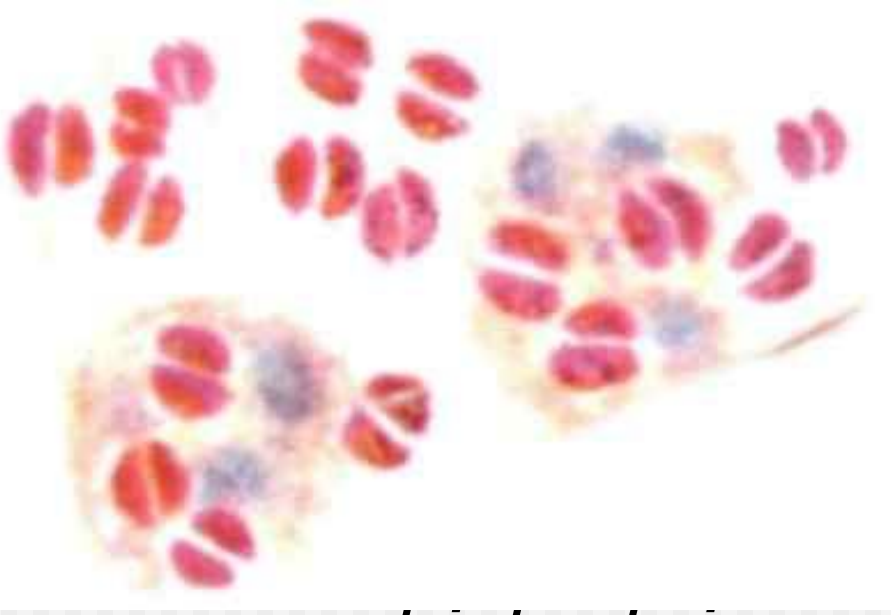
Meningococcal Infection

Etiology



- the ***causative agent*** is meningococcus *(Neisseria meningitidis).*
- *this microorganism has the form of a **diplococcus**, which stains well with aniline dyes, and is **gram-negative***
- *grows on media containing **human protein** (blood serum)*
- *very **unstable** and perishes rapidly outside the organism*
- ***several serotypes** of meningococ (A, B, C, D, Z, X, and Y) have been discovered*

Epidemiology

- the ***sources of infection*** are patient and carriers
- meningococcus ***expel*** the causative agent with the secretions from the nasopharynx and upper respiratory passages
- ***Infection is transmitted*** by the aerial-droplet route
- The ***susceptibility*** of man to meningococcal infection is slight: the susceptibility index does not exceed 0.5 %
- The meningococcal infection is characterized by ***periodic rises*** of the incidence every 10-15 year or longer

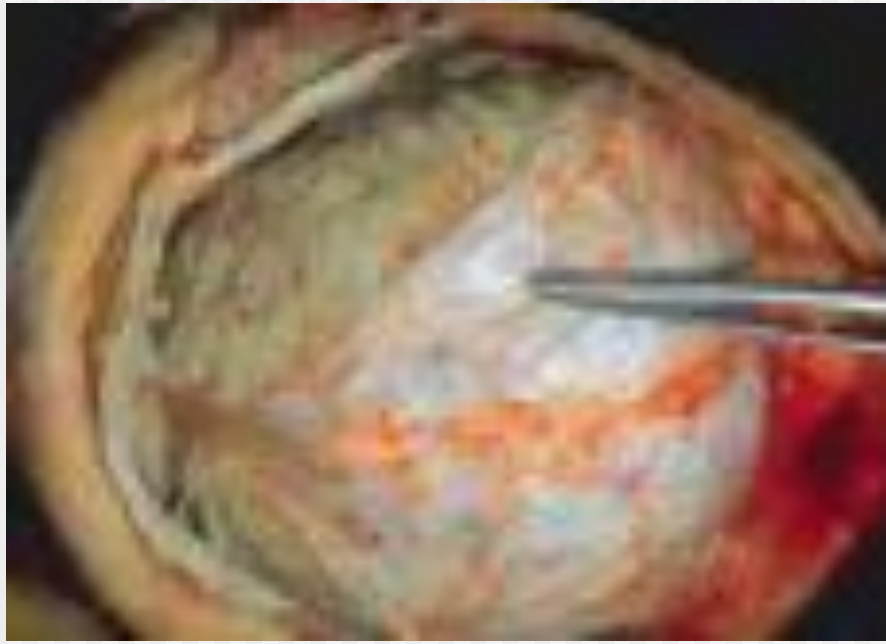
Pathogenesis and Pathology

- The ***portal of the infection*** entry is the nasopharyngeal mucous
- The ***carrier*** state develops frequently, while nasopharyngitis and generalized form (in 0.5-1 % of cases) occurs significantly less frequently
- The important role in mingococcemia belongs to marked ***intoxication with the endotoxin*** released during decomposition of the microbial bodies - microcirculation is thus affected to ***provoke thrombosis and extravasates***
- Necrosis in the adrenal glands with diffuse hemorrhages and decomposition of the glandular tissue - fulminating forms (***Waterhause-Friderichsen syndrome***)

Pathogenesis and Pathology

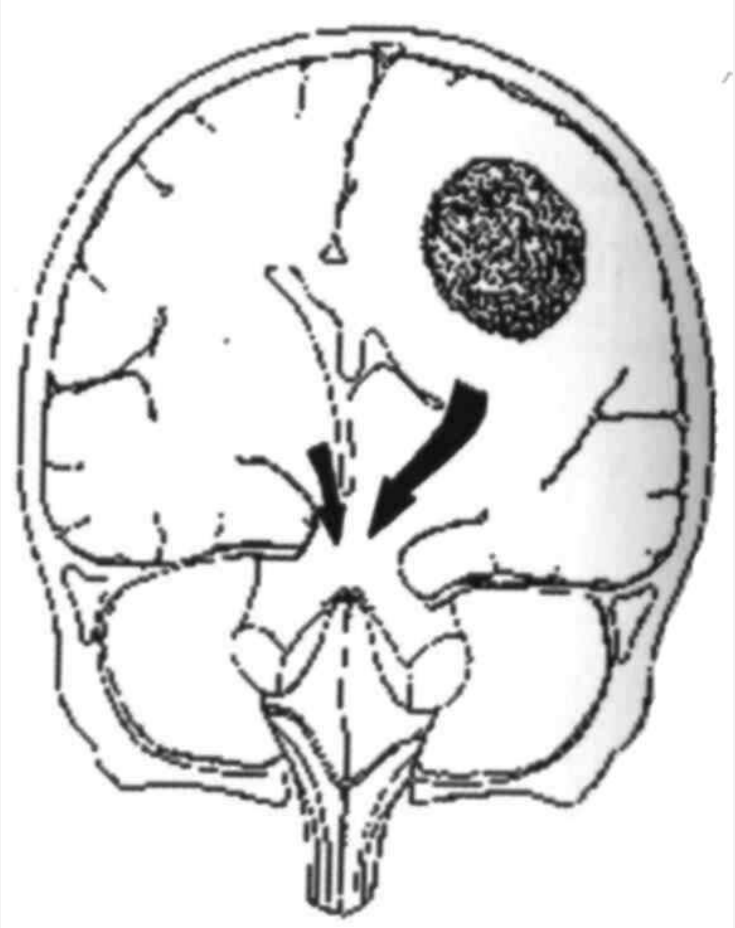
- ***Purulent meningitis*** develops due to the ingress of the meningococcus into the soft meninges of the brain and the spinal cord

Purulent exudates is particularly abundant in the base, and on the surface of the frontal and parietal lobes of the brain - ***"purulent cap"***



Pathogenesis and Pathology

- Acute swelling and edema of the brain can cause ***protrusion of the cerebellar tonsil*** into the great foramen



Classification

Location form:

- Nasopharyngitis;
- Carriers.

Generalized form

- Meningitis;
- Mingococcemia;
- Fulminating form;
- Meningitis+ mingococcemia.

Atypical form:

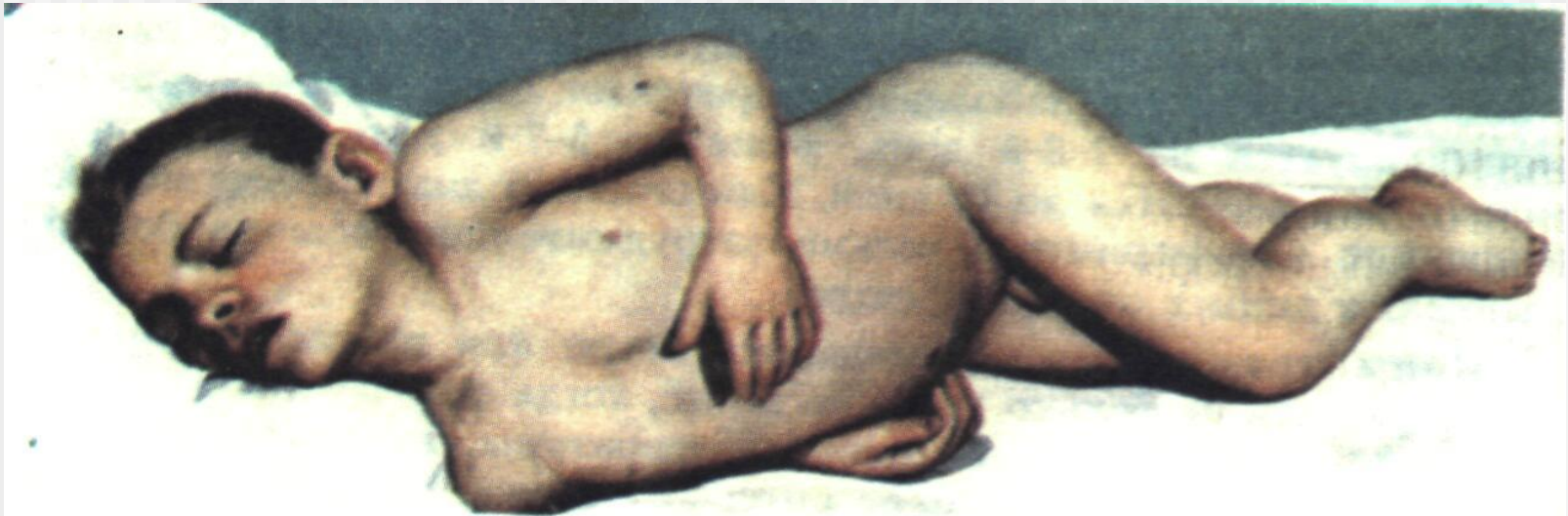
- Iridocyclochorioiditis;
- Pneumonia
- Endocarditic.

Nasopharyngitis

- headache, painful swallowing, subfebrile temperature
- hyperemia of the nasopharyngeal mucosa and hyperplasia of lymphoid nodes
- rhinitis with scanty discharge, and difficult nasal breathing

Meningitis

- The ***onset of the disease*** is usually violent, and a considerable ***elevation of temperature***; severe ***headache***, ***vertigo***, and ***vomiting***
- The ***patient's posture*** is lying on his side with head tossed back and legs flexed to the abdomen



Meningeal symptoms

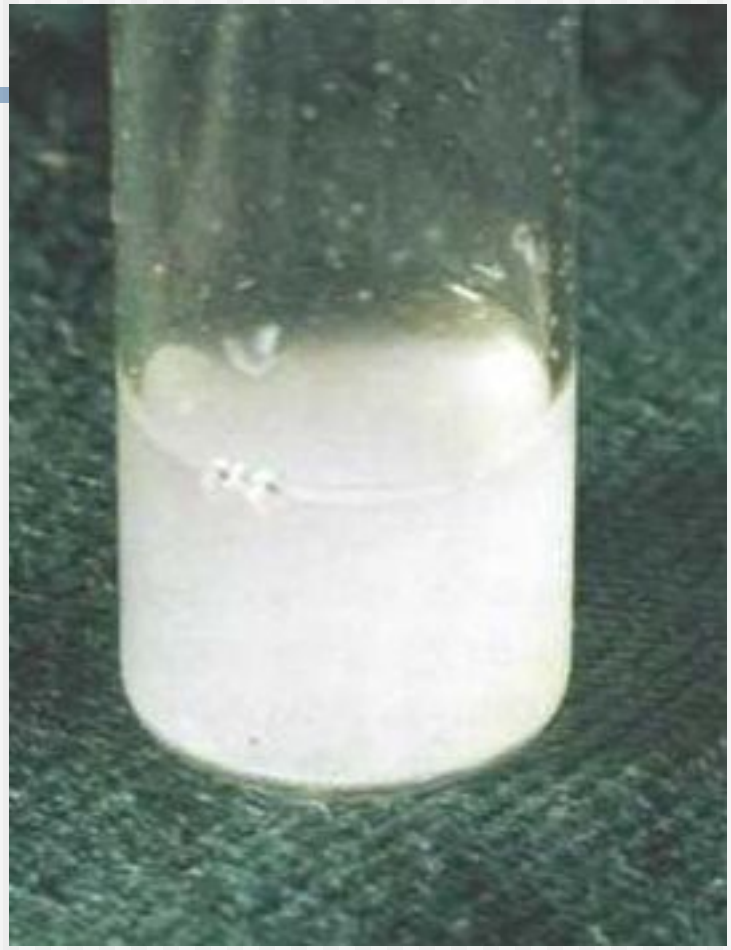
- ***hyperesthesia of the skin and increased sensitivity to light and sound***
- ***stiffness of the occipital muscles***
- ***Kernig's***
- ***Brudzinsky's***

Mental disturbances are also frequent (lethargy, drowsiness, etc.).

In young children ***clonik and tonic convulsions*** are not infrequent

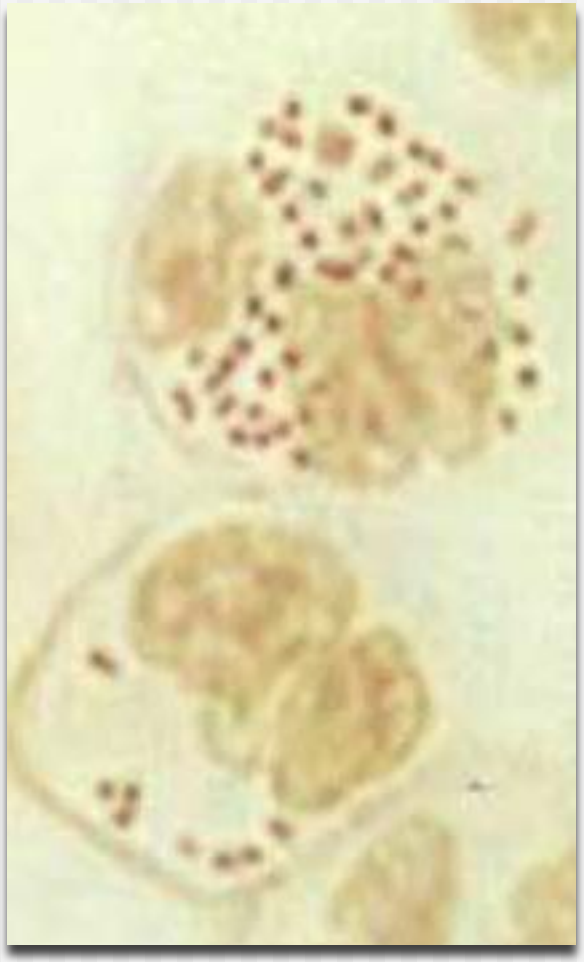
Spinal fluid

- increased pressure
- turbid and purulent
- neutrophilosis (from several hundreds to several thousands of cells per mm^3)
- considerable protein content (up to 1-2 g/l)
- sugar content is lowered



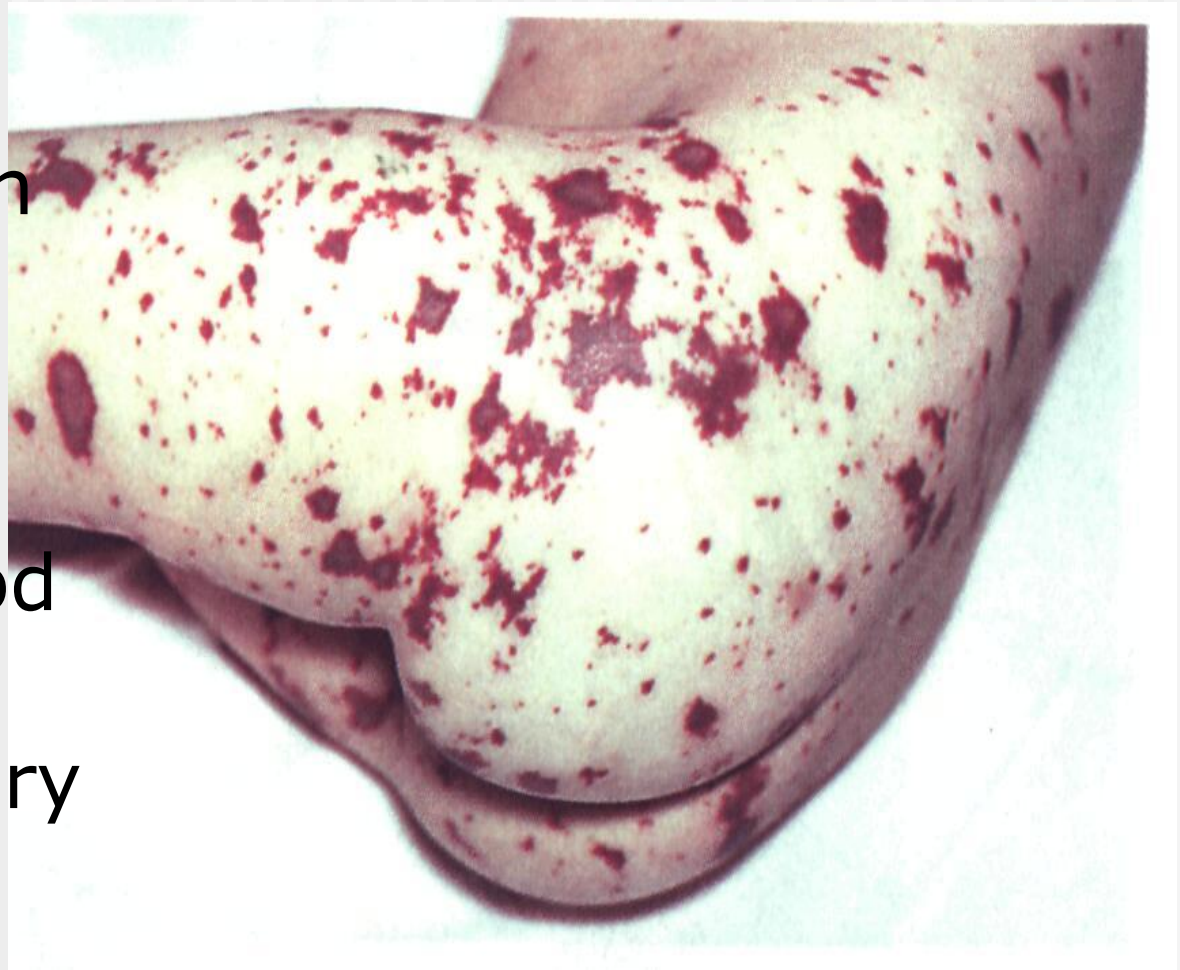
Blood

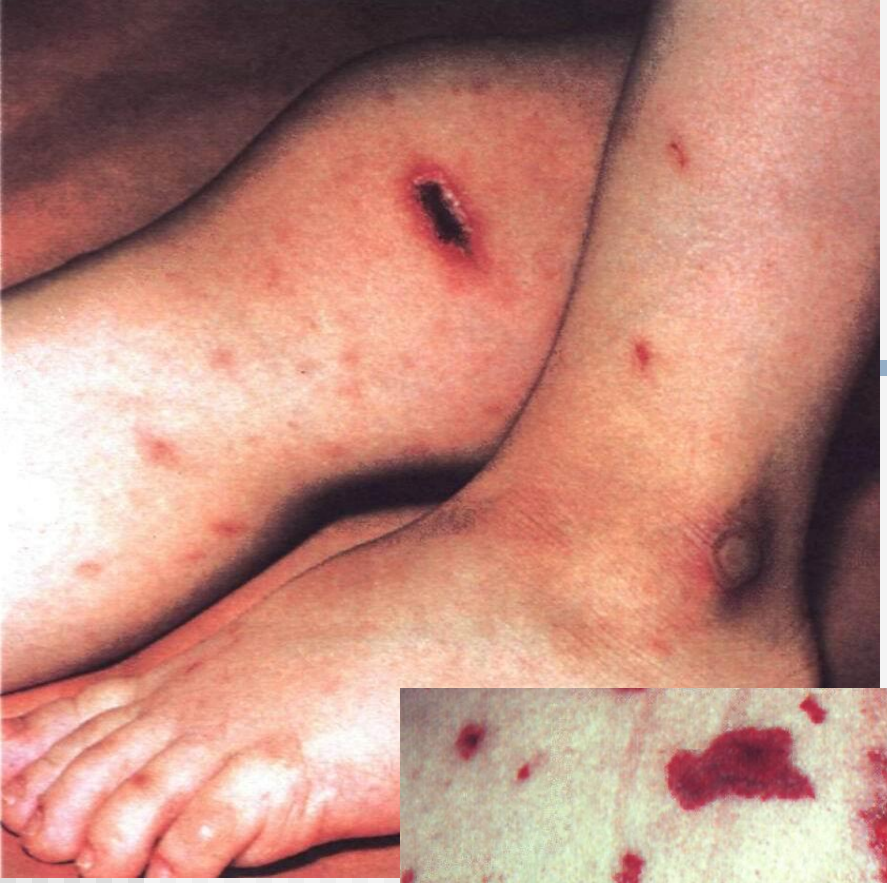
- leukocytosis (up to 20-40-10^9/1)
- neutrophilosis with a shift to the left
- aneosinophilia
- the ESR is considerably increased

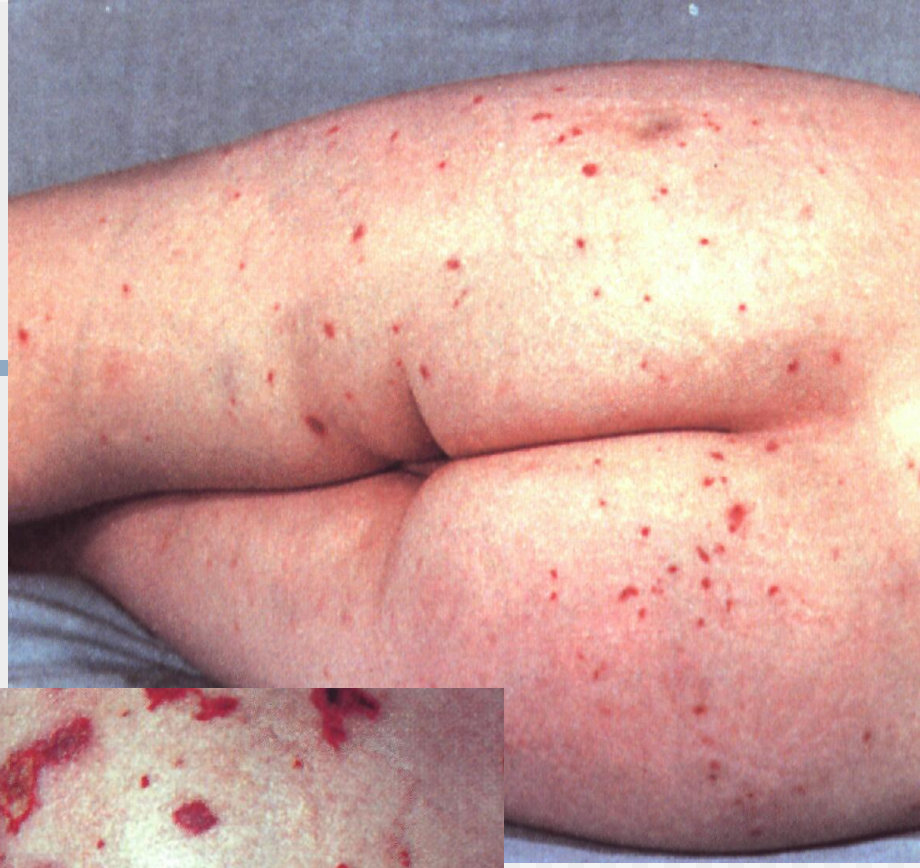


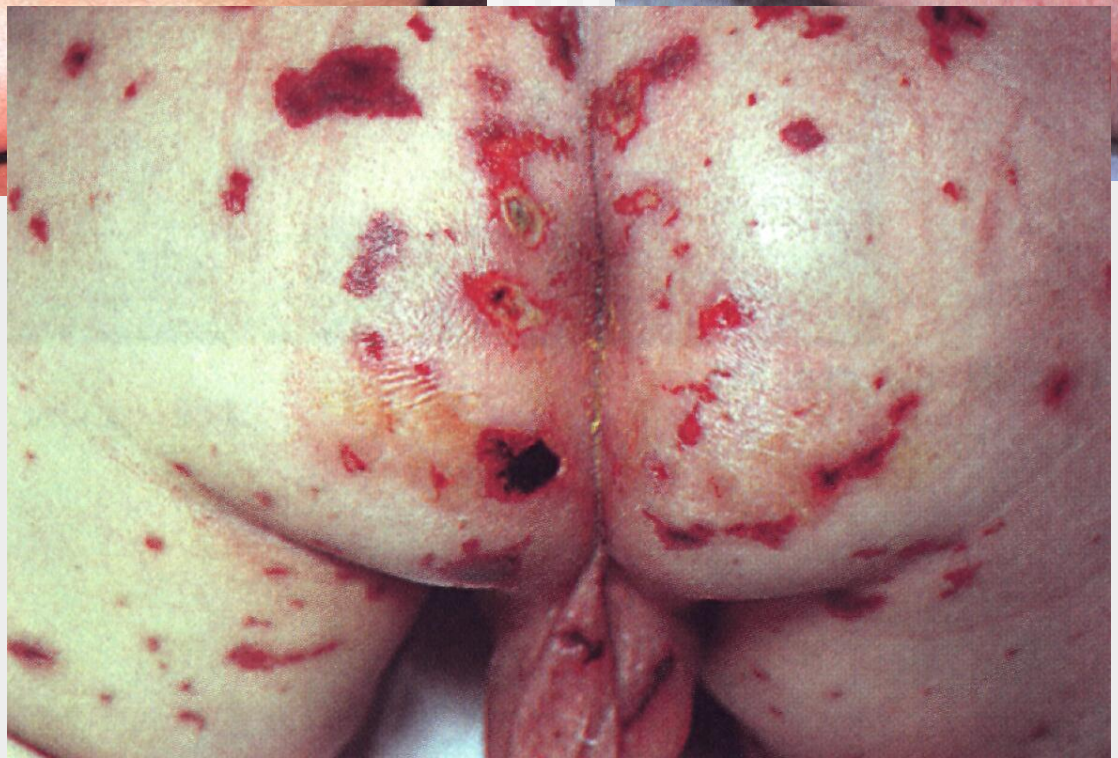
Meningococcemia

- The onset is acute and violent, with intermittent fever
- The rash is hemorrhagic satellite formations varying in size; they are hard on palpation and are often elevated
- Meningococcal are found in blood smears taken from the periphery of the lesions







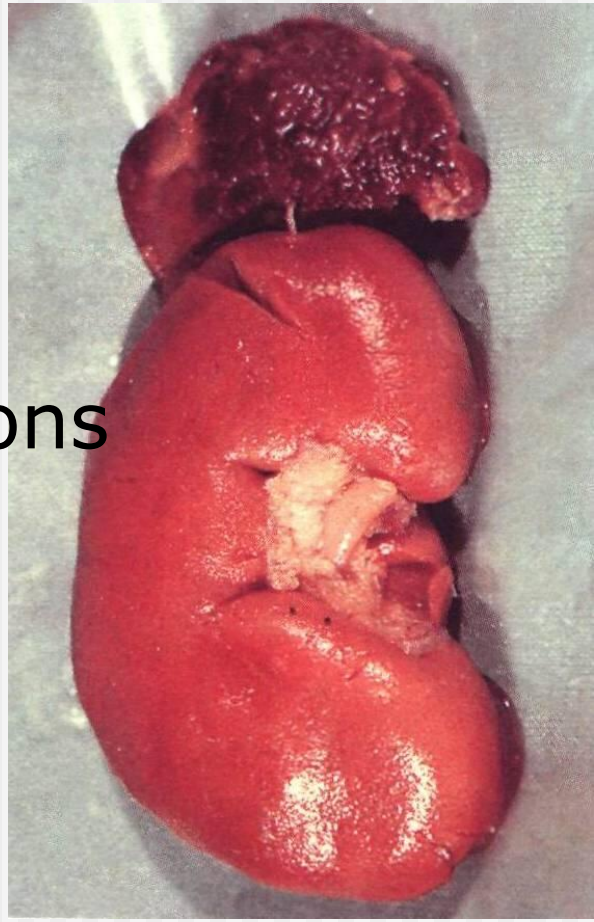


Hypertoxic (fulminating) **form**

- A sudden turbulent onset
- Severe toxemia (uncontrollable vomiting, convulsions, mental confusion, cardiovascular weakness)
- Meningeal symptoms are sharply pronounced
- Death usually ensues within 12 to 24 hours after the onset
- Swelling of the brain and protrusion of the cerebellar tonsils into the great foramen is one of the frequent causes of death

Waterhouse-Friderichsen syndrome

- Multiple petechiae and hemorrhage into the skin
- The arterial pressure falls progressively
- The pulse is rapid and hard
- Cyanosis, vomiting (often with blood) and convulsions
- The patient dies in 16-30 hours after the onset of the disease unless an urgent and effective therapy is given

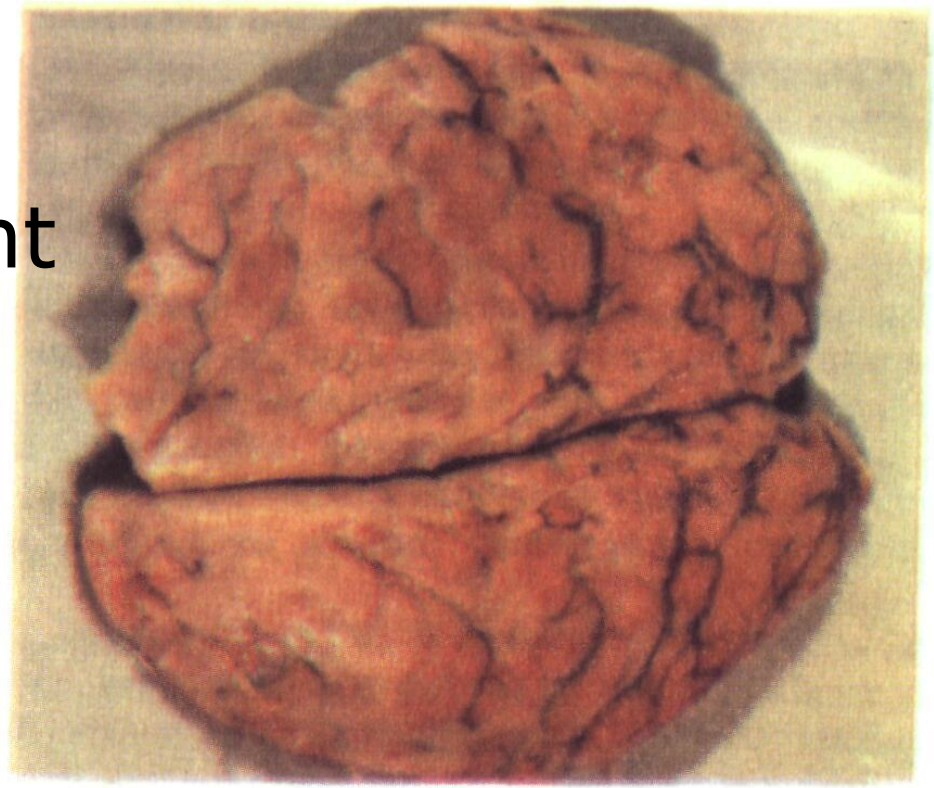


Features peculiar to meningitis in infants

- The disease is accompanied with high temperature, general restlessness, vomiting, and refusal to suckle
- Frequent dyspeptic disturbances
- Infants cry loudly
- Meningeal symptoms and red dermographism are often mild or absent
- Even with modern methods of treatment, mortality remains high

Complications

- Pneumonia,
- Purulent otitis
- Hydrocephalus
- The symptoms of which appeared already at the height of the disease
- Paralysis, paresis
- Asthenic syndrome, headache
- Various functional disorders



Diagnosis

- the ***clinical symptomatology*** and its course: acute onset and rapid development of meningeal symptoms
- The most important diagnostic aid is lumbar puncture and examination of the ***cerebrospinal fluid***

The diagnosis is undiscutable when meningococcus is detected by ***bacterioscopy*** or is found in a cerebrospinal fluid culture

Differential diagnosis

Tuberculosis meningitis

- starts gradually and is accompanied with moderate pyrexia
- anamnesis and the results of tuberculin tests
- the X-ray of the lungs
- cerebrospinal fluid is ***slightly opalescent;*** cell count is moderately increased due to an increase in the ***lymphocyte number; sugar and CL content is lowered; protein is elevate***

Differential diagnosis

Acute serous meningitis

- differs in the cerebrospinal fluid findings : complete transparency; moderately increased cell count due to a ***higher number of lymphocytes***; normal sugar content

Differential diagnosis

Meningeal form of poliomyelitis

- The cerebrospinal fluid is transparent
- A slight or moderately increased cell count and normal or slightly increased protein content (cellular-protein dissociation)
- Lymphocytes predominate among the cells

Differential diagnosis

Other purulent meningitis (staphylococcus, pneumococcus, Afanasyev-Pfeiffer bacillus, streptococcus)

- develops secondarily to purulent otitis, pneumonia, sepsis
- gram-positive cocci and diplococci are found in the cerebrospinal fluid

Differential diagnosis

Meningococcemia of thrombopenic purpura and hemorrhagic vasculitis

- ***meningococcemia*** is characterized by high temperature, pronounced intoxication, marked changes in the blood (hyperleukocytosis with the shift to the left); and typical hemorrhagic eruption
- Accurate diagnosis is established ***bacteriologically***

Prognosis

- ***Mortality*** from epidemic meningitis was very high (30 to 40 % on average)
- The worst outcome in meningitis is prognoses in cases with the ***Waterhouse-Frederickson syndrome*** and the hypertoxic clinical form

Etiotropic treatment

- ***Penicillin*** was first given dose of 300 000-400 000 units per kilogram of body weight at intervals of 3 to 4 hours. Treatment lasts for 8-10 days without reducing the dose
- ***Levomycetin*** sodium succinate can be given (100 mg/kg a day), ***ampicillin*** (150-200 mg/ kg a day), cephalosporins, oxacillin or methicillin are also recommended

Stopped antibiotic therapy need after sanayshin liquor: citosis is less then 100 cell of lymphocytes!

Pathogenetic treatment

- ***Toxicosis*** can be controlled by administration of large amounts of liquids electrolyte balance and osmotic pressure should be watched closely
- ***Dehydration*** therapy should be especially intensive in the presence of brain swelling
- ***Corticosteroids*** should be given simultaneously 5-10-15 mg/kg with septic shock

Prophylaxis

The following in an epidemic focus

- The ***patient*** is hospitalized and isolated to condition that the results of two bacteriological studies of the pharyngeal mucus are negative
- ***Contacts*** and ***carriers*** should be treated with rifampicini for 3 days as a prophylactic measure, the standard dose being given 3 times a day
- Terminal ***disinfection*** is carried out after isolation of the patient

Polysaccharide meningococcal vaccines have been recently developed in some countries

Acute Epidemic Poliomyelitis

Etiology

- the ***causative agent*** of ***polyomyelitis*** (Poliovirus hominis)
- a very small virus
- contains ***RNA***
- is ***very stable*** in the external environment, and is resistant to low temperatures and disinfection
- Three ***types of poliovirus*** (I, II, III) are known

Epidemiology

- ***Sources*** of infection - patients with clinically manifest poliomyelitis, persons suffering from atypical and abortive forms
- The ***infectivity*** of patients is greatest during the acute stage. Most are free of the virus in 15 to 20 days after an attack
- ***The mechanism of infection -*** of fecal mode of transmission
- ***Susceptibility*** to poliomyelitis is low (75 to 90 %)

Pathogenesis

- The most probable ***portal of entry*** of the infection - the pharyngeal lymphoid ring and the intestinal tract
- The poliomyelitis virus is isolated, as a rule, from lesions of the ***nervous system***
- The ***most pronounced pathological*** changes are in the ventral horns of the gray matter of the cervical and lumbar enlargements of the spinal cord
- The ***nerve cells*** undergo dystrophic necrotic changes, and perish

Clinical Manifestations

- The ***incubation period*** of poliomyelitis averages from 5 to 14 days; it may sometimes be as short as 2 to 4 days or as long as 35
- ***Four stages*** are distinguished in the course of the disease:

  a) initial (preparalytic),

  b) paralytic,

  c) restitution,

  d) the stage of residual phenomena

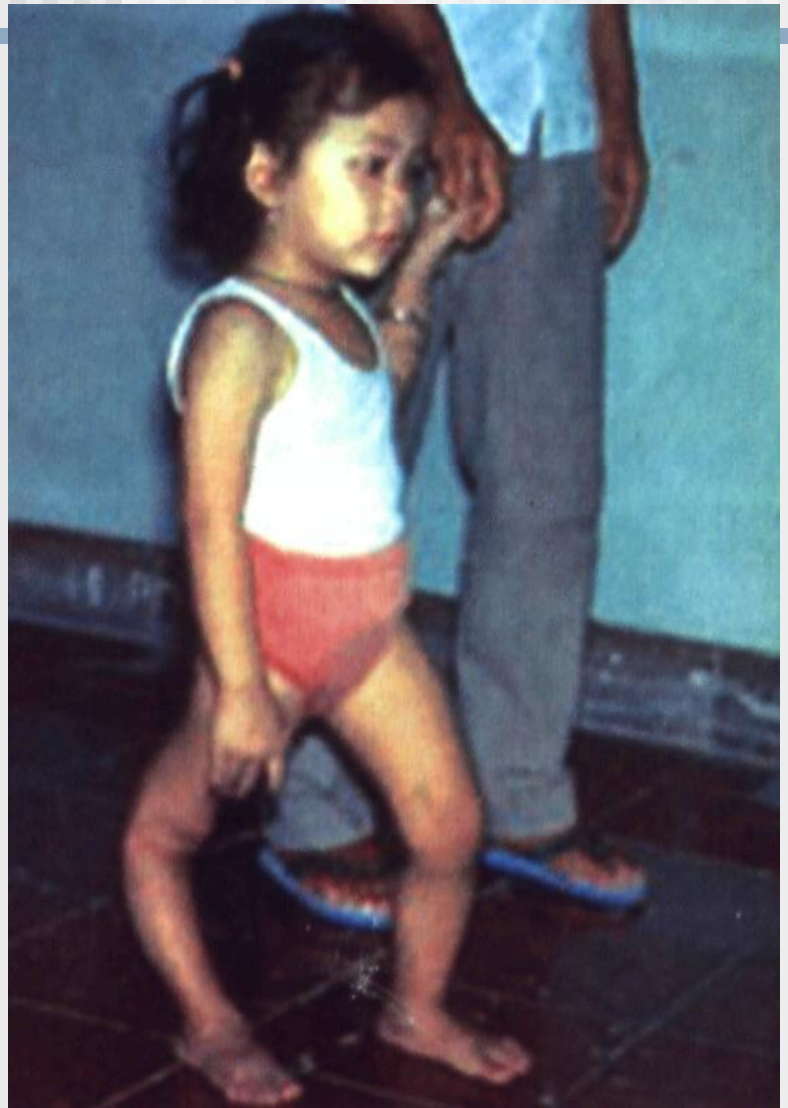
Preparalytic stage

- The disease ***starts*** acutely with a marked rise of temperature
- ***Catarrh*** of the upper respiratory tract and by ***gastrointestinal disturbances***
- General and local ***hyperhidrosis***
- Symptoms of irritation ***on the nervous system :*** headache, vomiting, adynamia, lassitude, drowsiness or insomnia, sometimes delirium, tremor, muscular jerking, and convulsions
- This stage usually ***lasts*** from 2 to 5 days

Paralytic stage

- The ***temperature falls*** at the end of the initial stage, and paresis and paralysis occur
- ***Paralysis*** usually suddenly; may wake up paralyses in the morning ***("morning paralysis")***
- Careful examination will have ***revealed hypotonia, muscular weakness***, and ***loss of reflexes***

Signs of damage of the peripheral neuron characterize



the paresis and paralysis in poliomyelitis:

- absence of tendon reflexes,
- cutaneous reflexes may also disappear,
- muscular appear one or two weeks after the onset of paralysis

Stage of **residual phenomena**

- The stage of ***residual phenomena*** is characterized by **stable** flaccid **paralysis**, **atrophy** of **definite muscular** groups, and **contractures** and deformities of the limbs and trunk

Clinical forms of poliomyelitis

paralytic poliomyelitis:

- a) spinal,
- b) bulbar,
- c) pontine,
- d) encephalitic

aparalytic poliomyelitis:

- visceral (or abortive)
- meningeal

Paralytic poliomyelitis

- The ***spinal*** form is characterized by flaccid paralysis of the limbs, trunk, neck and diaphragm
- The ***bulbar*** form, which is fraught with the greatest danger, is accompanied with swallowing, speech, and respiratory disturbances
- The ***pontine*** form is expressed in implication of the nucleus of the facial nerve with paresis of the facial muscles
- ***The encephalitic*** form is characterized by general cerebral phenomena and symptoms of focal lesions in the brain

Aparalytic poliomyelitis

- ***The visceral (or abortive)*** form shows symptoms of the initial stage of poliomyelitis. There are also signs of irritation of the nervous system. Sometimes there are no changes in the cerebrospinal fluid indicative of poliomyelitis
- In the ***meningeal*** form there are the same signs as in the visceral, with meningeal symptoms in addition. Findings in the cerebrospinal fluid - elevation of cell count (lymphocytes) and a normal or slightly elevated protein content

Diagnosis

- Rapid investigation suspected cases critical to identifying possible wild poliovirus transmission
- ***Clinical case definition***

  Acute onset of a flaccid paralysis of one or more limbs with decreased or absent tendon reflexes in the affected limbs, without other apparent cause, and without sensory or cognitive loss.

Laboratory Diagnosis

- **Viral Isolation**

  isolate wild polio virus from stool or pharynx;

  do ***genetic*** "finger printing" of virus to see wild type and where from

- **Serology**

  neutralizing antibodies: early and may be high

  by the time the patient is hospitalized

  may not see 4 fold rise in titer

Treatment

- ***NO curative treatment***
- Supportive care:

aseptic meningitis- fluids, acetomenophen,
rest until fever improves,
paralysis- pain medications, +/-ventilator,
manage muscle spasms, treat 2° infection,
longer term –physiotherapy &
occupational therapy

Prophylaxis

- ***Isolation of poliomyelitis patient and suspected cases –*** hospitalization in special departments is obligatory
- After the patient is isolated (for 21 days from the onset of the disease) ***final disinfections*** is performed in his swelling
- ***Contacts*** are observed for 20 days after isolation of the patient
- **Active immunization** - with pertussis-diphtheria-tetanus vaccine beginning from 3 months of age 3 times with 30 days