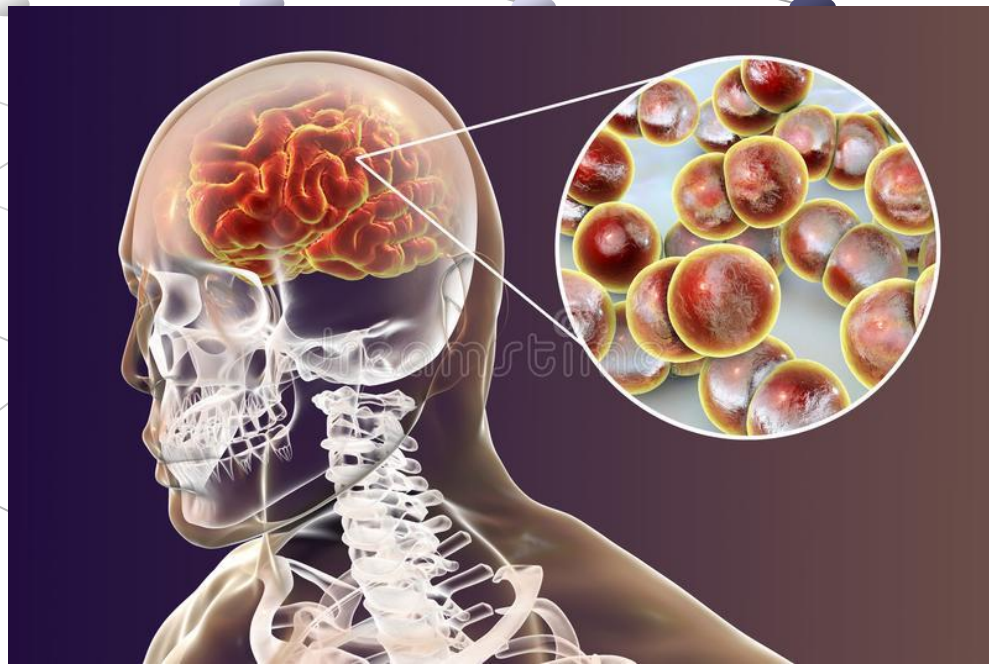
Meningococcal infection



Meningococcal disease - an acute infectious disease caused by meningococcus Neisseria meningitidis, with droplets (aerosol) mechanism of transmission; clinically characterized by lesions of the

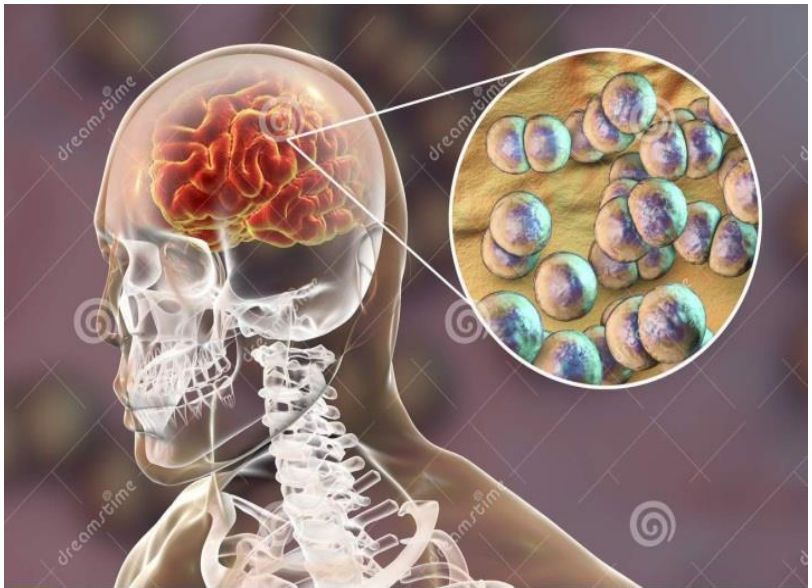
mucous membrane of nose and throat (nasopharyngitis)

generalization in the form of specific septicemia (meningococcemia)

inflammation of the meninges (meningitis).

Meningococcal disease is widespread and has a severe, adverse outcomes in inadequate therapy.

Meningococcus is one of the most common cause of bacterial meningitis in children and adults. Before vaccination it was the leading cause of bacterial meningitis among children in Europe. Infectious diseases tend to spread quickly wherever large groups of people gather together.



Meningococcal meningitis (*International Classification of Disease-9* [ICD-9] code: 036.0) has been recognized as a serious problem for almost 200 years.

EPIDEMIOLOGY

The geography of meningococcal infection is characterized by a very wide variability in the intensity of the development of the epidemiological process.

According to WHO, meningococcal infection is registered in more than 155 countries of the world. In a number of territories, "meningitis belts" have formed - zones of high incidence. An increase in the incidence was registered in the countries of the European continent - Belgium, Greece, Spain, Norway, Slovenia.

The incidence is especially high in some African countries - sub-Saharan Africa (Chad, Niger, Nigeria, Sudan) - 40-50 times higher than in European countries. But after successful use of vaccination it has strike tendency of decrease now.

Neisseria meningitidis **is carried, usually harmlessly, in the nose and throat of around 3-10% of the population.**

These are healthy "carriers". The bacteria are passed from person-to-person by close prolonged contact (family contact, cazarms, cambus et al.). Cigarette smoking, both active and passive, appears to increase the risk of a person developing meningococcal disease. Contact with saliva from the front of the mouth (for example, from sharing drinks or cigarettes) has not been shown to cause meningococcal disease.

EPIDEMIOLOGY

Meningococcal disease occurs worldwide as isolated (sporadic) cases, institution- or communitybased outbreaks, and large epidemics. Despite effective antibiotics and partially effective vaccines, N. meningitidis is still a leading global cause of meningitis and rapidly fatal sepsis, often in otherwise-healthy individuals.

N. meningitidis is unique among the major bacterial agents of meningitis in that it causes epidemic as well as endemic (sporadic) disease. In all, 300,000–500,000 cases of meningococcal disease occur worldwide each year—numbers that frequently are increased by large epidemics.

The annual incidence of meningococcal disease is 1–2 cases per 100,000 population for sporadic disease, 5–10 cases per 100,000 for hypersporadic disease (localized outbreaks and case clusters), and 10–>1000 cases per 100,000 for pandemic and epidemic disease (e.g., serogroup A epidemics).

The relevance of meningococcal infection is determined by:

- ♦ ease of spread of the disease,
- ♦ mainly by airborne droplets - when coughing, sneezing, talking,
- with a long and close communication;
- ♦ primary symptoms of meningococcal infection are often similar to those of other acute respiratory infections (ARI), which sometimes makes it difficult to diagnose the disease;
- ♦high prevalence of patients with a generalized form;
- ♦severe clinical development of the disease;
- ♦ sufficiently high mortality;

♦ the danger of the disease is that can develop in a matter of hours and even minutes, the so-called "lightning-fast" forms of the disease, and it is not always possible to save the patient;

- ♦ More common in children under 3 years of age.

Frequent number of diseases occurs in the winter-spring period (February-April). During the period of epidemic rise, an increase in the incidence is observed from November to December.

Factors affecting seasonality are climatic conditions (sudden fluctuations in temperature, high humidity), changes in the nature of communication between people in winter (prolonged stay in closed rooms, insufficient ventilation, etc.). Periodically, in 10–15 years, there is an epidemic increase in the frequency of this infection.

The causative agent of meningococcal disease - Neisseria meningitidis.

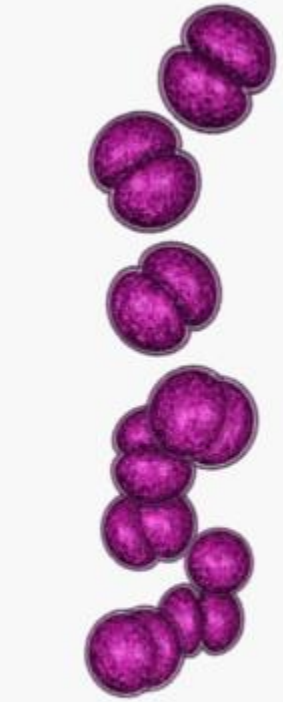
It is a gram-negative diplococcus,
motionless,
flagella and capsules has not,
does not form spores.

Cultured in a medium containing human or animal protein.

Optimum growth temperature - 37 ° C.

Aerobic and facultative anaerobic

with a “kidney” or “coffee-bean” shape.

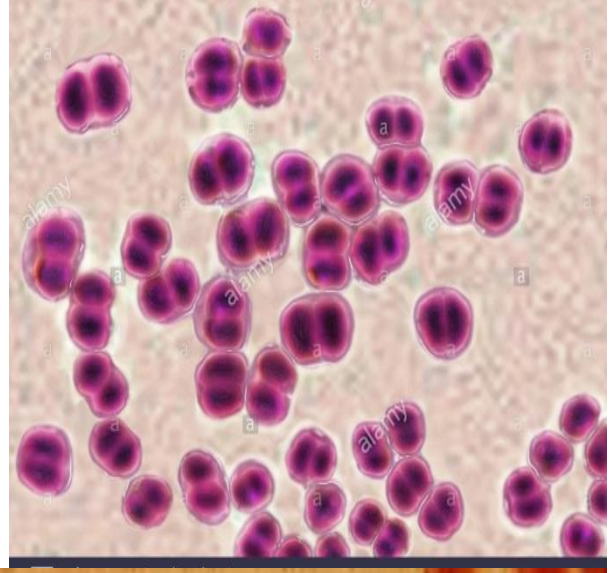


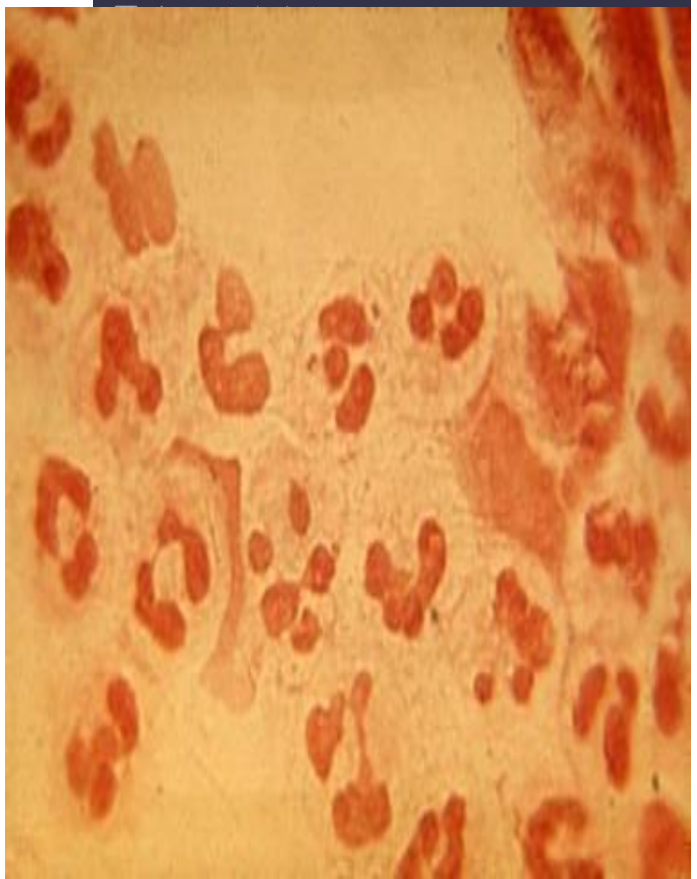
Neisseria meningitidis

Neisseria meningitidis

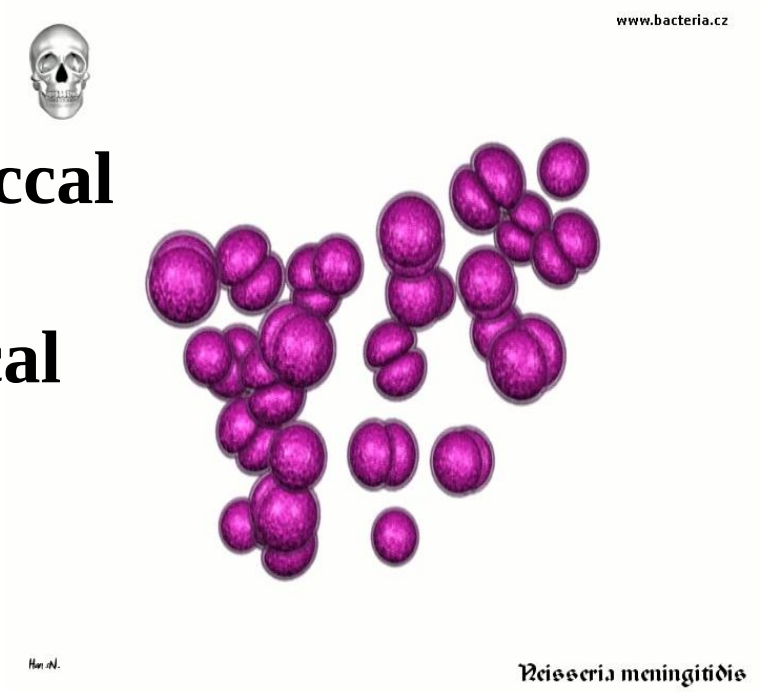
has a three-layer wall, the outer membrane of the cell wall is represented by a polysaccharide.
When it is destroyed, endotoxin is released - lipopolysaccharide, which is the main factor of pathogenicity.
According to the composition of capillary polysaccharides, 12 serogroups of menigococci are distinguished:
A, B, C, D, E, H, I, K, L, W-135, X, Y, and Z.

Dominant serogroups: A,B,C that cause generalized forms of the disease





More than 99% of meningococcal infections are caused by serogroups A, B, C, 29E, or W-135.
In Europe and the America, serogroup B is the predominant agent causing meningococcal disease, followed in frequency by serogroup C.
Historically, serogroup A was the main cause of epidemic meningococcal disease globally, and it is still the predominant cause of meningococcal meningitis in Africa and Asia.



Neisseria meningitidis **grows best on enriched media, such as Mueller-Hinton or chocolate agar, at 37°C and in an atmosphere of 5-10% carbon dioxide.**

Neisseria meningitidis on chocolate agar plate. :

Epidemiology

- ***Routes of transmission***
  - (1) Respiratory tract:
  - (2) Close contact:

cough/sneeze

bosoming/kiss/breast-feed

Meningococcal meningitis

Epidemiology

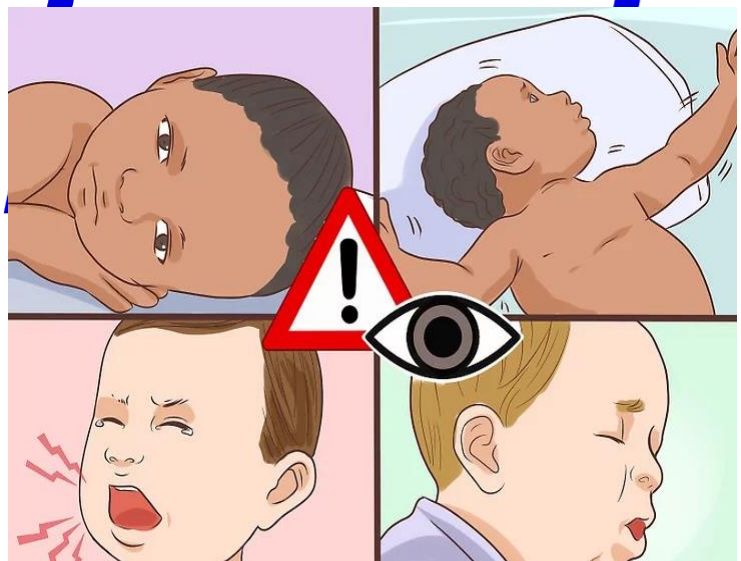
Source of infection

Sick person

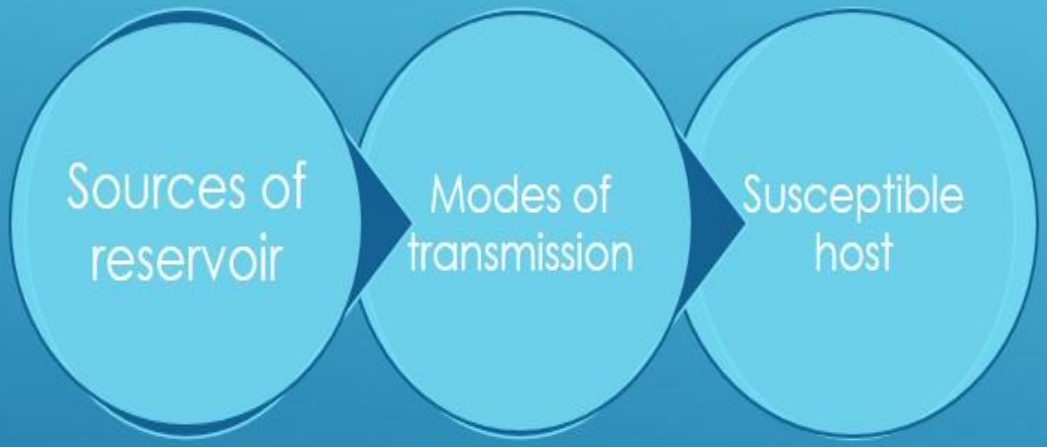
Most contagious at the onset of the disease, especially when there are catarrhal symptoms in the nasopharynx

Carriers

Healthy carriers without acute inflammatory symptoms of nasopharynx are less dangerous, but their number is many times higher than the number of patients



Dynamics of disease transmission



Susceptibility to meningococcus is low. The contagious index is 10-15%

In the pathogenesis of MI, 3 factors play a role:

- Causative agent
- Endotoxin
- Allergic factor

Pathogenesis of bacterial meningitis:

Nasopharynx

↓

Nasopharyngeal colonisation *(in epithelial cells)*

↓

Local invasion into intravascular space

bacteria transported across epithelial cells in membrane bound vacuoles
OR by creating separations in apical tight junctions

↓

Bacteremia

(avoid phagocytosis due to presence of polysaccharide capsule)

↓

Reach choroid plexus / Adhere to cerebral capillary endothelium

↓

Bacteria gain access to CSF

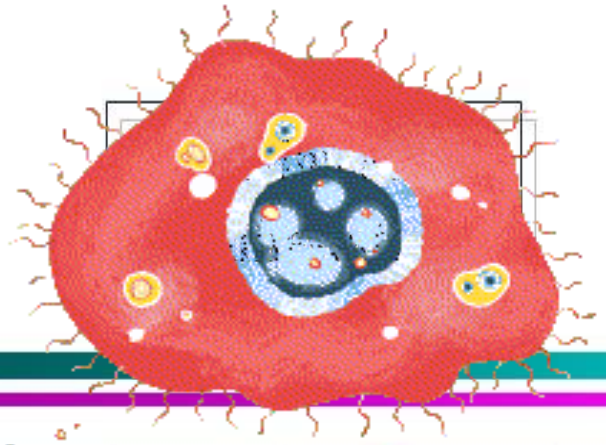
↓

Rapid multiplication in CSF

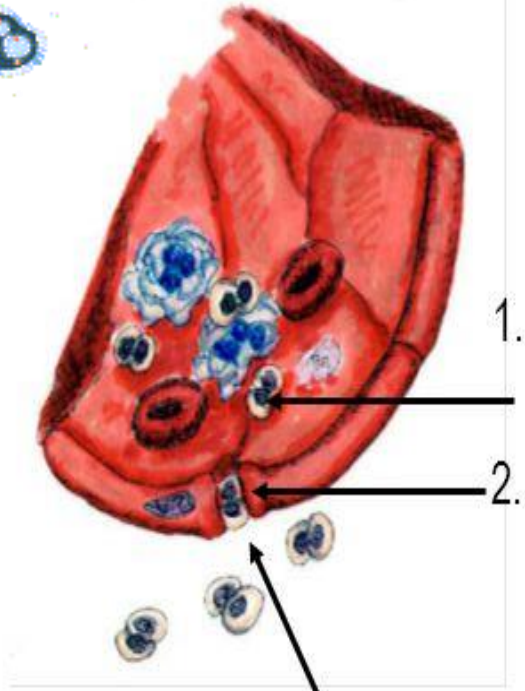
↓

Lysis of bacteria

...contd



Pathogenesis - Meningitis



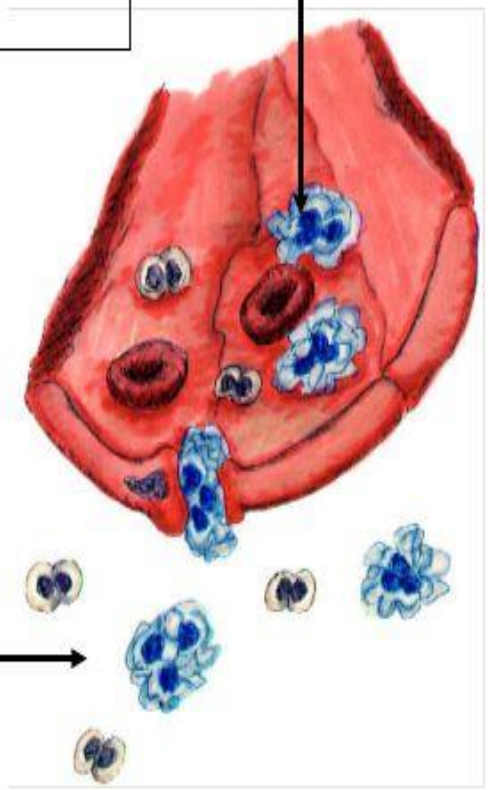
4. Neutrophils summoned;
   attach to endothelium

1. Meningococci
   release endotoxins

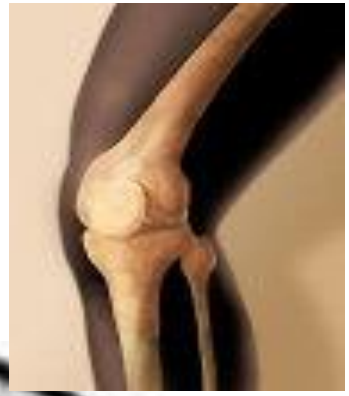
2. Cytokines
   summoned;
   endothelial cell
   inflamed

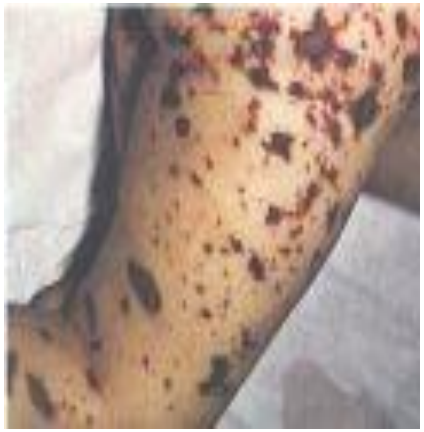
3. Blood Brain
   Barrier disrupted

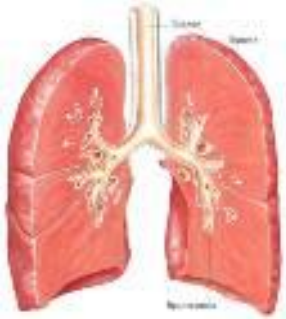
5. Neutrophils enter brain;
   secrete inflammatory factors;
   further BBB disruption



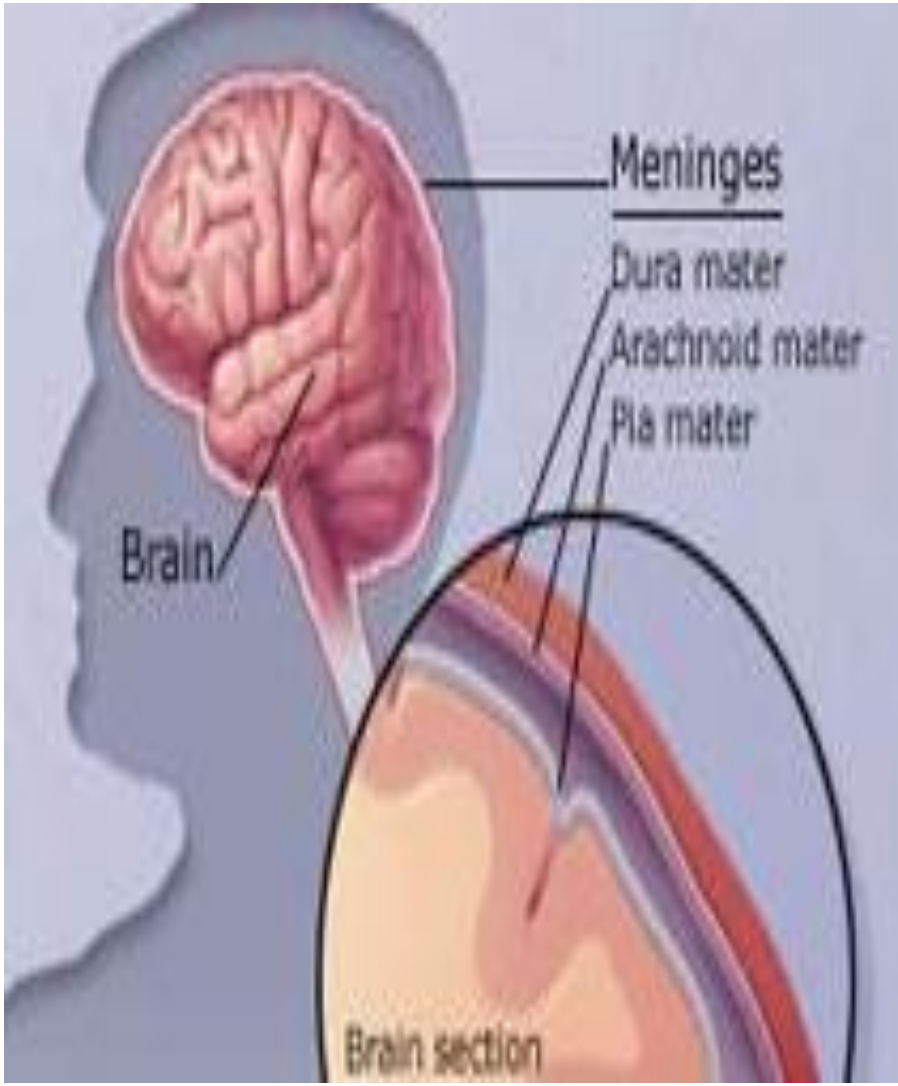
The bloodstream of meningococcus is carried into various organs and tissues: skin, joints, adrenal glands, choroid, kidney, endocardium, lungs, etc.







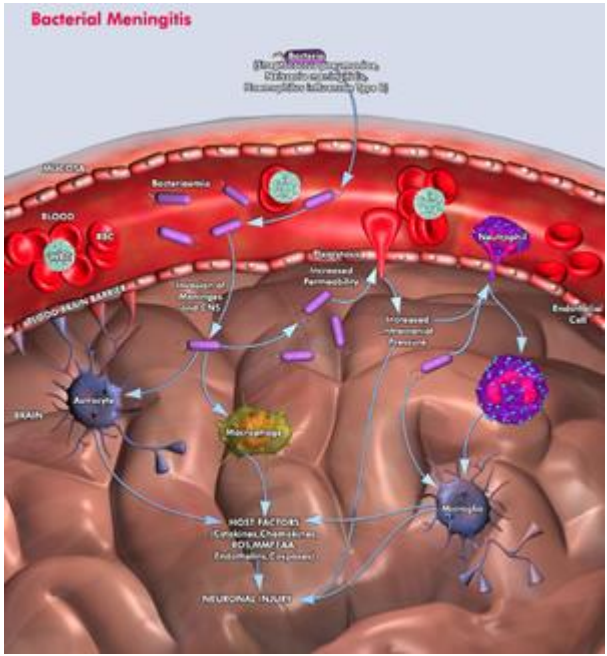
Meninigococcus can overcome blood brain barrier and cause brain damage shells and brain substances with development clinical picture of purulent meningitis or meningoencephalitis



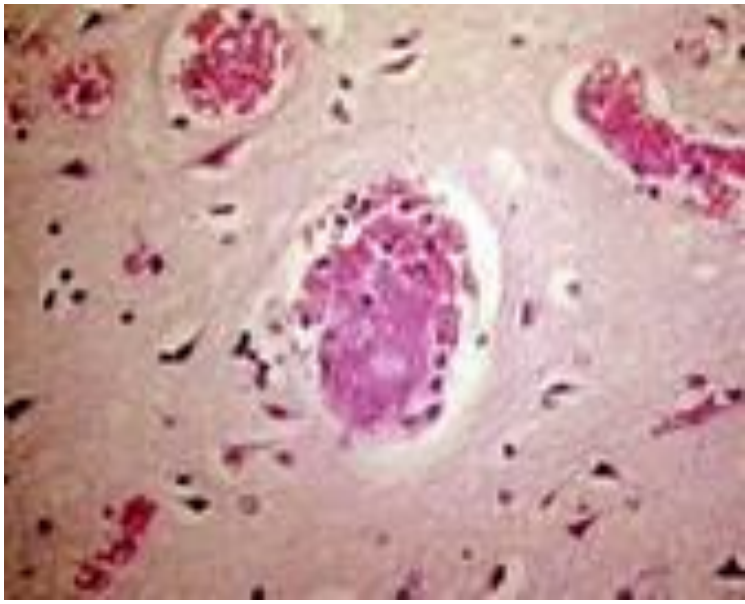
In the pathogenesis of generalized forms meningococcal infection - meningococcemia and meningitis along with meningococcus plays a big role endotoxin released in large quantity at death of meningococci.

Meningococcal endotoxin - it is a strong vascular poison.

When exposed to it vascular endothelium arise microcirculatory disturbances (capillary spasm, violation of their permeability

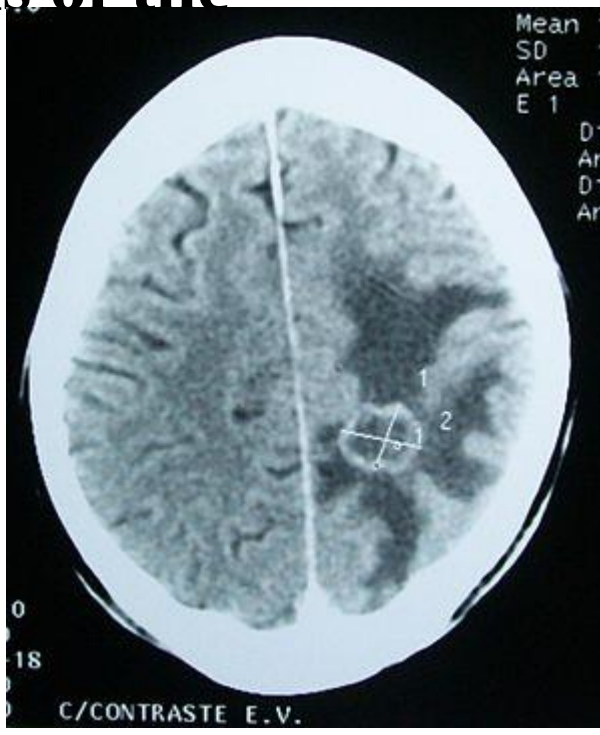


Changes in hemocoagulation develop according to thrombohemorrhagic type that leads to disseminated intravascular coagulation of blood with the formation of a huge number of bacterial blood clots in small arterioles and the development of consumption coagulopathy, which results in extensive hemorrhages in the skin and internal organs, including the kidneys, adrenal glands, the substance of the head brain, myocardium, etc.

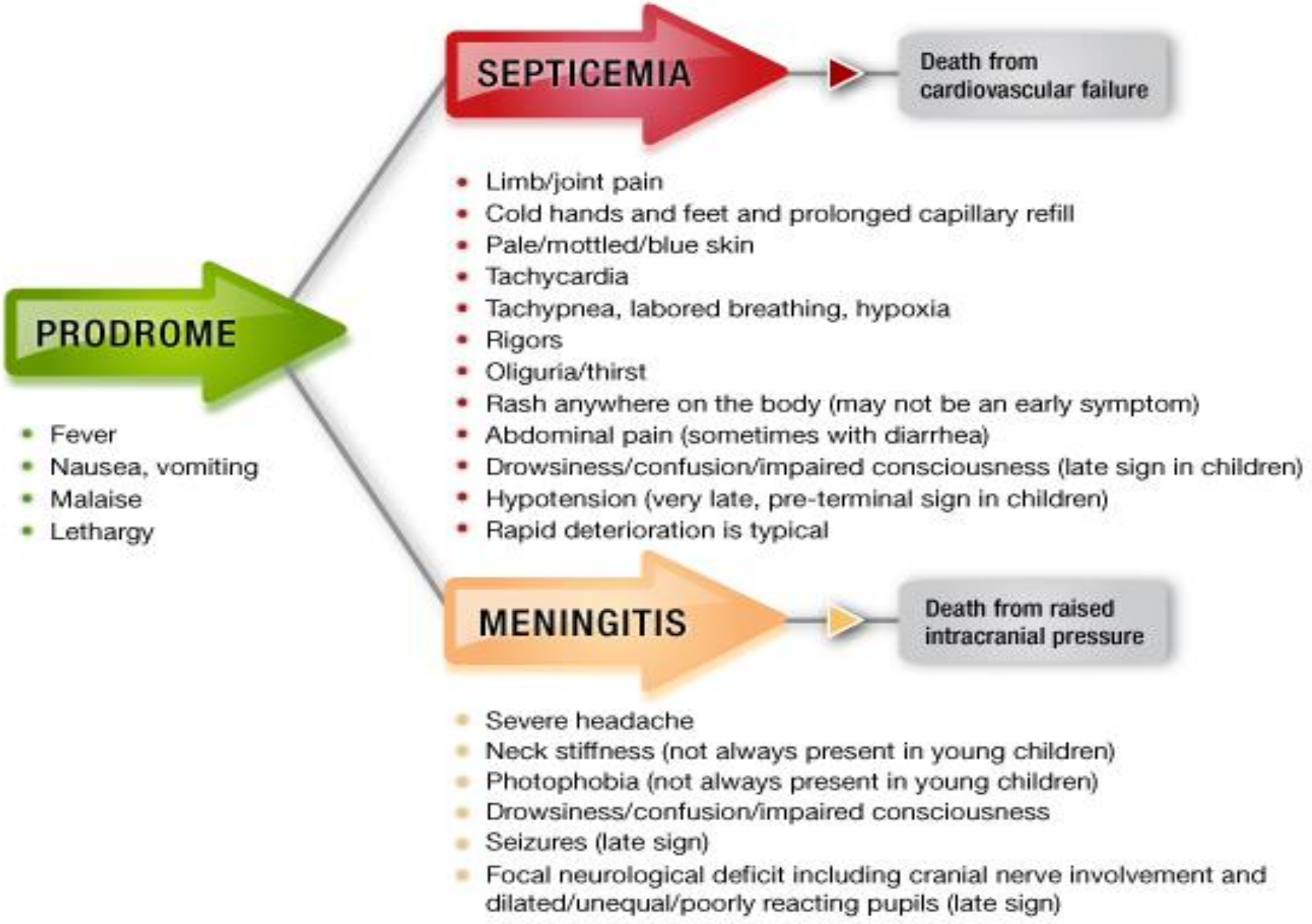


The consequence of endotoxemia, hemodynamic and metabolic abnormalities may be acute swelling and swelling of the brain.

As a result of cerebral hypertension possible inclination of the tonsils cerebellum in large occipital foramen compression of the medulla oblongata, and therefore death can occur from paralysis of the respiratory center.



Consider meningococcal disease in patients who present with the following symptoms and signs



Order in which the symptoms appear may vary. Some symptoms may be absent.

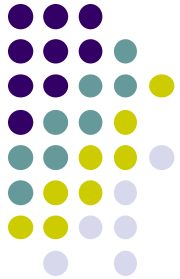
Clinically infectious toxic shock occurs with

severe hemodynamic disorders

and acute adrenal insufficiency syndrome

I. Localized forms:

1. meningococcal nasopharyngitis
2. Carriage of menigococcus



II. Generalized forms:

1. meningococcemia
2. purulent meningitis
3. purulent meningoencephalitis
4. combined form (meningitis with meningococcemia)

III.Rare forms:

-arthritis

-myocarditis

-osteomyelitis

- iridocyclitis

Meningococcal nasopharyngitis

is one of the local forms of infection (as carriage also).

Common manifestation is

not high fever

symptoms of cold,

thouthrout. It could be hyperplasia of lymphoid pholiculles at the back surface of pharyngs.

Diagnosis confirms bacteriologically.

Meningococcal nasopharyngitis has significal role in spreading meningococcal infection.

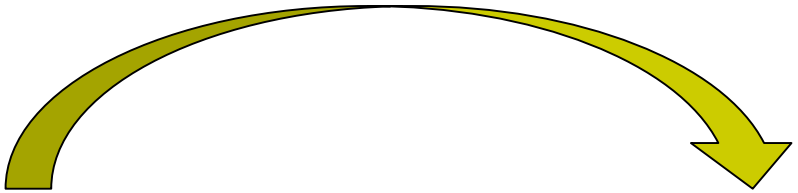
Meningococcal meningitis

epidemic cerebrospinal meningitis - obsolete., Russian.;
meningitis cerebrospinalis epideinica - Lat .;
cerebrospinal fever - Engl .;
epidemische cerebrospinale Meningitis - therein;
meningite epidemique - fr .;
meningitis epidemica - icp.

Meningitis and meningococcemia

are the most important variants of meningococcal diseases.

Clinical diagnostic of meningitis is usually based on the most typical symptoms.



The symptoms include:

- **sudden onset,**
- **fever,**
- **headache (without effect of common used analgetics),**
- **stiff neck.**

Some authors prefer to choose

“meningococcal triada ”

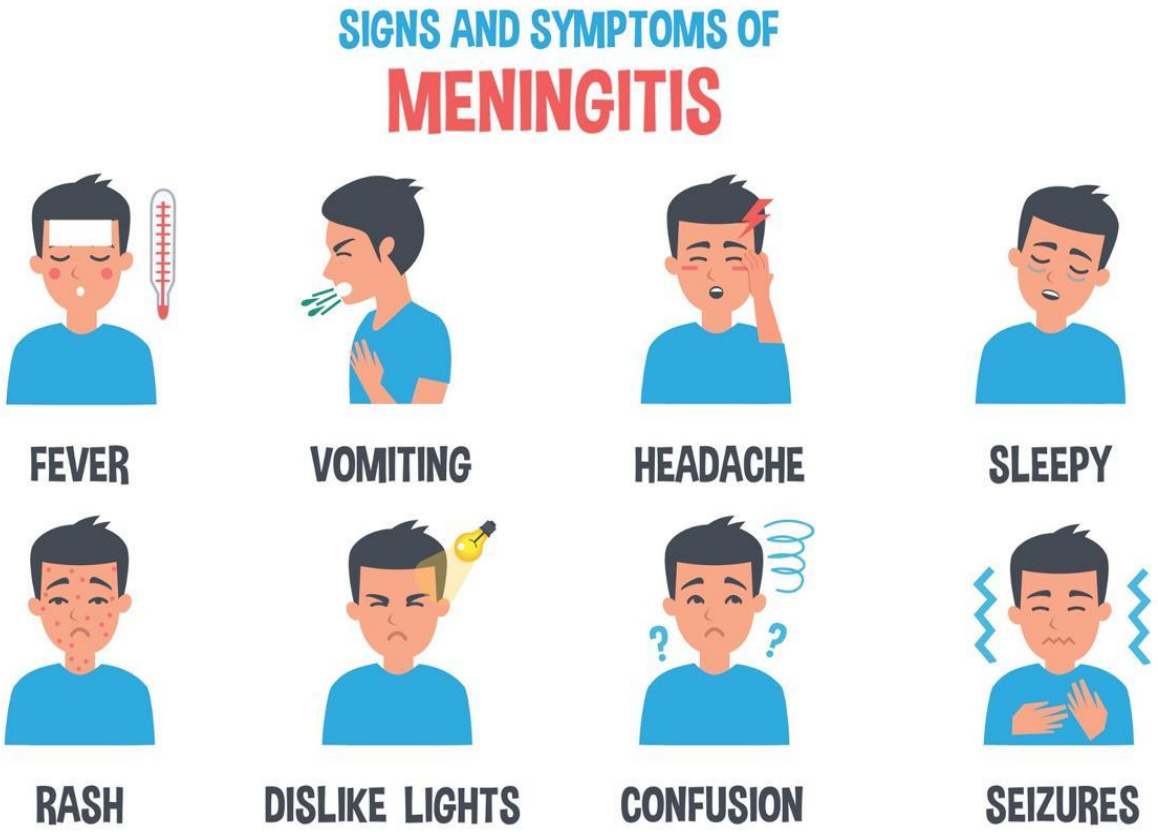
fever

headache

vomiting

There are often additional symptoms, such as

weakness, confusion, hyperstesia, photophobia et al.

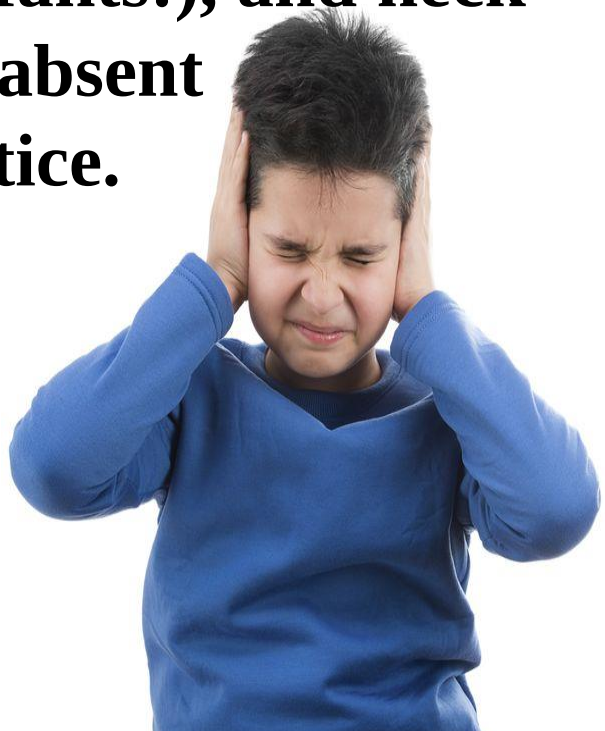


The symptoms of meningococcal meningitis can appear very quick or over few days.

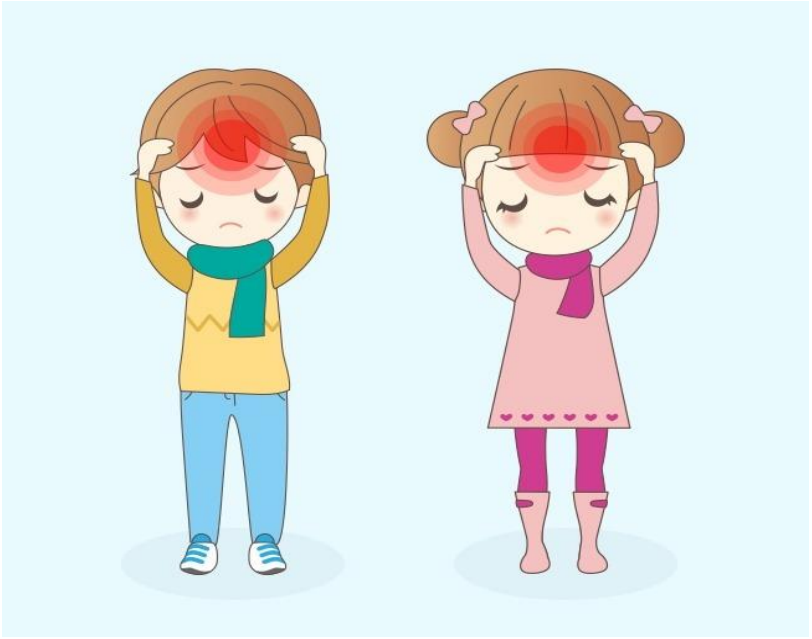
Typically they develop within 3-7 days after exposure.

In infants, it is sometimes very difficult to find classic symptoms of meningitis. It could be fever and usually vomiting.

But headache (infants!), and neck stiffness may be absent or difficult to notice.



Meningeal signs (stiffness neck,
Brudzinsky and Kernig symptoms and others) are of great diagnostic importance.



But they fluctuate because of the patient's reactivity, so can depend of age, immunologic status, physical conditions.

Meningococcemia
(Meningococcal Septicemia)

is the most severe variant of meningococcal infection.

In fatal cases, deaths can occur in as little as a few hours (fulminant meningococcemia).

During meningococcal septicemia, the bacteria enter the bloodstream and multiply, cause DIC, damage the walls of the blood vessels and causing bleeding into the skin and inner organs.

The main sign is sudden outbreak of fever and increasing of intoxication.

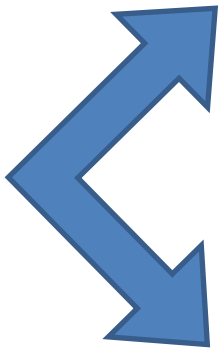
The signs of intoxication are mostly severe, but in early period of the disease, the status of the patient could be compensated.

The most important symptom of meningococcemia is hemorrahic rash, which usually appears in the first 4-15 hours of the disease.

Other symptoms may include:

- **fatigue,**
- **vomiting,**
- **cold hands and feet,**
- **severe aches or pain in the muscles, joints, chest or abdomen,**
- **sometimes diarrhea,**
- **in the later stages, a dark purple rash.**
- **prostration is common.**

The main complications of meningococcemia are

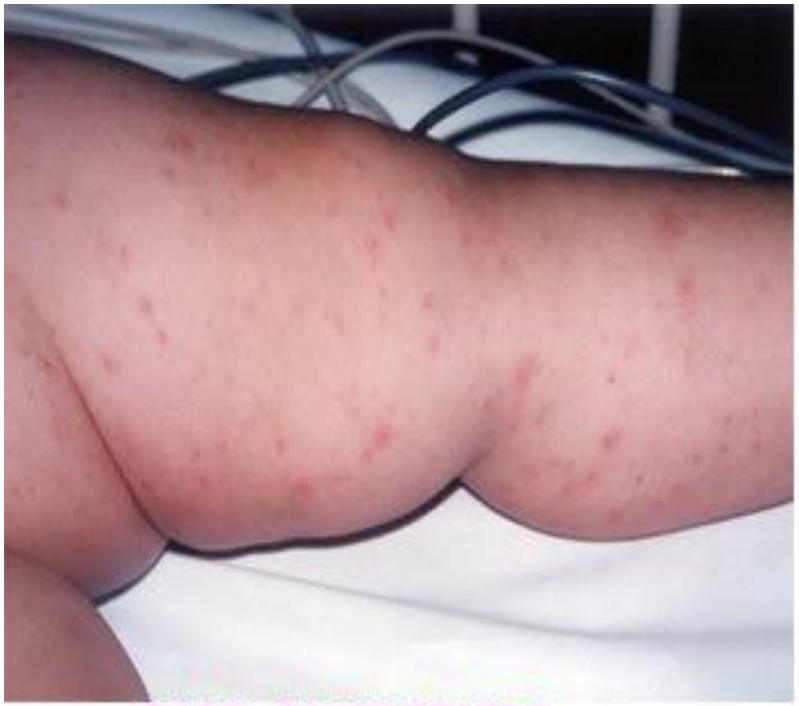


septic shock

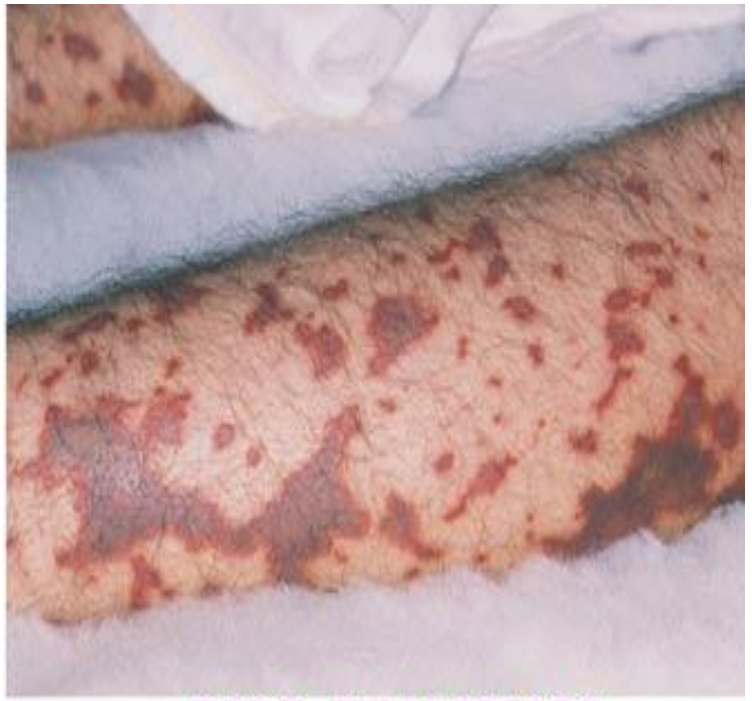
polyorganic insuffiency

The most distinctive feature is rash. Erythematous macules rapidly become petechial and, in severe cases, purpuric. Although the lesions are typically found on the trunk and lower extremities, they may also occur on the face, arms, and mucous membranes. The petechiae may coalesce into hemorrhagic bullae or may undergo necrosis and ulcerate.

Patients with severe coagulopathy may develop ischemic extremities or digits, often with a sharp line of demarcation between normal and ischemic tissue.



RASH-EARLY STAGES

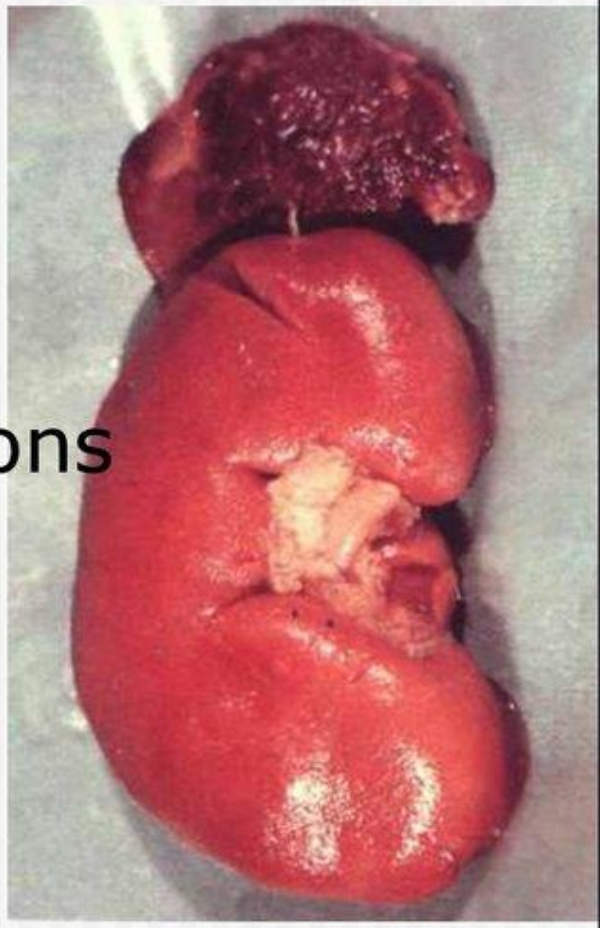


RASH-FINAL STAGES

The *Waterhouse-Friderichsen syndrome* is a dramatic example of DIC-induced microthrombosis, hemorrhage, and tissue injury. Although overt adrenal failure is infrequently documented in patients with fulminant meningococcemia, patients may have partial adrenal insufficiency and be unable to mount the normal hypercortisolemic response to severe stress or cosyntropin stimulation.Almost all patients who die from fulminant meningococcemia have adrenal hemorrhages at autopsy.

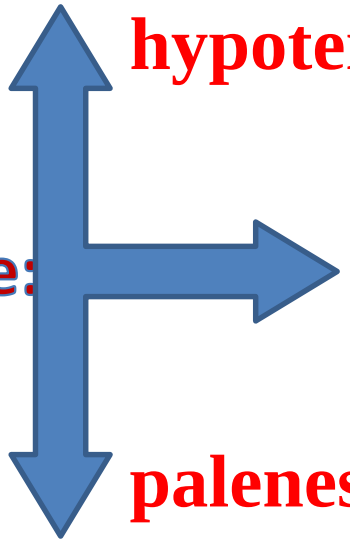
Waterhouse-Friderichsen syndrome

- 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



meningitis posture - The patient lies with his head thrown back and his legs pulled up to his stomach. Posture indicates irritation of the meninges, usually observed with meningitis

Main signs of the septic shock are:



hypotension (!)

mental disorder

paleness

▼ arthritis,

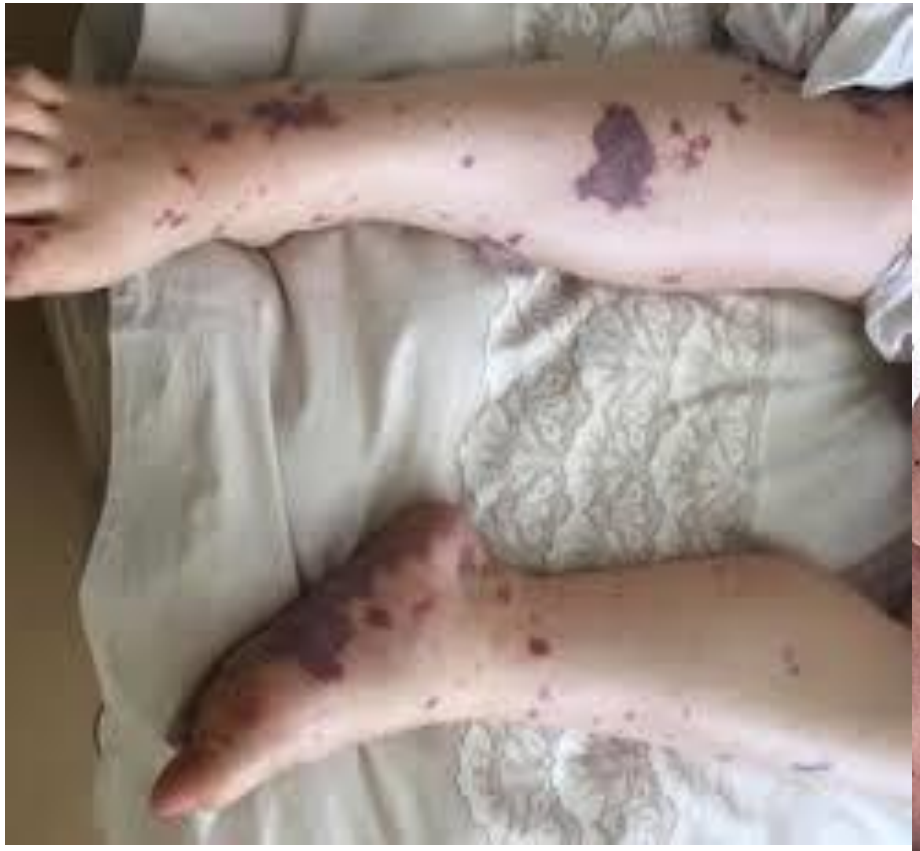
▼ disseminated intravascular coagulation (DIC),

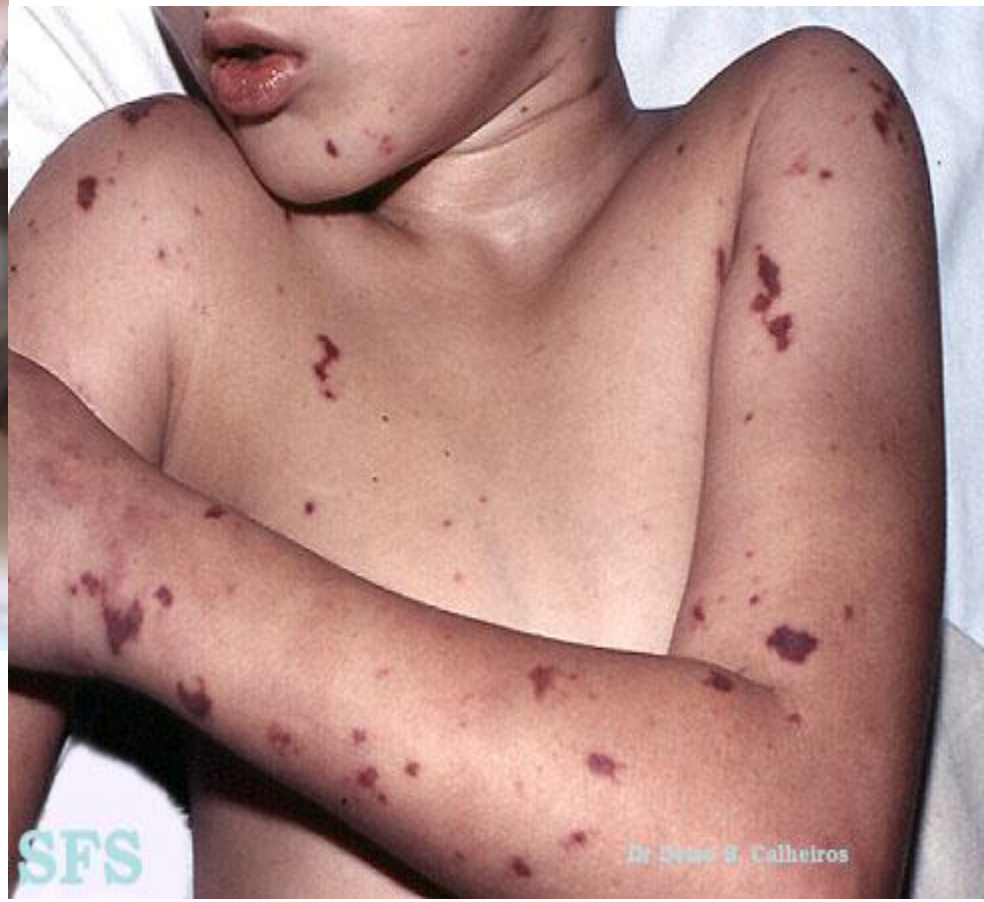
▼ inflammation of blood vessels in the skin (cutaneous vasculitis) and

▼ gangrene due to lack of blood supply,

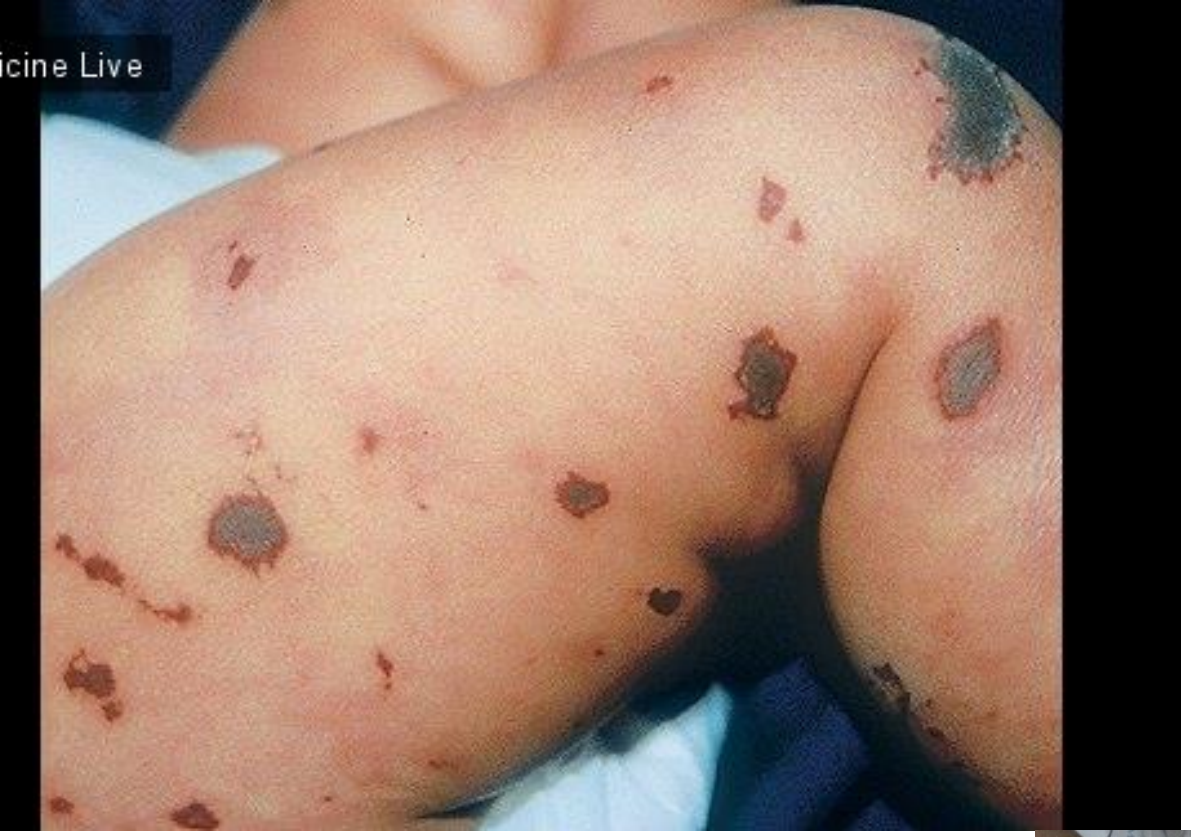
and, of course,

▼ septic shock.





Meningococcemia: first hour after diagnosis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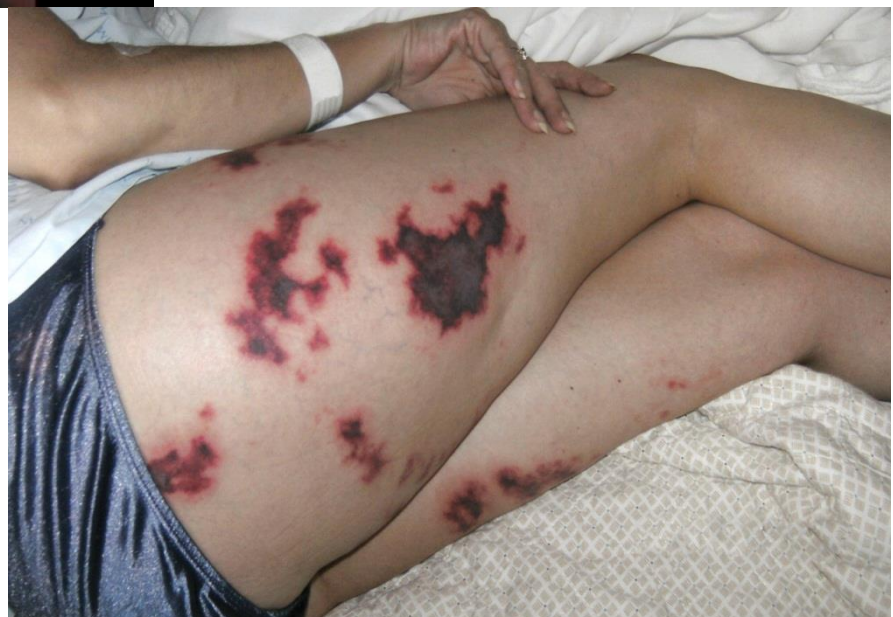
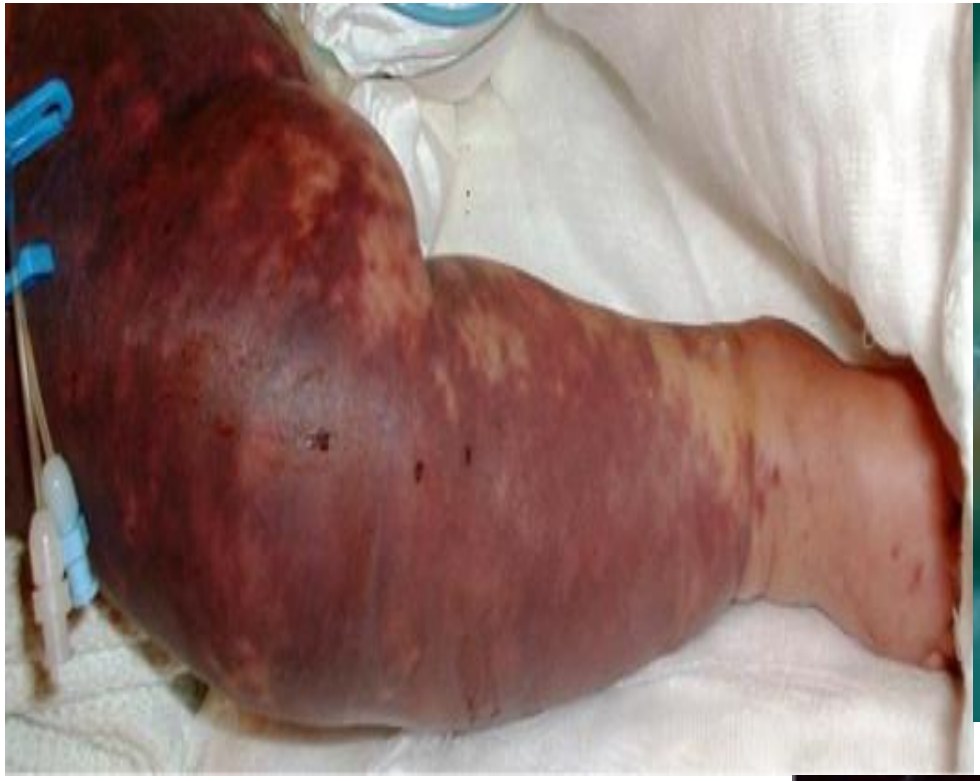
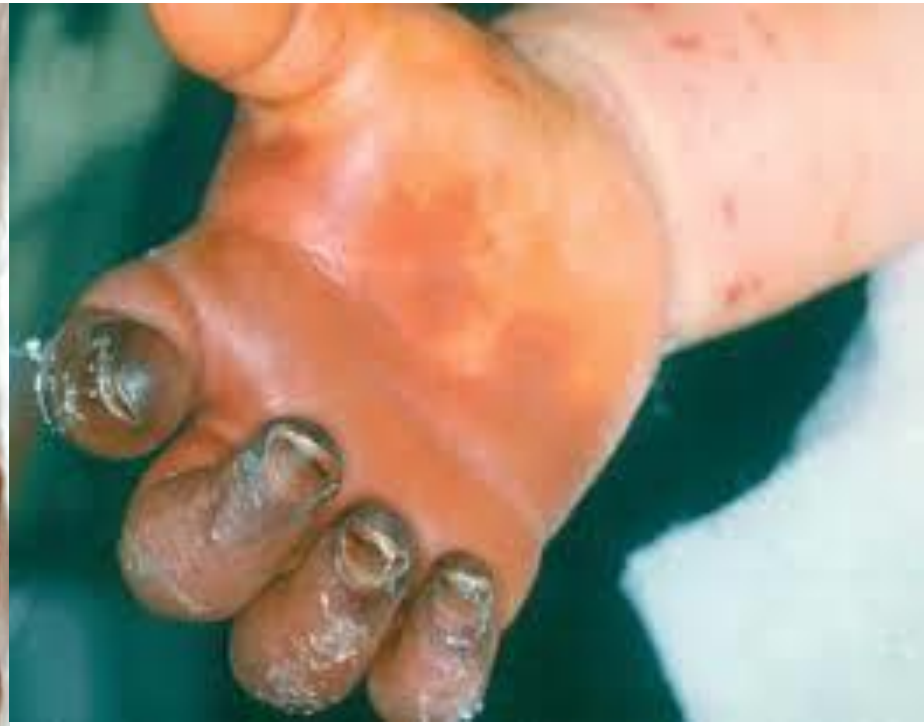
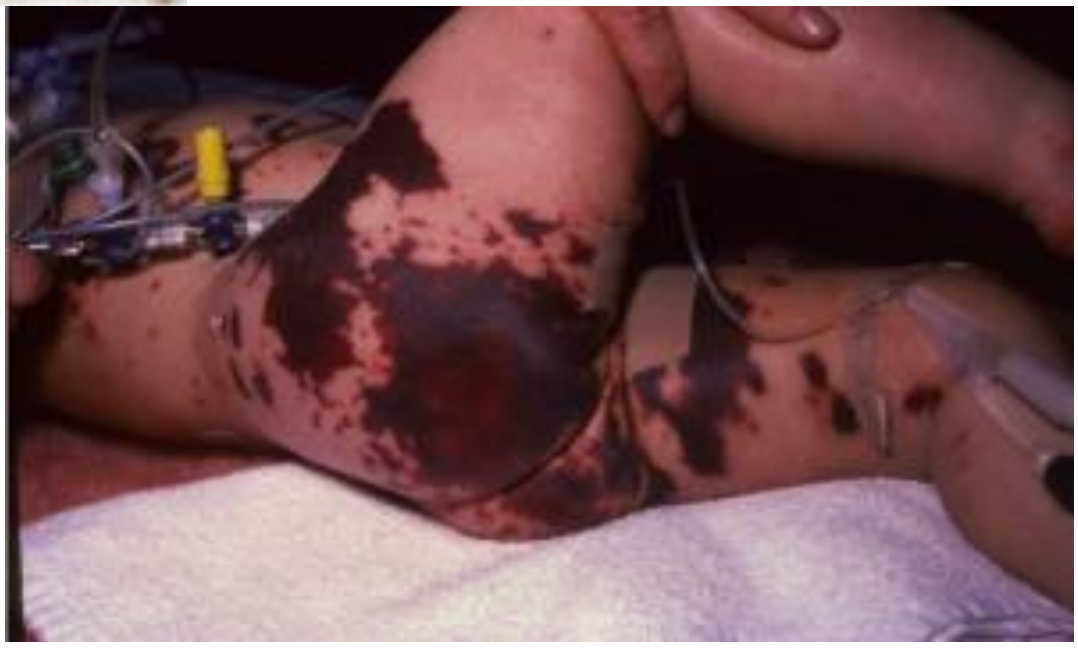
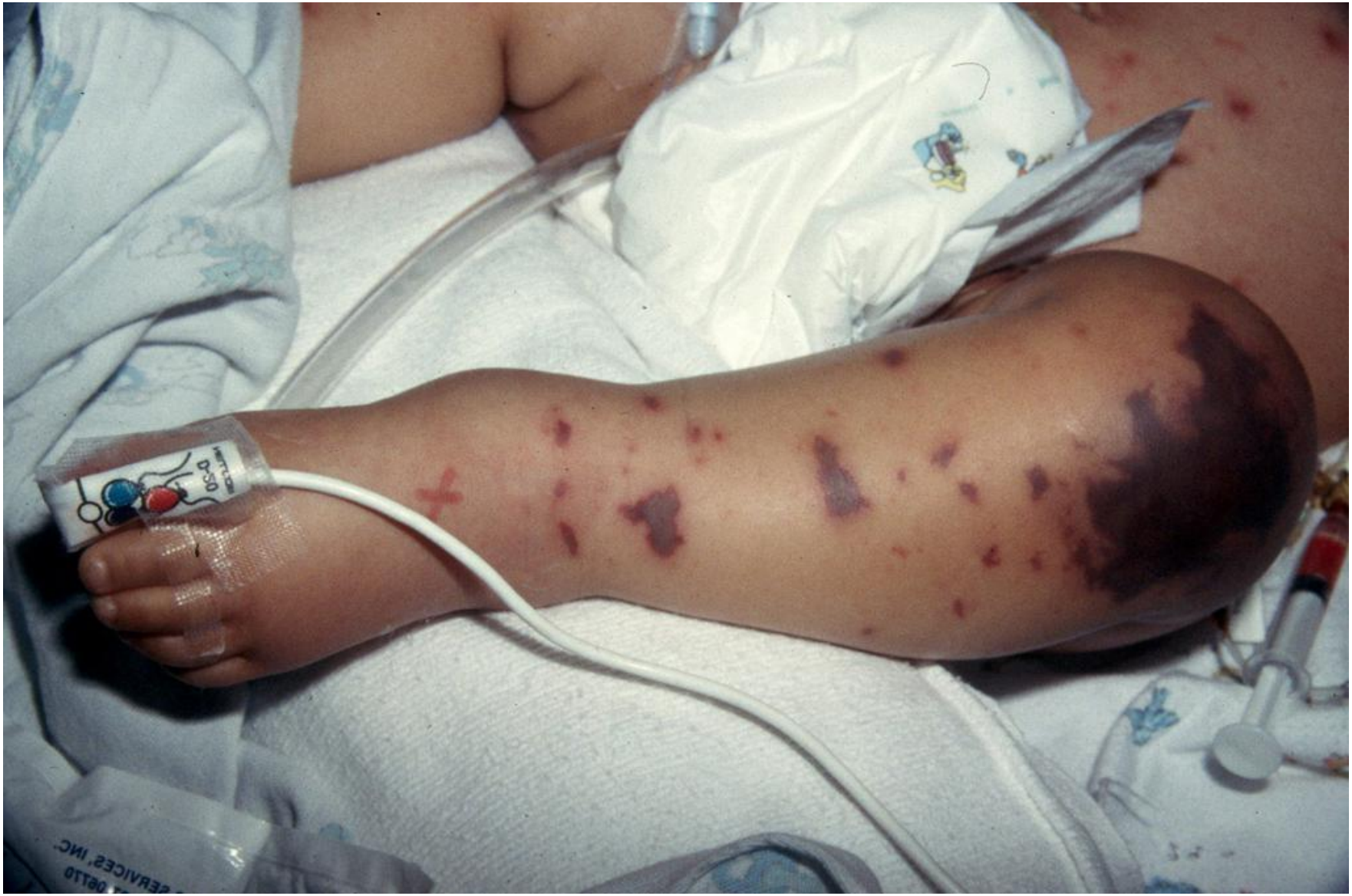


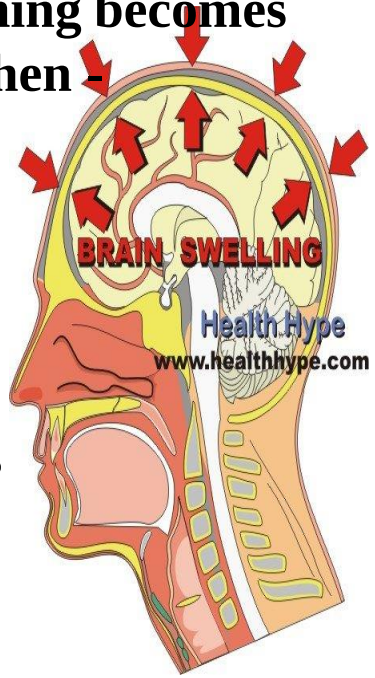








Acute swelling and brain swelling occur more frequently at the end of the first - the beginning of the second day of the disease.
Against the background of the rapid flow of meningitis with sharp signs of intoxication, cerebral disorders and agitated patient comes unconsciousness. The patients do not respond to strong stimuli. They appear and grow the overall tonic-clonic seizures.
There have been fading corneal reflex, pupillary constriction and their sluggish response to light. Bradycardia, tachycardia quickly replaced. Labile blood pressure at the beginning, with a tendency to a significant decrease in the terminal phase - high, up to 150 / 90-180 / 110 mm Hg.
Rapidly growing shortness of 50-60 breaths in 1 minute, breathing becomes noisy, superficial, with the participation of auxiliary muscles, then – arrhythmic.
Meningeal symptoms fade, elevated cerebrospinal fluid pressure is reduced. There have been involuntary defecation and urination.
Pulmonary edema, hemiparesis occur.
Death occurs when the patient stops breathing due to paralysis of the respiratory center, cardiac activity can continue for 5-10 minutes



Infectious-toxic shock occurs on the background of the rapid flow of meningococcemia. Patients with high fever and severe hemorrhagic syndrome critical body temperature drops to normal or subnormal numbers. In the early hours of the patients are fully conscious. Characterized by acute hypersensitivity, general excitement. The skin is pale. Pulse frequent, subtle. Blood pressure falls rapidly. Growing cyanosis, shortness of breath. Stops urination (kidney failure). The excitement is replaced by prostration, seizures occur. Without intensive care, death can occur within 6-60 hours after the first signs of shock. The environmental-conditions occupational stress in young people toxic shock occurs, usually in combination with an acute edema and swelling of the brain. Against the backdrop of a sharp intoxication of brain disorders appear purpura and disorders of the cardiovascular activity.
The skin is pale, cyanosis of the lips and nail phalanges.
Tachycardia increases, blood pressure drops rapidly. Sharply growing signs of brain disorders, breathing quickens to 40 or more in 1 minute, there is complete loss of consciousness, there are common kloniko-tonic convulsions, corneal reflexes fade, the pupils narrow and almost do not react to light. There anuria. The lethal outcome occurs within 18-22 hours after the first signs of associated complications.

Complications

Patients with meningococcal meningitis may develop

- **cranial nerve palsies,**
- **cortical venous thrombophlebitis,**
- **and cerebral edema.**

Children may develop ▲Subdural effusions.

- **Permanent sequelae can include**
- **mental retardation,**
- **deafness, and**
- **hemiparesis.**

The major long-term morbidity of fulminant meningococcemia is the loss of skin, limbs, or digits that results from ischemic necrosis and infarction.

Diagnosis

Diagnosis is based on clinical and epidemiological data.

Among the most important clinical symptoms include

acute onset of the disease,

severe symptoms of intoxication
chills,
loss of appetite,
pain in the eyeballs,
stupor, or excitement:

meningeal syndrome –
headache,
hypersensitivity,
nausea,
vomiting,

symptoms Kernig, Brudzinski's.

Given the fulminant course of meningococcal disease, optimal timing of the diagnosis should be considered as the first 12 hours of illness onset. Rational treatment, begun in these terms, leads to complete recovery of patients. However, it should be noted that in the first hours of the disease may not have the support meningeal signs (neck stiffness, Kernig et al.).

Diagnosis

Laboratory studies

Laboratory examination of the cerebrospinal fluid (CSF) usually confirms the presence of meningitis. Typical CSF abnormalities in meningitis include the following:

♦ Markedly increased intracranial pressure (ICP) (>180 mm water)

♦ Pleocytosis of polymorphonuclear leukocytes (white blood cell [WBC] counts between 10 and 10,000 cells/µL, predominantly neutrophils)

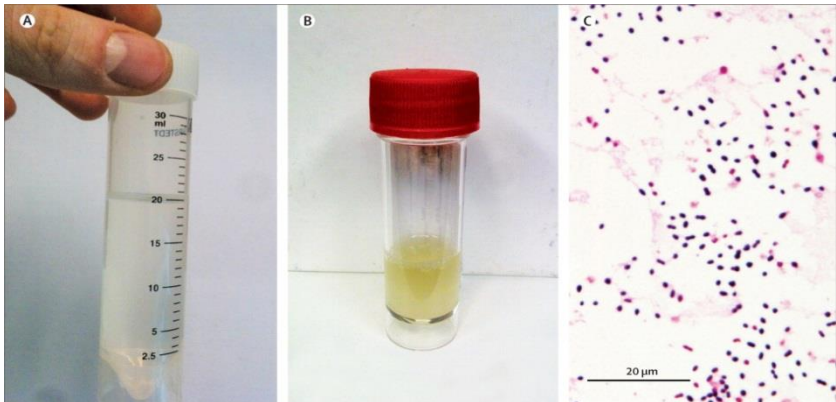
♦ Decreased glucose concentration (< 45 mg/dL)

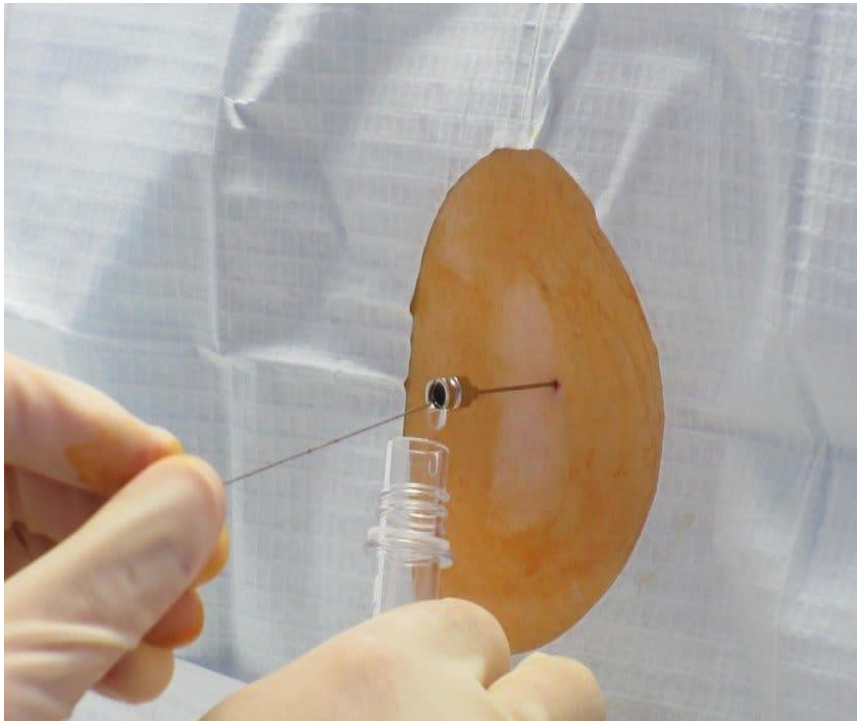
♦ Increased protein concentration (>45 mg/dL)

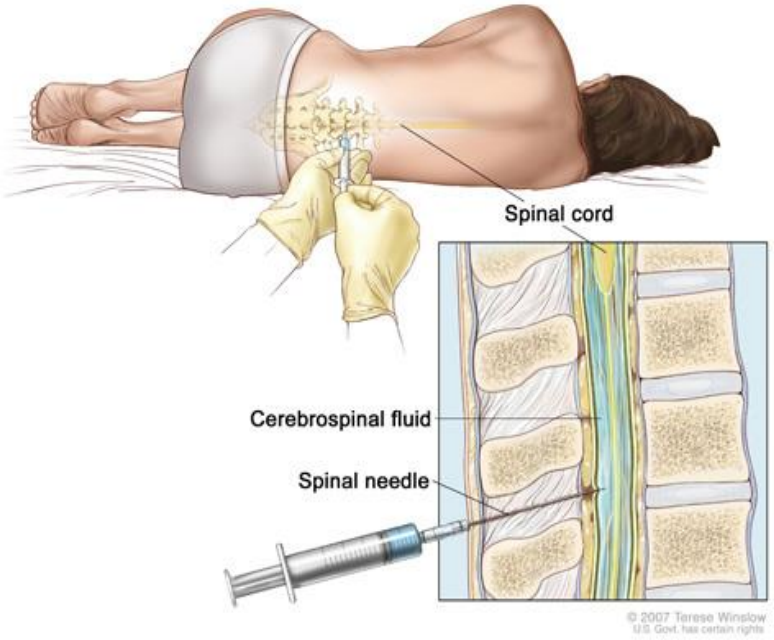
Other laboratory tests can include the following:

♦ Culture of CSF and blood specimens - To identify *N meningitidis* and the serogroup of meningococci, as well as to determine the bacterium's susceptibility to antibiotics

▼Polymerase chain reaction (PCR) assay – For confirmation of the diagnosis






Spinal cord
Cerebrospinal fluid
Spinal needle
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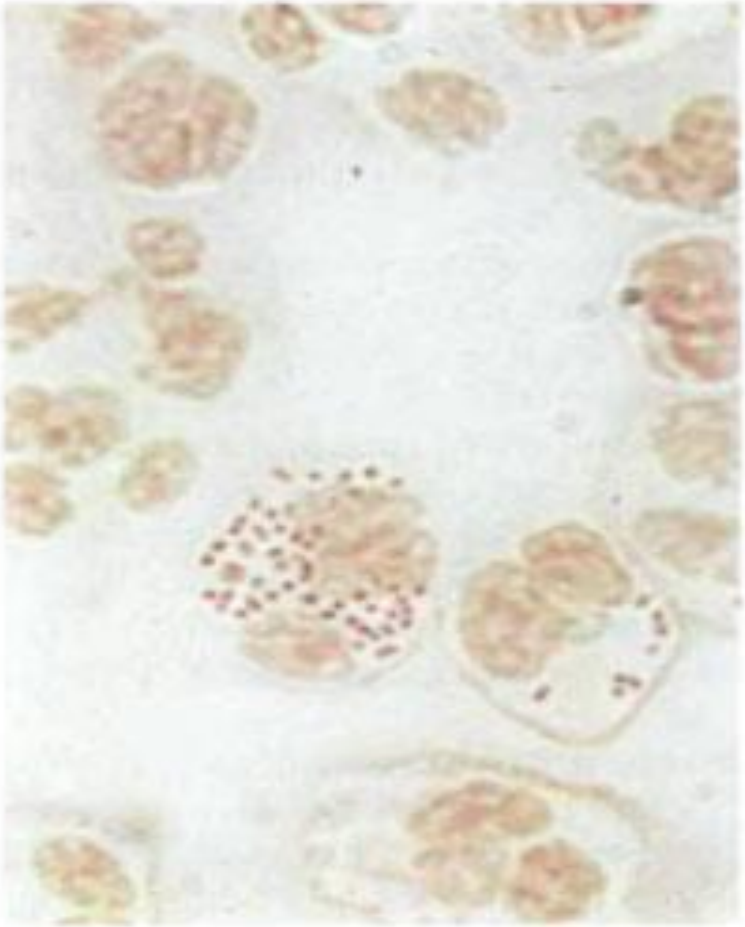

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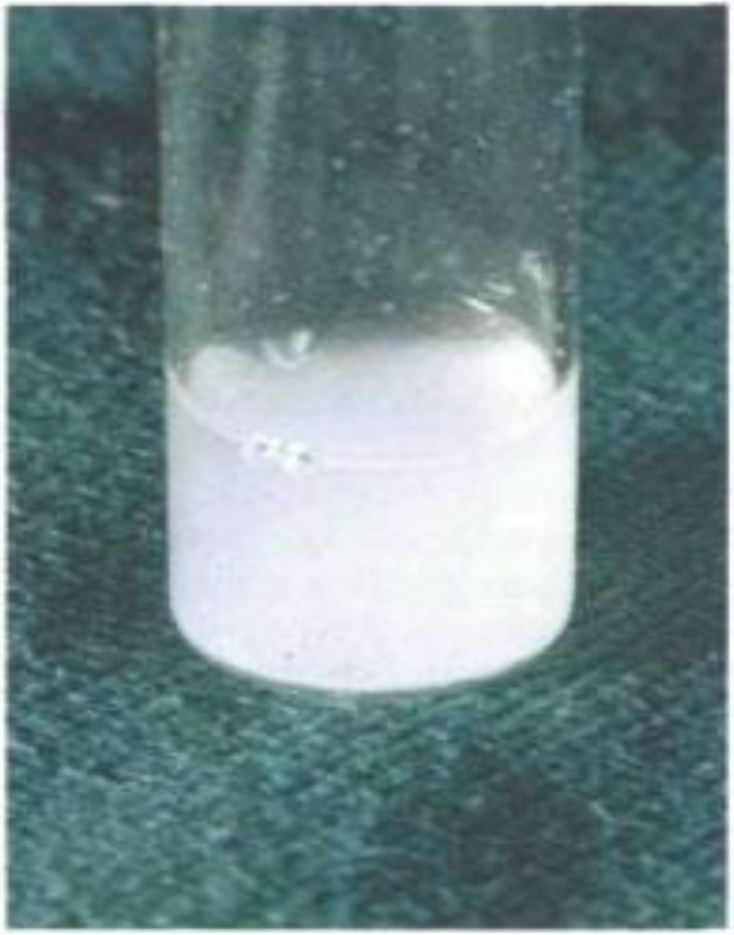


61

Purulent

cerebrospinal fluid (CSF)

Imaging studies

Computed tomography (CT) scanning - Indications for performing CT scanning prior to lumbar puncture include an altered level of consciousness, papilledema, focal neurologic deficits, and/or focal or generalized seizure activity

Magnetic resonance imaging (MRI) - MRI with contrast is preferred to CT scanning, because MRI better demonstrates meningeal lesions, cerebral edema, and cerebral ischemia

Electroencephalography

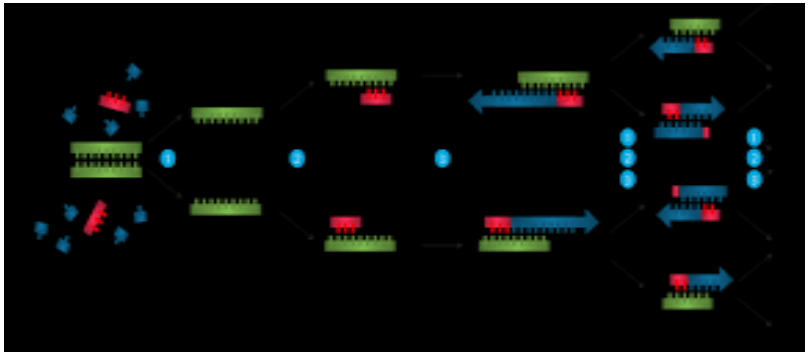
An electroencephalogram (EEG) study is sometimes useful to document irritable electrical patterns that may predispose the patient to seizures.

Polymerase Chain Reaction

The polymerase chain reaction (PCR) may be used to complement standard laboratory procedures for the diagnosis of meningococcal meningitis.

The IS1106 PCR is a rapid and sensitive test for confirmation of the diagnosis; its sensitivity is not affected by prior antibiotic treatment.

PCR of the *nspA* gene was also reported to be a fast diagnostic test.



MRI with contrast is preferred to CT scanning, because MRI better demonstrates meningeal lesions, cerebral edema, and cerebral ischemia. T1 may show obliterated cisterns.
Contrast enhances the cisterns, and extension of enhancing subarachnoid exudate deep into the sulci may be seen in severe cases.
Strokes can be seen with the development of vasculitis and cerebritis. CNS complications that can be visualized with MRI include hydrocephalus, aqueductal obstruction, ventriculitis (especially in neonates), choroid plexitis, subdural effusion, and empyema.

The differential diagnosis

is carried out with meningitis caused by various bacterial flora: pneumococci,
Haemophilus influenzae,
Staphylococcus,
Streptococcus,
fungi and others.
Meningococcemia must be distinguished from measles, rubella and others.

Treatment.

Meningococcal disease can be treated with antibiotics, but quick medical attention is extremely important.

Treatment

1

General treatment

①Isolation hospitalization:
②Careful monitor nursing.
③Prevent complication.
④Maintain the balances of fluid and electrolytes

2

Etiological treatment

① Antibacterial activity.
②Concentration in CSF.
③ Resistance to drugs
E. Penicillin G
（200~400u/kg/day）
B.Chloromycetin
C.Cephalosporis

3

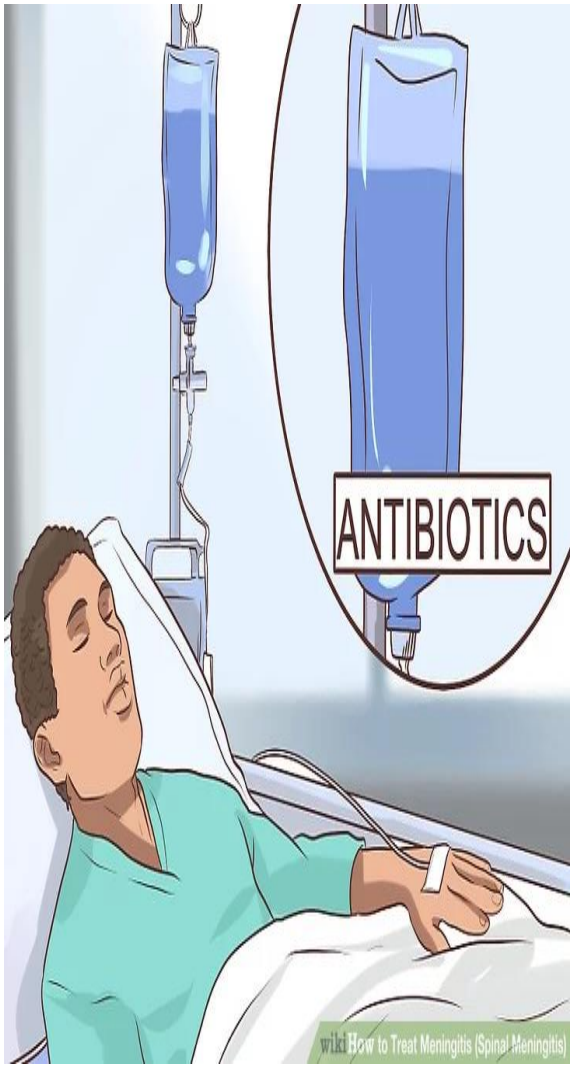
Other treatment

• High fever: anti-pyretic (physical chemical) measures.
• Increased intracranial pressure:
20 % mannitol (0.5g/kg~2g/kg)

Standard empirical therapy

Institute antimicrobial therapy as soon as possible after the lumbar puncture is performed.

At presentation, meningitis due to *N. meningitidis* may be impossible to differentiate from other types of meningitis. Thus, empirical treatment with an antibiotic with effective CNS penetration should be based on age and underlying disease status, since delay in treatment is associated with adverse clinical outcome.



Initial empirical therapy until the etiology is established should include dexamethasone, a third-generation cephalosporin (eg, ceftriaxone, cefotaxime), and vancomycin.

***Acyclovir* should be considered according to the results of the initial cerebrospinal fluid (CSF) evaluation.**

A 7-day course of intravenous ceftriaxone or penicillin is adequate for uncomplicated meningococcal meningitis.

If imaging studies are indicated before lumbar puncture, draw blood for culture and begin administration of empiric antibiotics.

A third-generation cephalosporin, such as cefotaxime or ceftriaxone, is preferred for initial therapy.

One of these cephalosporins in combination with other agents may cover other bacteria (such as Streptococcus pneumoniae and Haemophilus influenzae) that can cause the same syndromes. Penicillin G remains an acceptable alternative for confirmed invasive meningococcal disease in most countries.

However, the prevalence of meningococci with reduced susceptibility to penicillin has been increasing, and high-level penicillin resistance has been reported.

Other options include meropenem. In the patient who is allergic to β-lactam drugs, chloramphenicol is a suitable alternative;

Although glucocorticoid therapy for meningitis in adults is controversial, many experts administer dexamethasone, beginning if possible before antibiotic therapy is initiated; the schedule is 10 mg IV given 15–20 min before the first antibiotic dose and then every 6 h for 4 days.

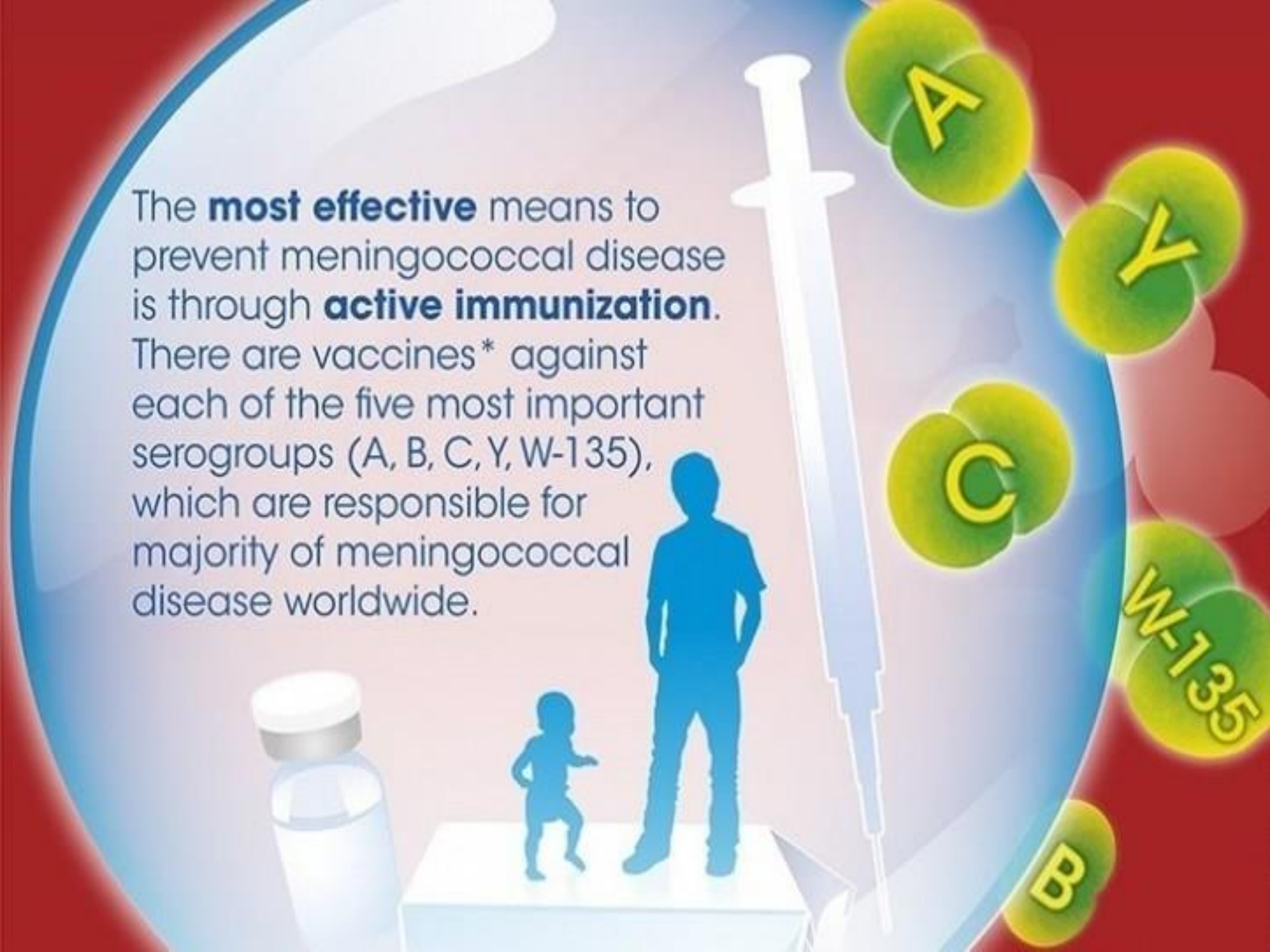
The data regarding steroid use to diminish CNS inflammation are strongest for H. influenzae and S.pneumoniae meningitis, especially in children.

Prevention.

People who are in close contact with patient should be given antibiotics to prevent infection.
Such people are: household members, roommates, people, who are into close and long-term contact with patient.
Vaccination is the best defense against meningococcal disease.

Vaccine Strategies

Three strategies for utilization of meningococcal Vaccines have been proposed:
(1) immunization of high-risk populations;
(2) universal immunization,
usually as part of infant immunization;
and
(3) epidemic response, or mass immunization of an affected population to curtail an epidemic
Because disease patterns vary widely by regions and through time, these strategies and their adoption/ relation to local disease patterns are discussed here.


The most effective means to prevent meningococcal disease is through active immunization. There are vaccines* against each of the five most important serogroups (A, B, C, Y, W-135), which are responsible for majority of meningococcal disease worldwide.
A
Y
C
W-135
B

VACCINES

Polysaccharide capsules of serogroups A, C, Y, and W135 elicit bactericidal antibodies that are serogroup specific.
These antibodies have been shown to correlate with protection from disease.

Vaccines against meningococcus A, C, W, and Y are available.
ACIP guidelines include a recommendation for primary immunization for children aged 11-12 years, with a booster dose at age 16 years.
The vaccine is also recommended for adults and children at high risk (aged 2 months or older).
High-risk persons include military recruits, contacts to index cases, individuals travelling to areas of high incidence or areas affected by outbreaks, patients with asplenia, adolescents with HIV infection, and persons with terminal complement disorders. Serogroup B vaccine is indicated as a 3-dose series in adolescents and young adults aged 10 through 25 years. College students also benefit from vaccination.

Meningococcal Vaccines

- **Purified capsular polysaccharide vaccines**
  - Bivalent vaccine against serogroups A and C
  - Quadrivalent vaccine for serogroups A,C, Y and W135
- **Protein-polysaccharide conjugate vaccines.**
  - Three monovalent meningococcal C conjugate vaccines (Not available in India)
  - Two quadrivalent conjugate vaccine (ACYW135) (Menactra & Menveo--Not available in India)
  - **Conjugate vaccines may be a better choice when available in India**

Types of Meningococcal Vaccines

- **Plain Polysaccharide** mono, bi, tri or tetravalent
- **Conjugate vaccines:**
  - Monovalent A (MenAfrivac)* & C*
  - Quadrivalent ACWY

Carrier Protein	Vaccine
DT	Menactra
TT	Nimenrix*
CRM 197	Menveo*

*Not available in india

1. Halil Özdemir et al. J Pediatr Inf 2014; 8: 178-86

- **OMV, OMP** Recombinant vaccines
- Not available in India yet

Serogroup B antigen has a great similarity with the polysaccharide epitopes of human nerve tissues, it has an immune tolerance to serogroup B capsule[1].