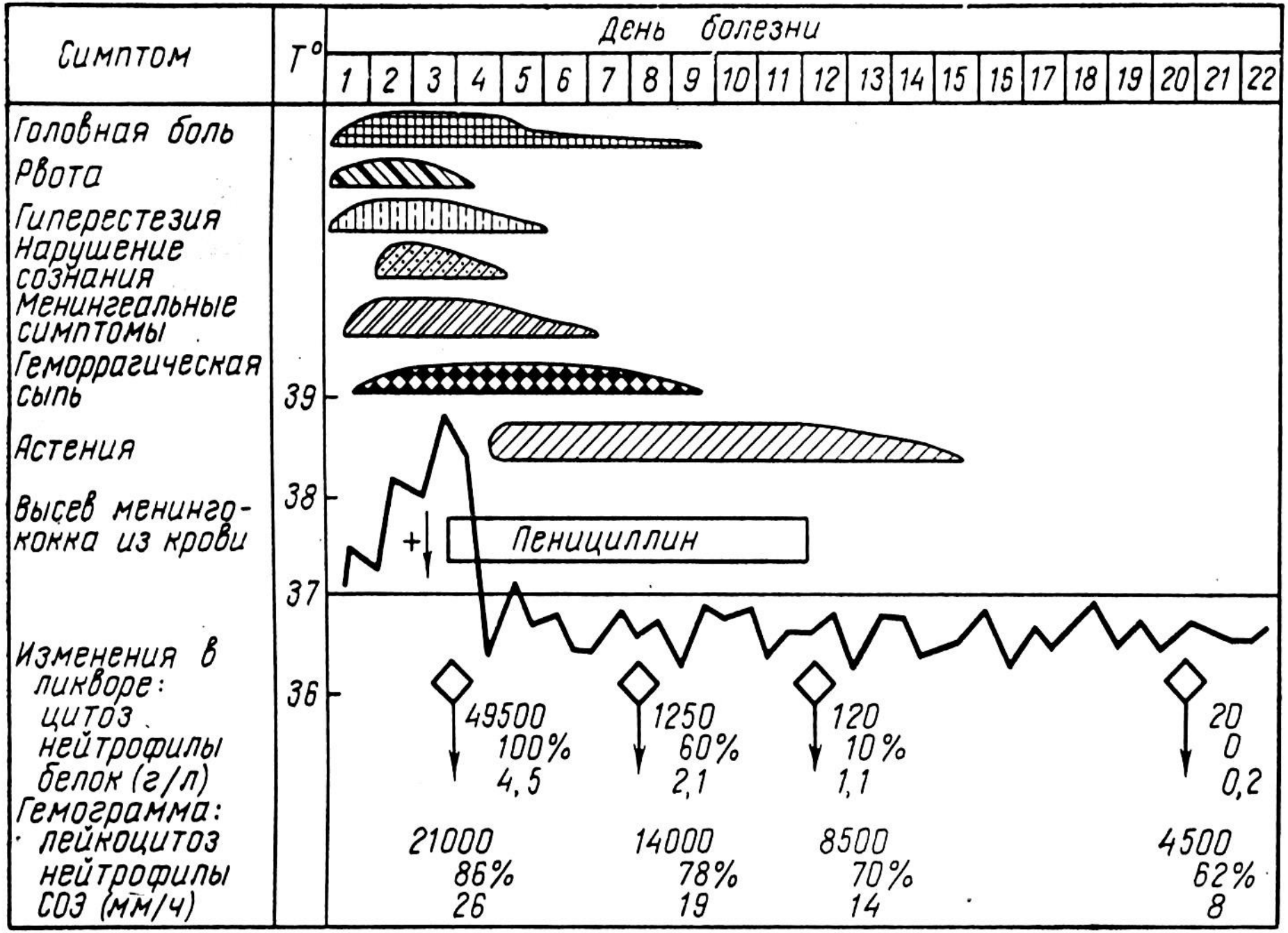
- haematosepsis,
- severe influenza,
- hemorrhagic vasculitis,
-Werlhof's disease (idiopathic thrombocytopenia purpura).

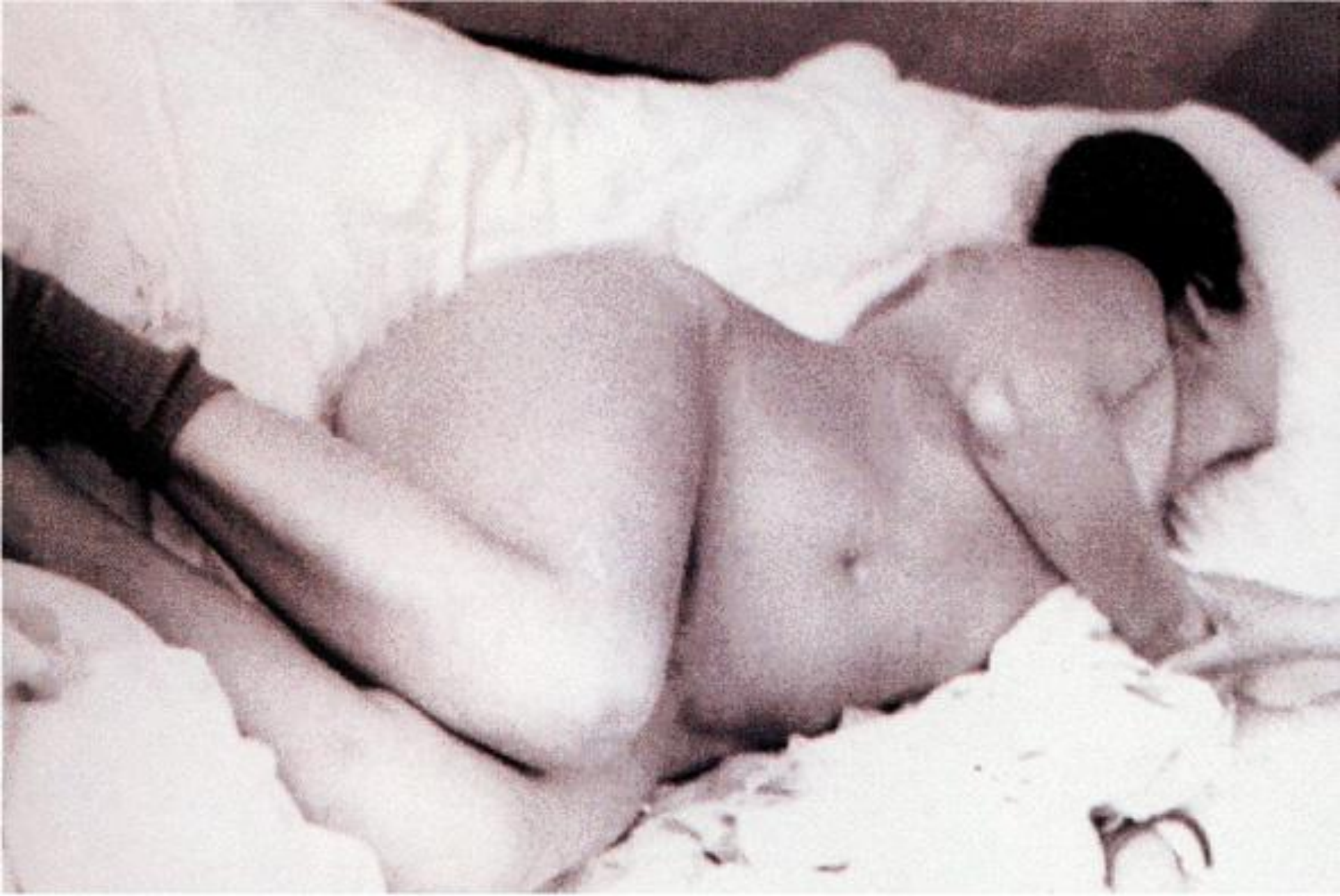
CHRONIC MENINGOCOCCAL SEPSIS is more often characterized by:

- loss of appetite
- loss of body mass
- increased body temperature
- arthralgia or purulent arthritis
- spotted or papular rush on the skin
- meningococcal subacute **ENDOCARDITIS!!!**

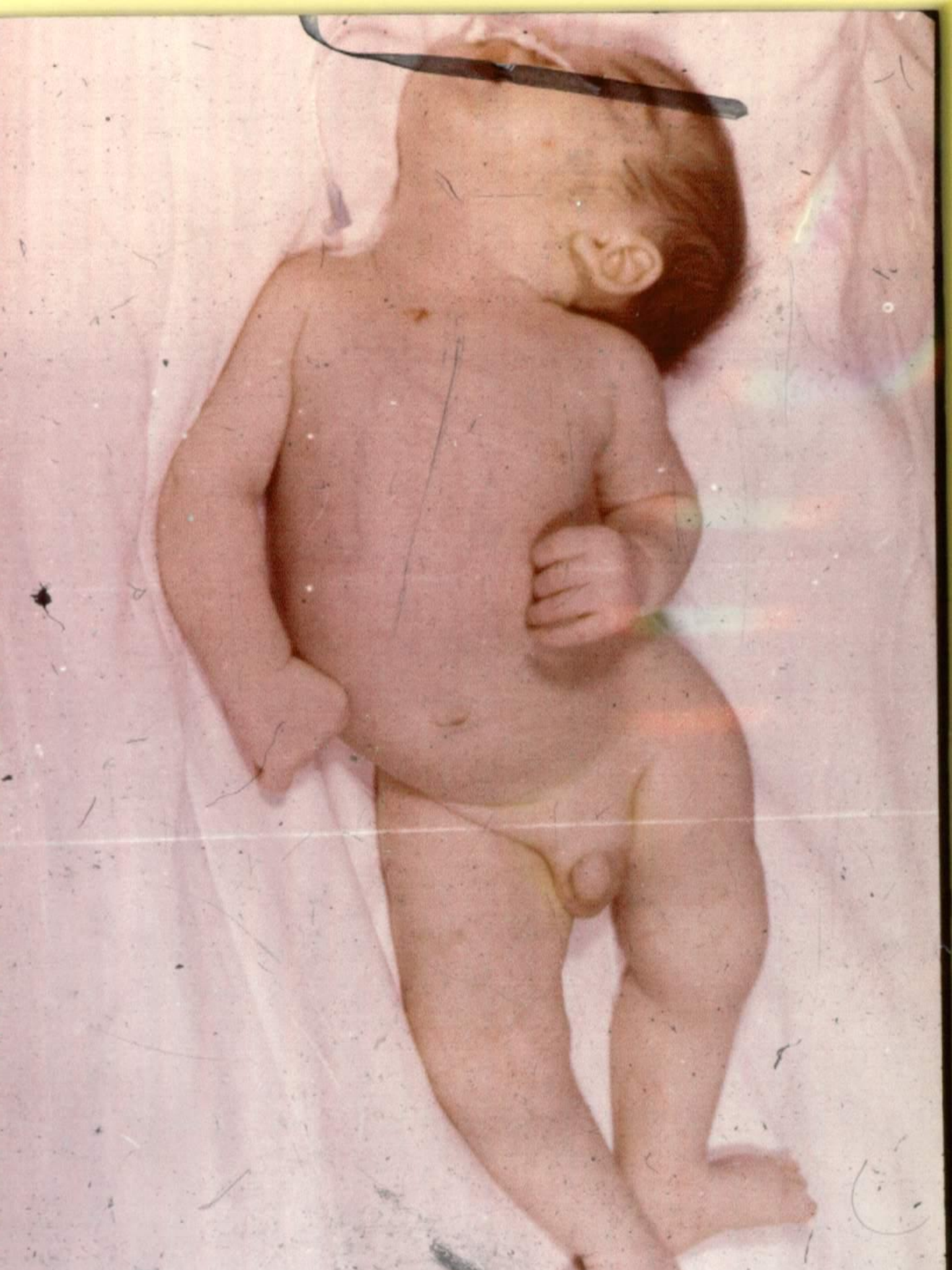
MENINGITIS

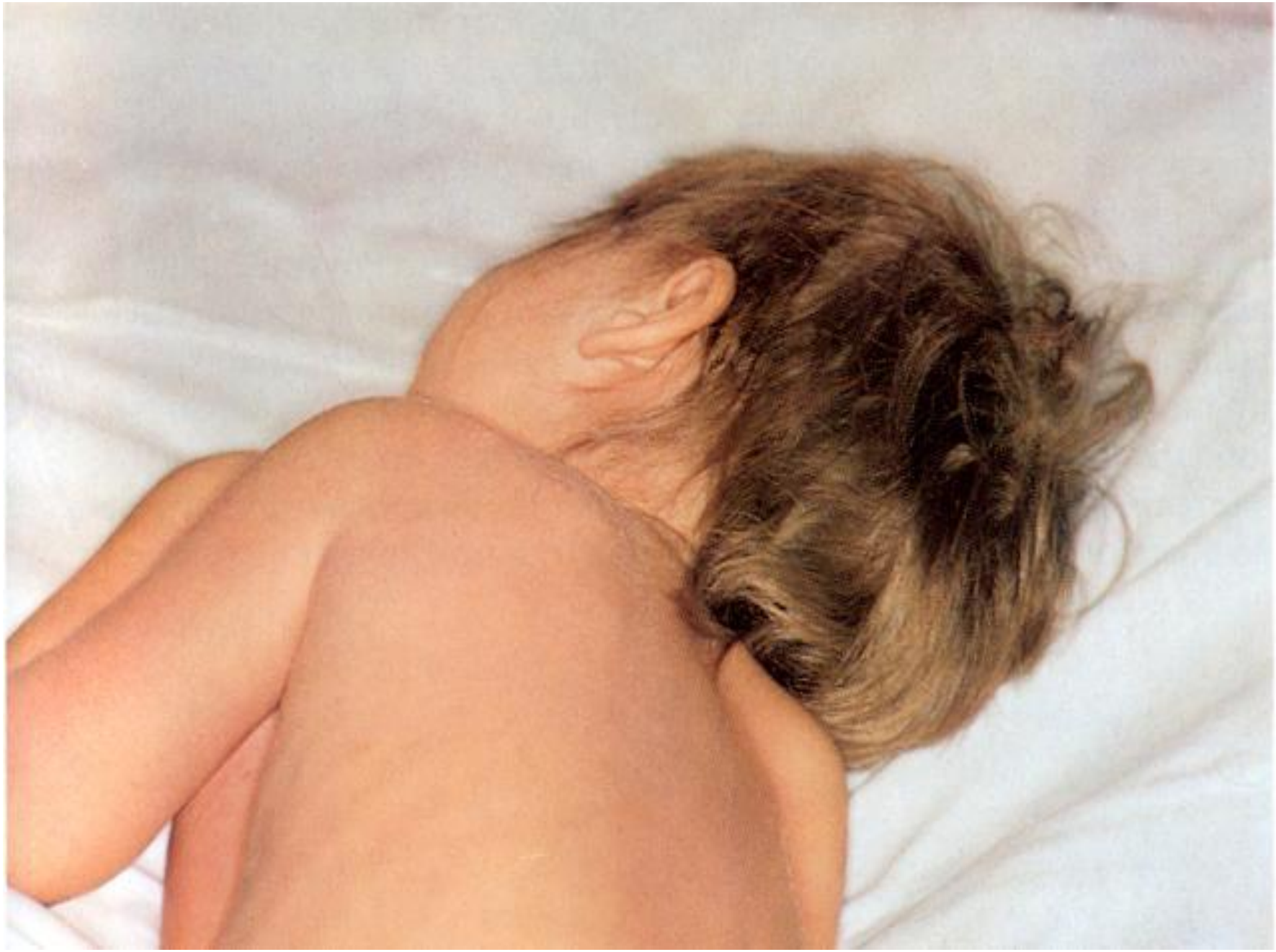
- the sudden beginning (in first hour of disease)
- high fever
- intensive headache in the occipital area
- vomiting, which does not bring relief
- hyperesthesia, hyperacusia, photophobia
- appearance and increase of an aggressiveness
- Meningeal sms - rigidity of neck muscles, “+” Kernig’s, Brudzinski’s symptoms)
- damage of cranial nervous:
- 3-4 pair (diplopia, ptosis, anisocoria)
- 7-8 pair (12,7 %)
- In CSF – neutrophils are more then 1000 in 1 mcl (83 %), increased protein and dicreased level of a glucose.

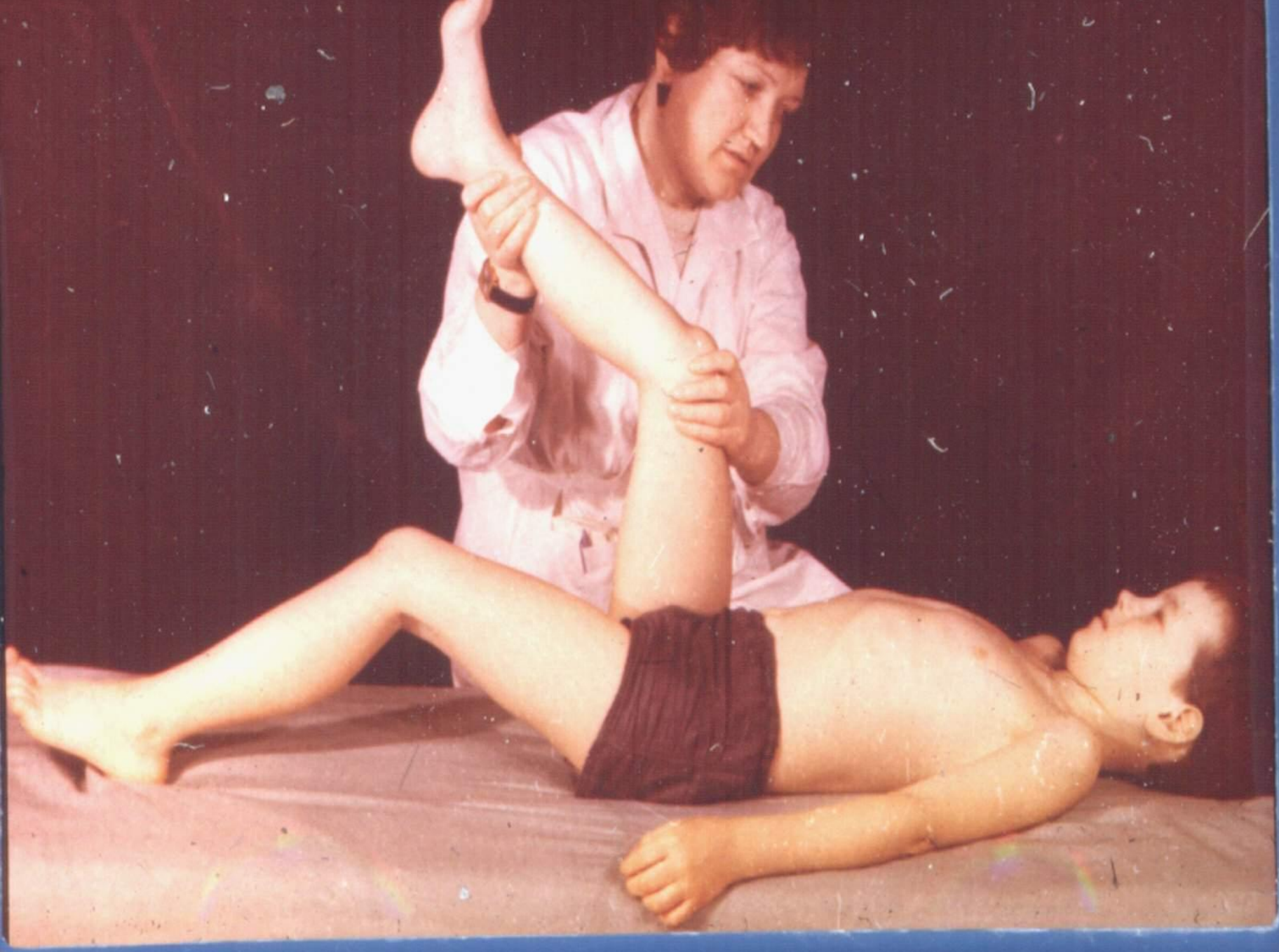

Симптом
T°
День болезни
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22
Головная боль
Рвота
Гиперестезия
Нарушение сознания
Менингеальные симптомы
Геморрагическая сыпь
Астения
Высев менингококка из крови
39
38
37
36
+
Пенициллин
Изменения в ликворе:
цитоз
нейтрофилы
белок (г/л)
49500
100%
4,5
1250
60%
2,1
120
10%
1,1
20
0
0,2
Гемограмма:
лейкоцитоз
нейтрофилы
СОЭ (мм/ч)
21000
86%
26
14000
78%
19
8500
70%
14
4500
62%
8

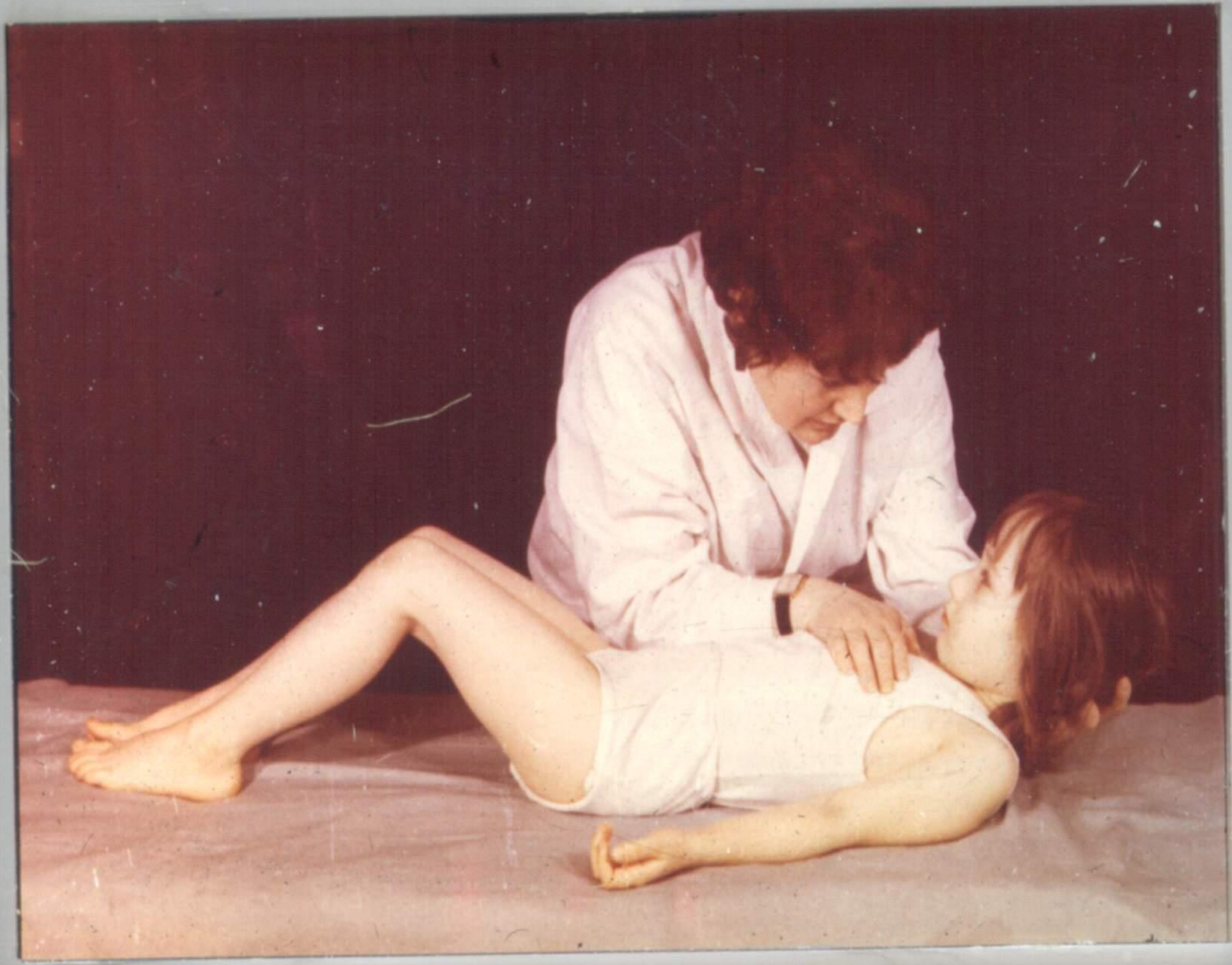


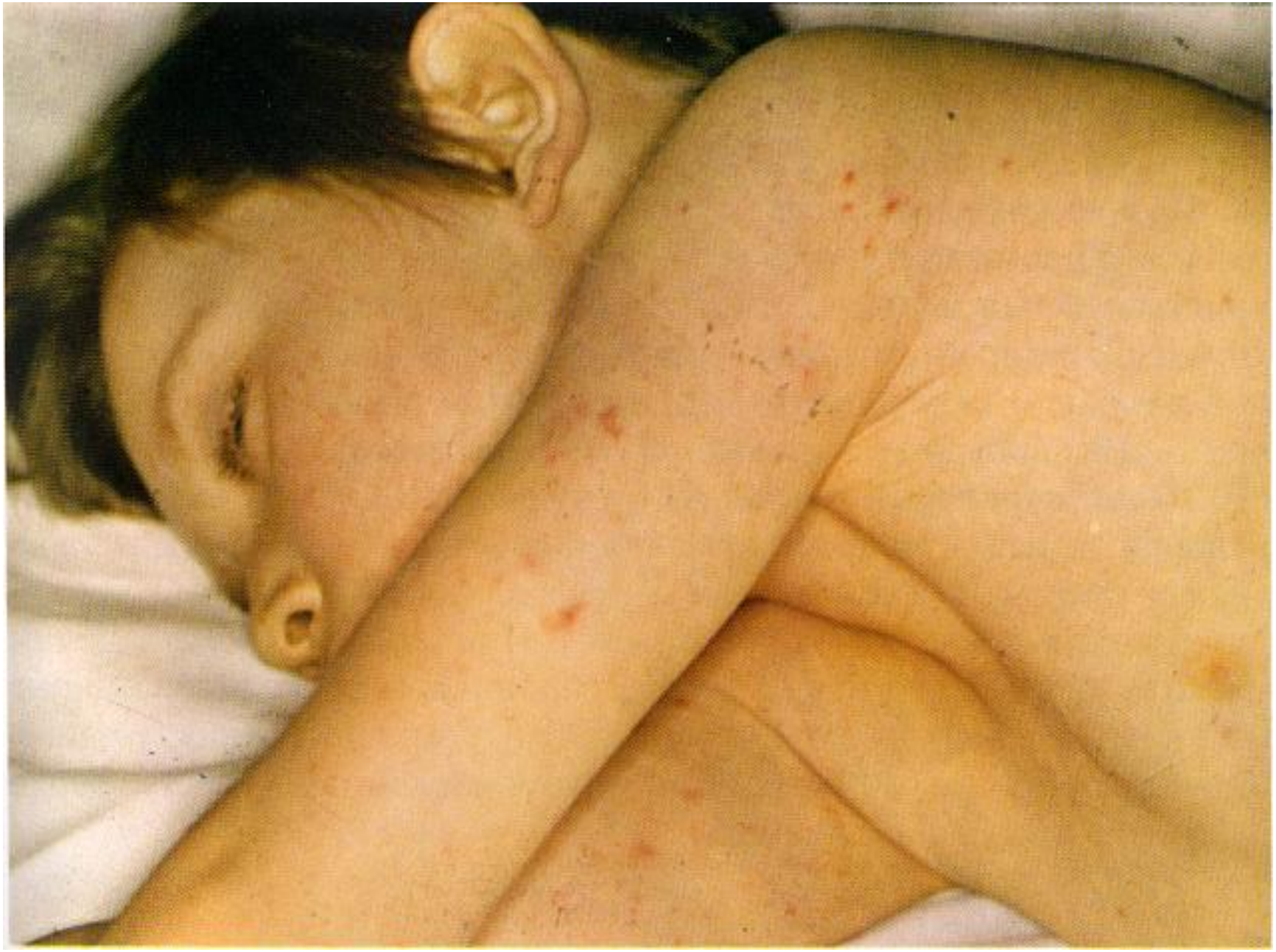
Менингококковый менингит (2-й день болезни).
Синдром «укорочения» (характерная поза).

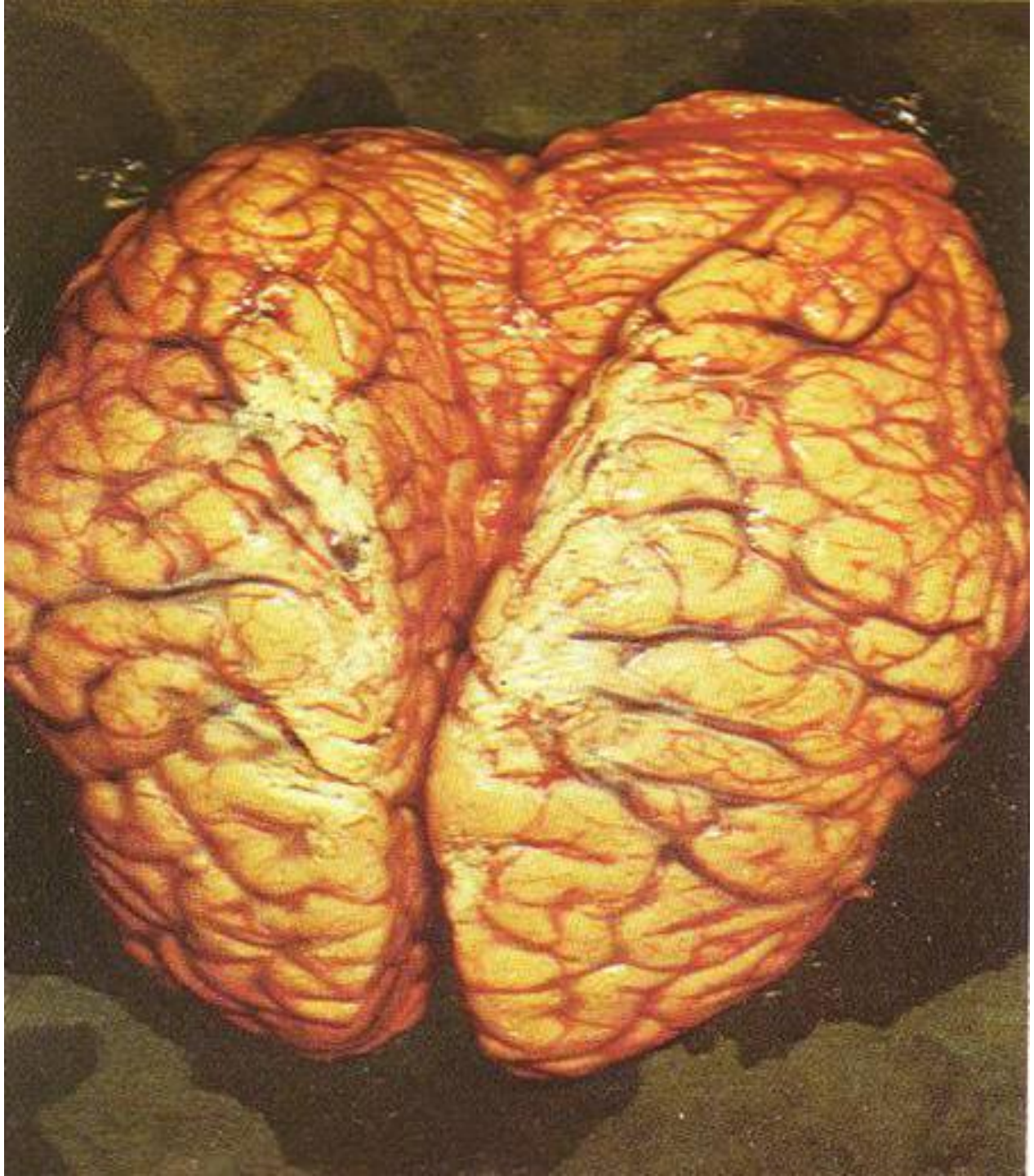


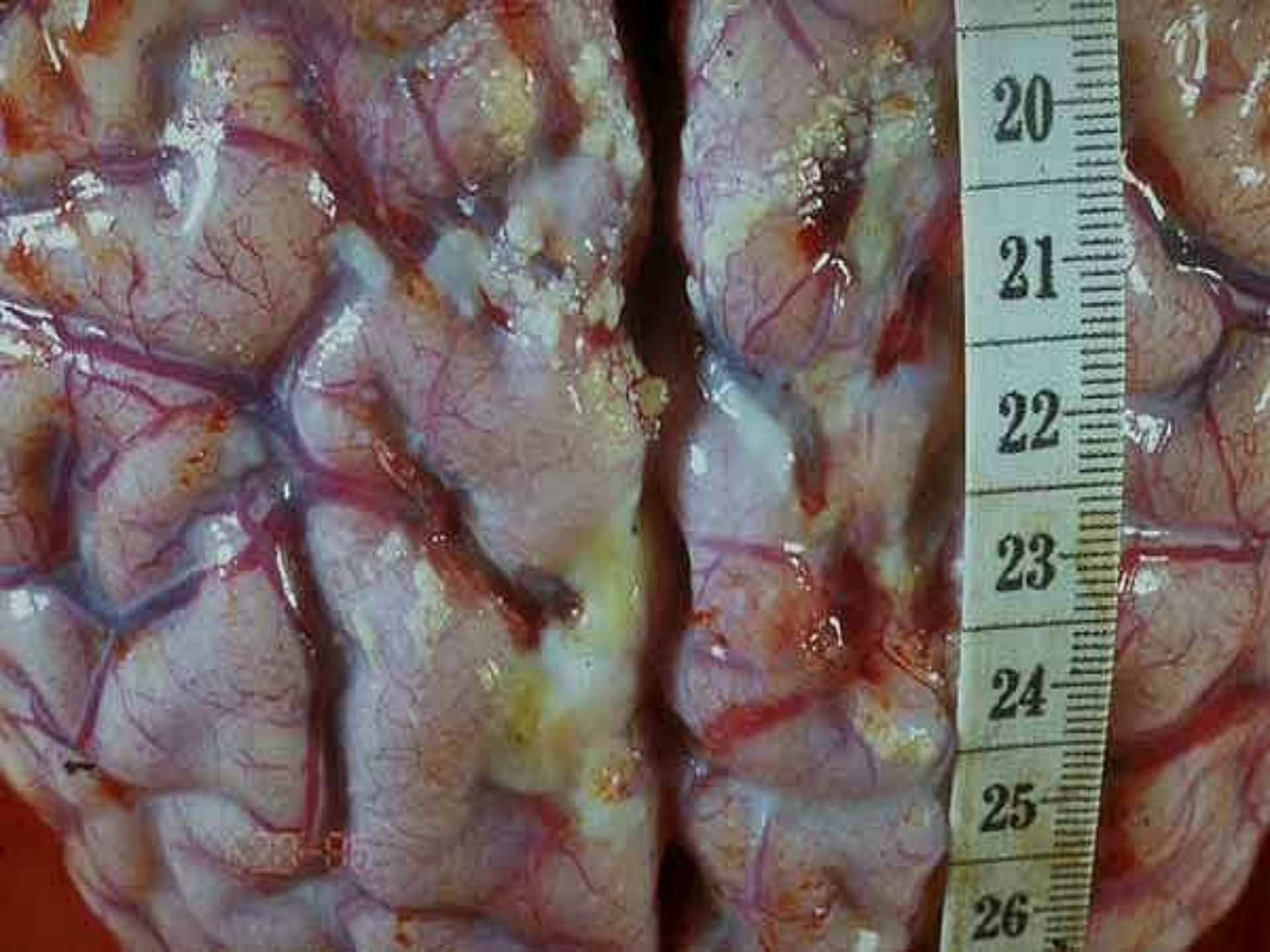








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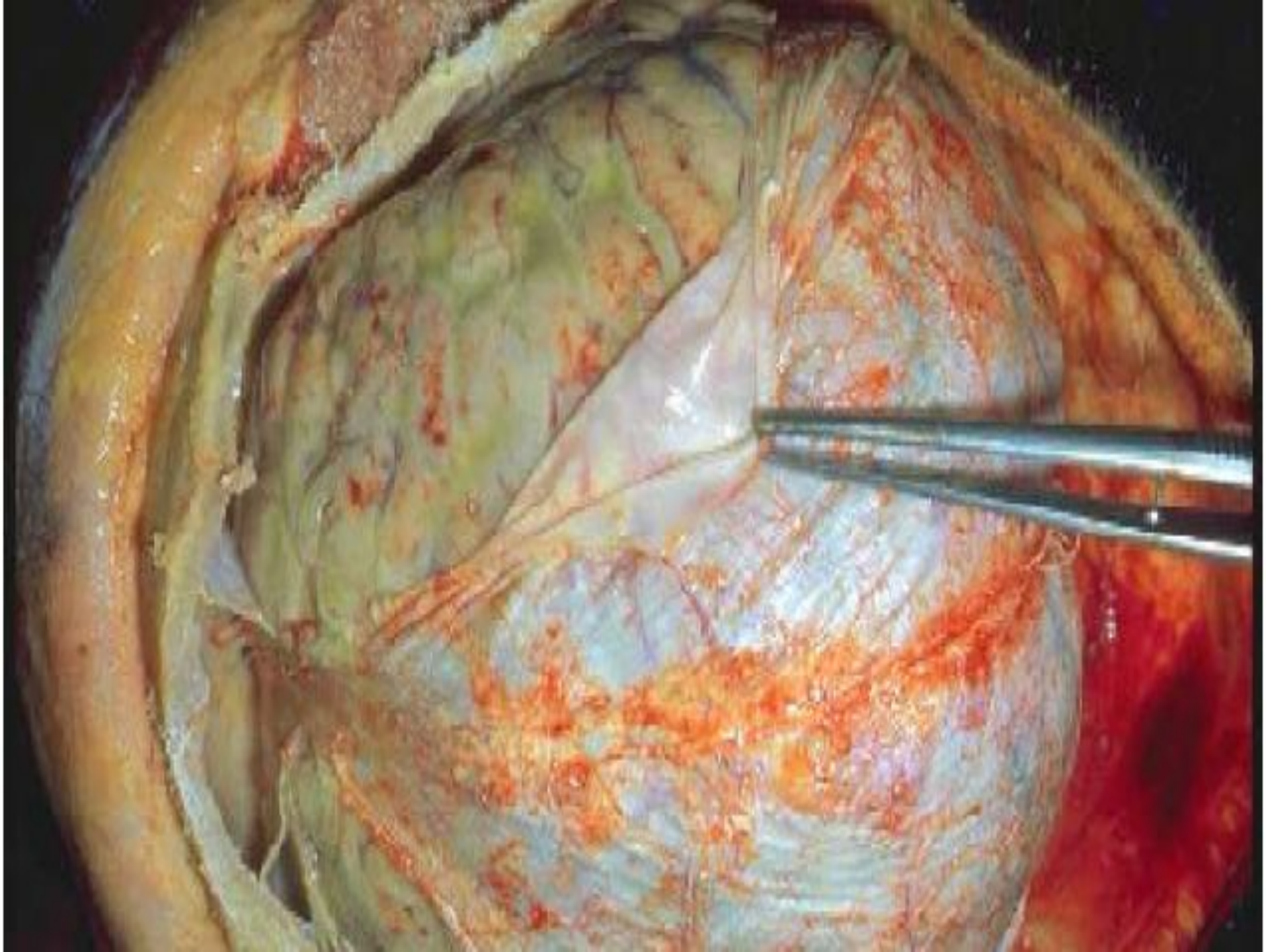


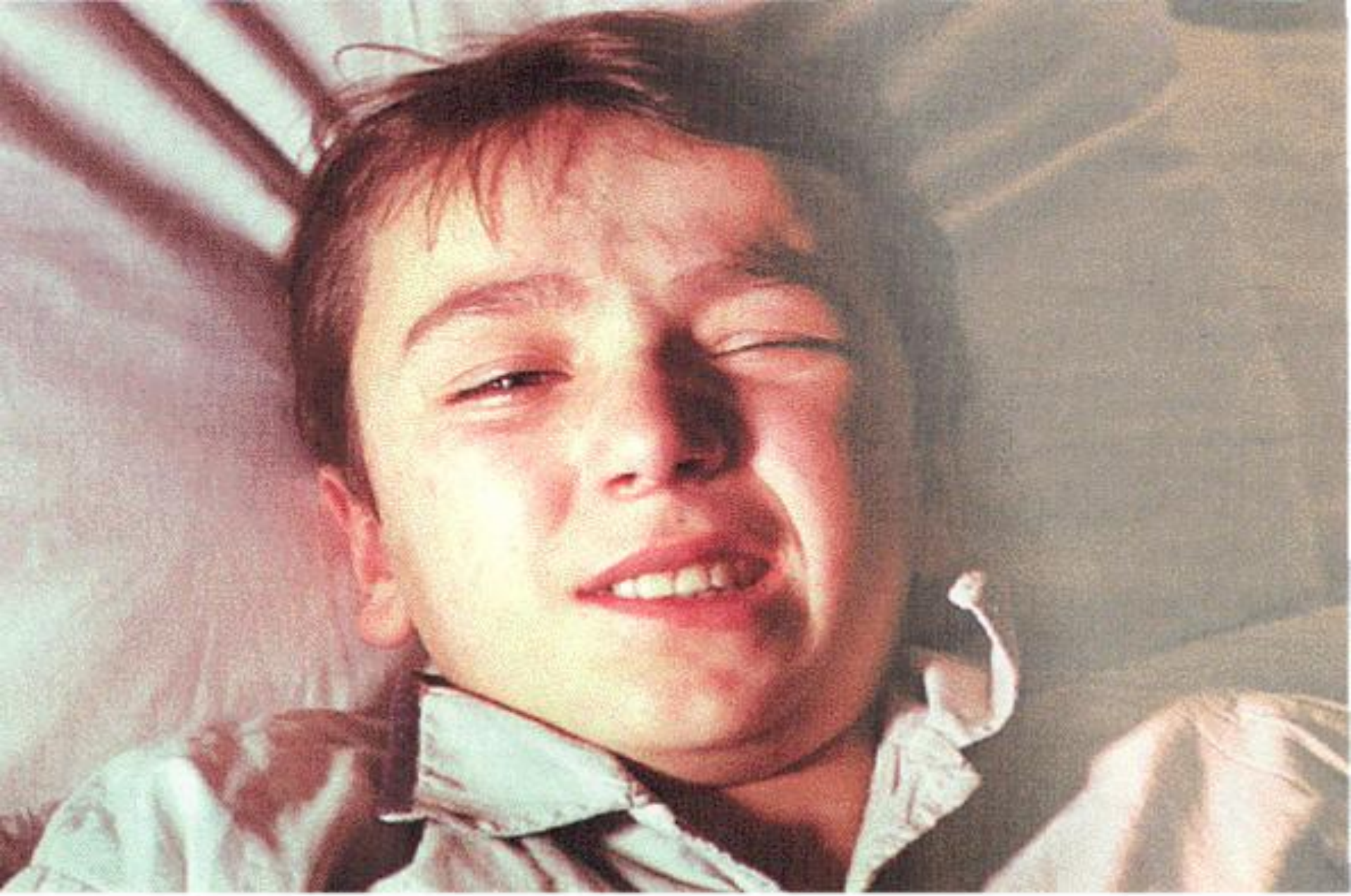
MENINGOCOCCAL MENINGOENCEPHALITIS

-more often appears due to diffuse damage of a brain with desorded consciousness and focal changes:

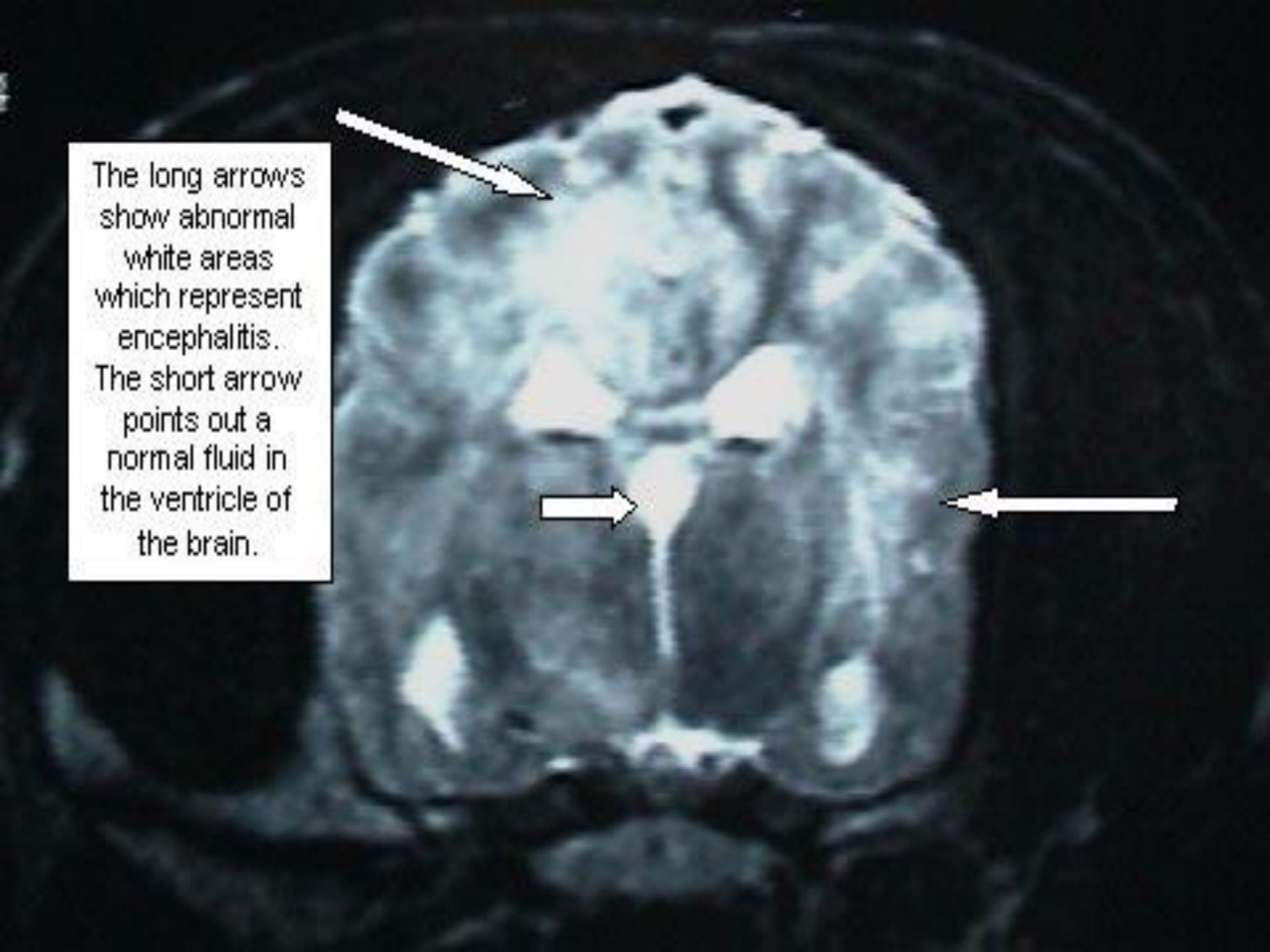
- Aphasia - 3 %
- Psychosensorial disorders - 1 %
- Cramps, mono- and hemiparesis - 3 %
- Oculomotor disorders - 27 %

COMPLICATIONS:

- Acute renal unsufficiency
- Dural and subdural exudates
- Viral or bacterial superinfections
- Activation of simple herpes in 38 %
- Pneumonia - 6 - 22 %, otites, cystopyelitis 4,1 %



Менингококковый менингит (период выздоровления).
Парез лицевого нерва.


The long arrows show abnormal white areas which represent encephalitis. The short arrow points out a normal fluid in the ventricle of the brain.

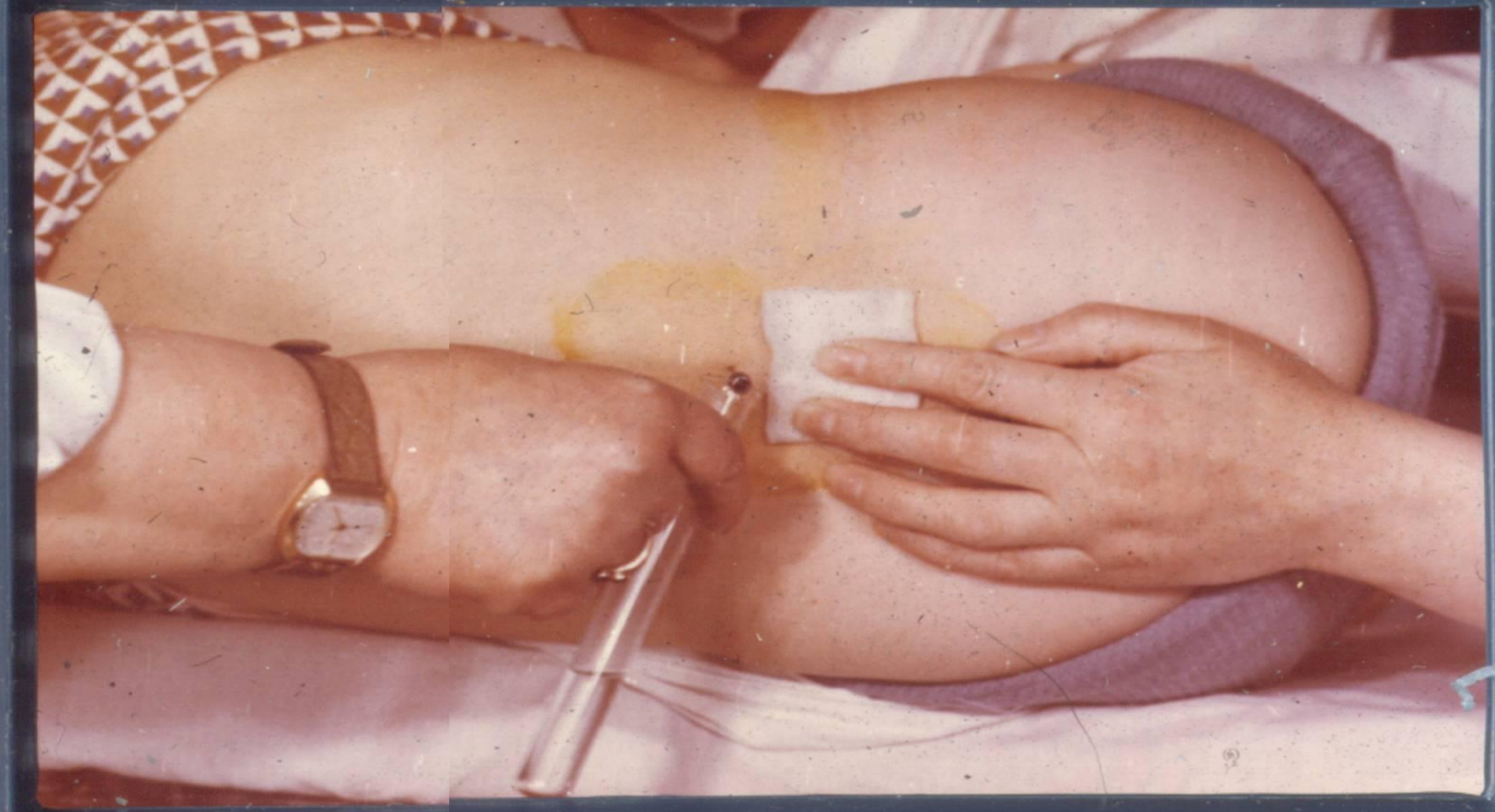
LABORATORY DIAGNOSIS:

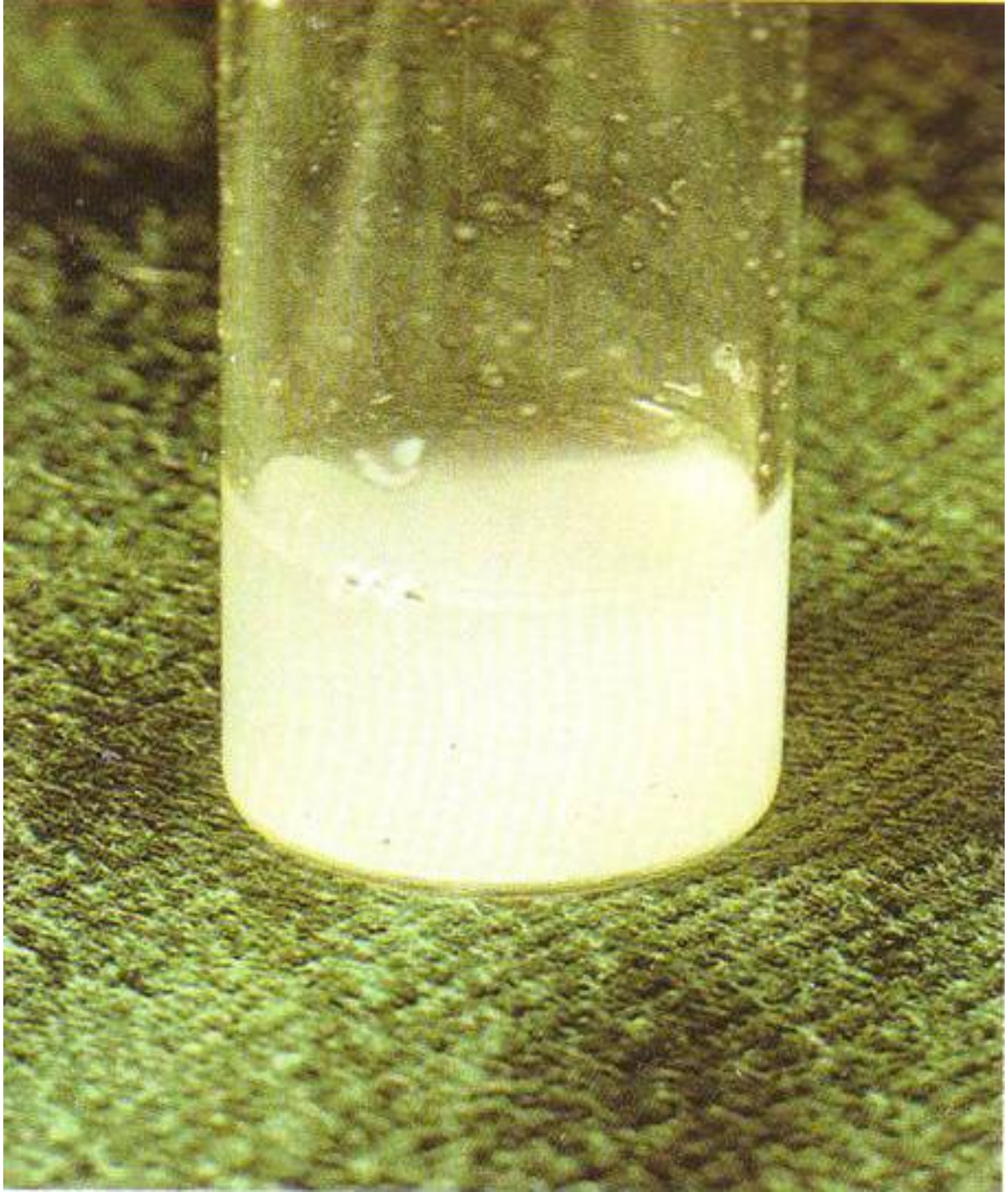
Microscopy of smear from a mucous of nasopharynx, CSF and thick drop of a blood (detection of intracellular gramme (-) diplococci)

Biochemical and morphological investigation of CSF (cytosis, contents of protein and glucose, detection of a film at the bottom test tubes)

Common analysis of a blood and urine

Immunological investigation - RHA (diagnostic titer - 1:160) and RIHA (1:200), ELISA





Examination of cerebrospinal fluid

-The tube №1 (1 ml) – erythrocytes, leucocytes, cells ratio

-The tube №2 (1 ml) – protein, glucose, chlorides

-The tube №3 (2 ml) – growing of bacteria (a blood or chocolate agar)

-The tube №4 (1 ml) - after centrifugation and sedimentation coloring by Gram and with Indian ink

-The tube №5 (1 – 2 ml (ml) - is saved and used for detection of other causative agents

TREATMENT

Morbidity of generalized forms of a meningococcal infection is changed in 8.6 % up to 24 % and depends on efficiency of treatment!!!

The main role in treatment belongs to antibacterial therapy, which should start immediately within period of fevers + 3 days of apyrexia (at the sepsis) or at decrease of cytosis in CSF less than 30 cells (lymphocytes) :

Penicillinum - 18-24 mln of IU/d - i/m (divide on 6-8 times)

- Chloramphenicol - 15 – 25 mg/kg IV (divide on 2-3 times)

- Tetracycline in a dose 8 mg/kg IM in 6h

- Ampicillin, metacyclin, oxacillin in a dose 30-50 mg/kg in q4h IV or IM

- Cefatoxim 1g. IV or IM in q 12h
- Ceftriaxon 1 - 2 g. IV or IM in q8-12h
- Byceptol 980 мg PO, IV in q12h
- Sulfamonomethoxinum 4 g the first day, then 2 g. PO in q12h

The nasopharyngitis is treated 5-7 days with erythromycin, azytromicin or sulfanilamidums

Pathogenetic therapy:

- dehydrational therapy (at edema of brain)
- desintoxication therapy and glucocorticoids at TIS
- metabolites
- treatment of a hemorrhagic syndrome

Symptomatic therapy

Pneumococcal meningitis

- **Anamnesis –**

★ **Source** - **patient** with various forms of pneumococcal infection and **carriers** of the pneumococcus.

★ **Route** of transmission — **airborne**, but exogenous and endogenous route possible.

★ **18-51%** of purulent meningitis in adults is pneumococcal etiology,

★ **1/3** of cases are secondary and develop on the background of otitis, sinusitis, pneumonia, sepsis.

★ **100%** of recurrent meningitis with pneumococcal liquorrhea.

★ **Pathogen** – **Streptococcus pneumoniae**, stained gram positive.

• **Onset** is acute.

- Rapid rising of body temperature to 39-40ºC;
- Progressing of general intoxication and cerebral symptoms (headache, vomiting, loss of consciousness, repeated seizures).

• **Meningeal syndrome** appears early and expressed, but sometimes incomplete, accompanied by signs of edema-swelling of the brain.

• Quick involvement of brain substance in the pathological process, accompanied by the development of **meningoencephalitis**, appearance of mono - and hemiparesis, oculomotor disorders, hyperkinesis, ataxia.

On the 3-4 day of the disease can develop coma status with the symptoms of brain stem dislocation.

- **Course of the disease varies from malignant forms with lethal outcome to a proloned and recurrent.**

- **With the development of pneumococcal sepsis may be affection of other organs: hemorrhagic rash on the skin (like meningococcemia), pneumonia, endo - and pericarditis, arthritis.**

- **In 50% of patients residual effects remain, may be seen epilepsy, paresis, paralysis.**

- **Cerebrospinal fluid** is turbid, of yellow-grey color with metallic shade.

Cell count up to 30000 in 1 mkl, protein - 2-5 g/l.
Pressure of CSF is increased moderately due to accumulation of pus in the subarachnoid space and block of CSF pathways.

- **CBC** - expressed inflammatory changes.

- **Bacteriological** examination of blood, CSF detects the **Streptococcus pneumoniae**.

SEROUS MENINGITIS

- is a group of CNS diseases with the similar clinical manifestations and morphology, characterized by the serous inflammation of the meninges, mostly of viral, rarely bacterial, fungal or parasitic etiology, accompanied by acute development of hydrocephalic syndrome and nonexpressed meningeal syndrome.

Depending on the mechanism of CNS demerge serous meningitis are divided into primary and secondary.

The etiology of primary serous meningitis is most often of viral origin, the clinic is characterized only by meningeal syndrome (Acute lymphocytic choriomeningitis, mosquito and tick-borne encephalitis, the meningeal form of poliomyelitis).

Secondary serous meningitis develop on the background of main disease, usually of viral etiology (enterovirus infection, respiratory viral infections, chickenpox, measles, rubella, epidemic. mumps, HIV infection, herpesvirus infection, cytomegalovirus, rabies, arbovirus infection).

Less serous meningitis occurs by bacterial infections (typhoid, tuberculosis, brucellosis, leptospirosis, syphilis, Lyme disease, yersiniosis), parasitic (malaria, toxoplasmosis, amebiasis) or fungal infections (cryptococcosis, coccidioidomycosis, aspergillosis).

Pathogenesis

1.Penetration of the pathogens in the blood

2. Activation of biologically active substances, disturbed homeostasis, increases the permeability of cellular and vascular membranes, including the HEB

3. Causative agents (viruses, bacteria and their toxins) penetrate into the CNF

4. Hyperproduction of CSF due to irritation of the vascular plexus of the ventricles (lower protein level in the cerebrospinal fluid) or serous inflammation of the meninges (increased protein and lymphocytes in the exudate)

Formation of hydrocephalic-hypertensive syndrome is the main link of pathogenesis of serous meningitis and determines the clinical picture and severity of the disease.

Clinical manifestations in serous meningitis are formed from the same syndromes of purulent meningitis, but they have some peculiarities:

- General intoxication syndrome. Its symptoms depend on the character and properties of the pathogen, but common is the presence of fever (38-39ºC), moderate intoxication (headache, dizziness, lethargy, apathy or agitation, loss of appetite), pale skin, dull tones of the heart, sometimes dysfunction of the gastrointestinal tract;

- Meningeal syndrome may be absent in asymptomatic or mild forms of meningitis with inflammatory changes of the CSF.

Often reveals incomplete or dissociation of meningeal syndrome (loss of some symptoms). In most cases, the severity of meningeal syndrome corresponds to the value of intracranial hypertension.

The most constant symptoms are rigidity of occipital muscles, upper Brudzinsky-symptom, visual and tactile hyperesthesia;

-Hydrocephalic-hypertensive syndrome manifested by headache, vomiting, hyperreflexia, seizures, and pyramidal signs;

- CSF - increased pressure, colorless, transparent or opalescent, lymphocytic pleocytosis (100-2000/μl), protein level is normal or slightly increased, glucose is normal (in tuberculous meningitis is reduced).
In the first days of the disease in CSF can be up to 30-50% of neutrophils.

Enteroviral meningitis

- Is caused by ECHO and Coxacky virus of different serotypes;
- Infection is highly contagious;
- Spring-summer seasonality, often outbreaks;
- 70-80% of population - children of 5-9 years;
- Source of infection is a sick man and a virus carrier;
- Mechanism of transmission is aerogenic (airborne), but posible fecal-oral transmission of the pathogen.

1. **Incubation - 2 - 8 days;**
2. **Acute onset with expressed intoxication syndrome (wave-like fever in 2-7 days, headache in frontotemporal region, pain in the eyeballs, dizziness, repeated, sometimes uncontrollable vomiting, lethargy, weakness);**

3. **Encephalitic sd - may be convulsions or delirium, loss of consciousness;**

4. **Hyperemia of the face with pale nosal triangle, injection of sclera;**

5. **Sometimes combined with other forms of enterovirus infection – myalgia, herpangina, polymorphic spotted, papular or hemorrhagic rash.**

6. Hypertensive-hydrocephalic syndrome - in the first days of the disease (headache, vomiting, hyperreflexia, pyramidal signs);

7. Meningeal syndrome appears on the 2-3-d day , moderate, short-term, characterized by the fever and inflammatory changes in CSF;

8. CSF is transparent, colorless, pressure is increased, moderate lymphocytic pleocytosis (30 to 800 cells), protein content is normal or reduced, the level of glucose and chlorides are not changed;

9. CBC - leukopenia, nonexpressed neutrophilosis, with insignificant shift of the formula to the left, moderate increasing of ESR, at the beginning of the second week – eosinophilia.

10. Confirmation of the diagnosis is based on isolation of the virus from CSF, blood, stool, nasopharyngeal swabs;

11. Serologic investigation reveals increase of AB-titer in four times in paired serums (taken with an interval in 10-12 days);

12. Serouse meningitis is usually benign disease of moderate severity;

13. Feature of enteroviral meningitis is a tendency to recurrence: usually on the 20-28-th day of normal temperature the state is getting worse again (fever, increases pleocytosis in CSF).
Such recurrences may repeat, but relapsing forms are always severe.