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O. A. Kozyolkin, I. V. Vizir, M. V. Sikorskay

INFECTIOUS AND PARASITIC DISEASE OF THE NERVOUS SYSTEM

Textbook for students - foreign citizens

VI course of medical faculties of the specialty "Medicine"

Zaporizhzhia

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Reviewers:

L. V. Lukashenko - Head of the Department of Propaedeutics, Internal Medicine, Radiation Diagnostics and Radiation Therapy, Doctor of Medical Sciences, Professor.
O.V. Ryabokon - Head of the Department of Infectious Diseases, Doctor of Medical Sciences, Professor.

Authors:
O. A. Kozyolkin - professor of Neurology, the Head of Department of Neurology
I. V. Vizir– docent of Neurology, Department of Neurology;
M. V. Sikorskay - docent of Neurology, Department of Neurology.

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CONTENTS

INTRODUCTION

The term “neuroinfection” does not refer to any single disease. This is a whole section of fairly common neurological pathologies (more than 42% of the total statistics). It included lesions of the nervous system caused by an infectious agent. In other words, neuroinfection is the general name for infectious diseases, the causative agent of which is localized in the nervous tissue and causes inflammation:

• the brain or its membranes;

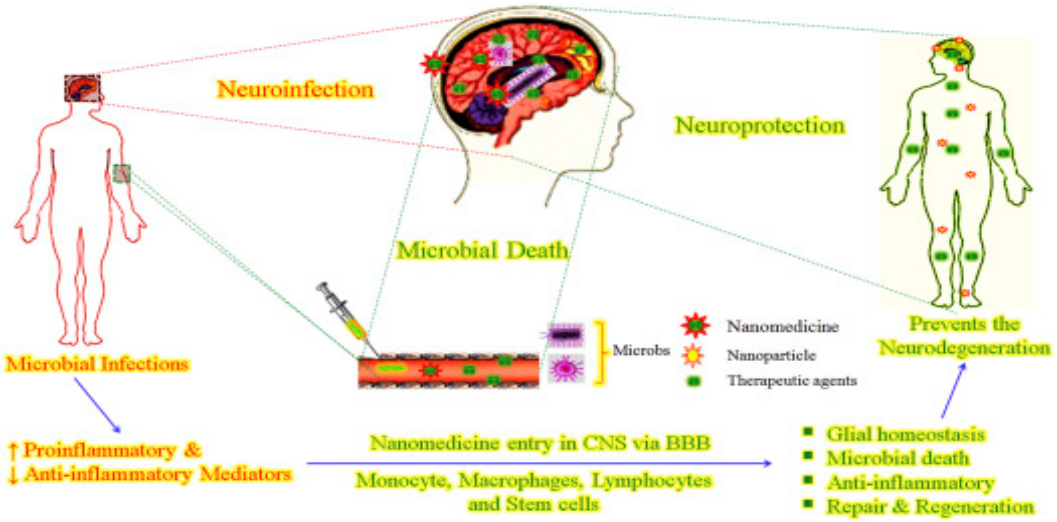
• spinal cord;

• peripheral nerves.

The etiological spectrum of neuroinfections is quite wide - viruses, bacteria, fungi, some types of protozoa. Of great importance are mixed infections, in which a symbiosis of pathogenic flora is detected. For example, herpetic neuroinfection - meningoencephalitis, encephalomyelitis.

Taking into account the duration of the course of the disease, there may be neuroinfections that have a chronic (sluggish) character, or acute and subacute forms of lesions of the nervous system.

INFECTIOUSOF THE NERVOUS SYSTEM



Picture 1. Infectious of the nervous system*(http://what-when-how.com)*

Infections disease of the nervous system are one the most common forms of neurological pathology, in recent years, the possibilities of diagnosing neuroinfection have significantly expanded. Due to the development and introduction into clinical practice of powerful antibacterial and antiviral drugs, significant. Success has been achieved in the treatment of some recently lethal diseases.

However the expansion of the spectrum etiopathogens that cause the development of neurionfections the appearance of mixed, atypical form the growth of resistance of the main bacterial pathogens to the most common antibiotics creates difficulties in the treatment of infections lesions of the nervous system.

Form of interaction of an infected agent with the human body

1. Innaparant (asymptomatic) infection (meningococcal carriage).
2. Acute (productive) infection: after the introduction of the infection, after a short time (incubation period) the picture of an infections disease develops sharply.
3. Persistent infection:

- Latent infection – asymptomatic persistence of the pathogen, in which reproduction of the pathogen and release to the external environment can occur. When the pathogen is activated the immune system decreases a disease develops (herpetic infection).
- Chronic infection – persistence of the pathogen accompanied by one or more symptoms of the disease. Pathological process is maintained for a long time, remissions alternate with exacerbations, with proper treatment, recovery occurs.

4. Slow infection – long, asymptomatic persistence of a deletion virus or prion. After a month's (or many years) incubation period, slowly and steadily. Increase of symptoms of disease. Ends in death (subactive sclerotic panencephalitis of Creutzfeldt-Jakob disease).

Damage to brain tissue

1. The direct action of the infections agent on the brain cells.
2. Toxic or toxic-allergic damage to cerebral vessels with the development of diapedetic hemorrhages, cerebral edema, ischemic foci).
3. Demyelination with secondary (usually reversible) damage to nerve cells.

Depending on the etiology one or another mechanism prevails.

Meningitis – acute infection disease primarily affecting soft membranes of the brain and spinal cord.

Encephalitis – inflammatory lesion of the brain tissue of infections or infections-allergic origin.

Brain damage in encephalitis lead to the development of focal and cerebral symptoms with secondary involvement of the meninges, meningeal syndrome develops and inflammatory changes in the cerebrospinal fluid are detected.

Myelitis – inflammation of the spinal cord.

Polyradiculoneuritis – diffuse damage to peripheral nerve fibers (spinal roots, nerve) of an inflammatory nature.

MENINGITIS

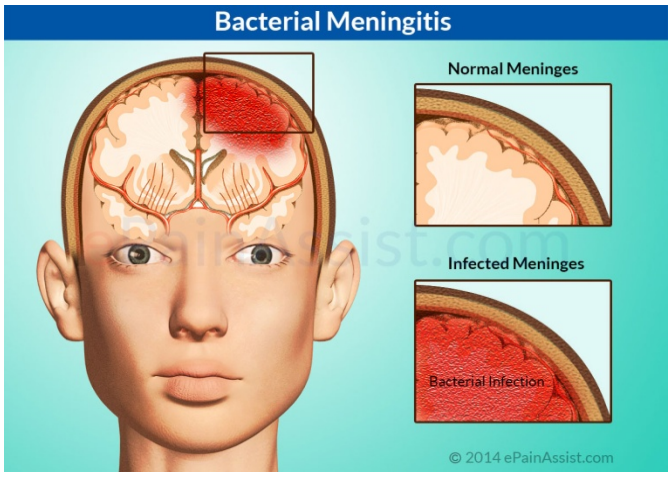
Meningitis is an acute infectious disease with primary involving of pia and arachnoid maters of brain and spinal cord.

Classification of meningitis.

Meningitis is classified according to several criteria.

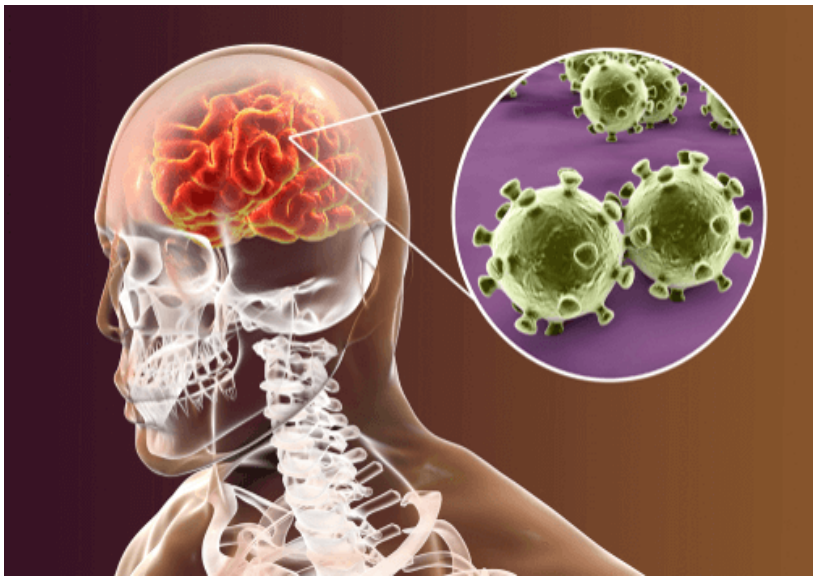
1. Due to etiology.

- bacterial (pneumococcal, tuberculosis, meningococcal)



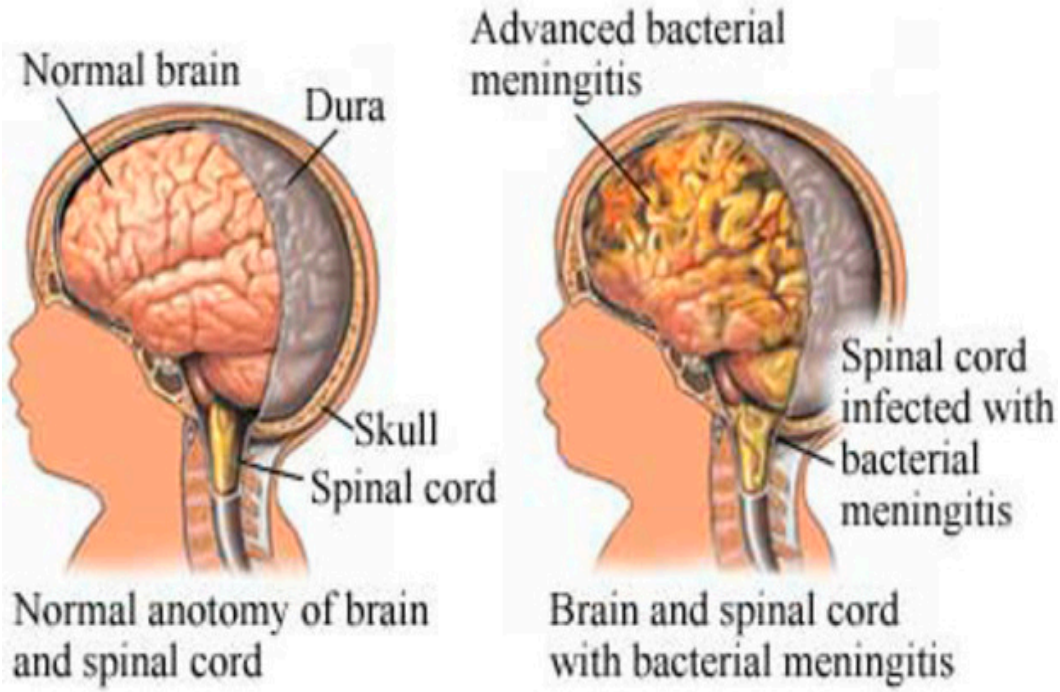
Picture 2. Bacterial meningitis *(https://www.medindia.net)*

- viral (enteroviruses: Coxsackievirus and ECHO viruses; herpes virus, acute lymphocytic choriomeningitisetc)



Picture 3. Viral meningitis *(https://u.osu.edu)*

- fungal (cryptococcal, candida etc)



Picture 4. Fungal meningitis *(https://www.thehour.com)*

- protozoa.

2. Due to the nature of the inflammatory process:

- purulent (CSF: neutrophils predominance) – is caused mainly by bacteria
- serous (CSF: lymphocytes predominance) – is caused viruses (mainly). Mycobacterium tuberculosis, spirochetes of Treponemapallidum, etc.

3. Due to pathogenesis:

- primary (no general history of infection or infectious disease of any organ)
- secondary

4. Due to the process prevalence:

- generalized
- limited

5. Due to the disease rate:

- fulminant
- acute
- subacute
- chronic

6. Due to the degree of severity:

- mild from
- moderate severity
- severe from
- extremely severe from

Pontes of entry into the body

1. Aspiration (airborne droplet airborne)
2. Fecal oral
3. Contact
4. Transmission

Ways of penetration into the body

1. Hematogenous
2. Lymphogenous
3. Contacnt

Clinical picture of meningitis

A. Infections disease syndrome.

1. Hyperhermia.
2. Inflammatory changes in peripheral blood.
3. Rach.
4. Catarrhal changes in the upper respiratory tract.
5. Dyspepsia.

B. Meningeal symptom complex.

1. General cerebral symptoms:

- headache
- cerebral vomiting
- psychomotor agitation
- change of consciousness
- convulsions
- mental disorders

2. Actual meningeal symptoms:

A. General hyperesthesia syndrome (phomo-photophosia).

B. Reactive pain syndromes

- Bechterew’szygomatic syndrome
- symptom of Mendel
- Flatau’s symptom

C. Muscle tonic symptoms:

- Kernig’s symptoms
- Brudinsks’s symptoms
- Le Sage’s symptoms

D. Cerebrospinal fluid syndrome.

1. Increased CSF pressure.

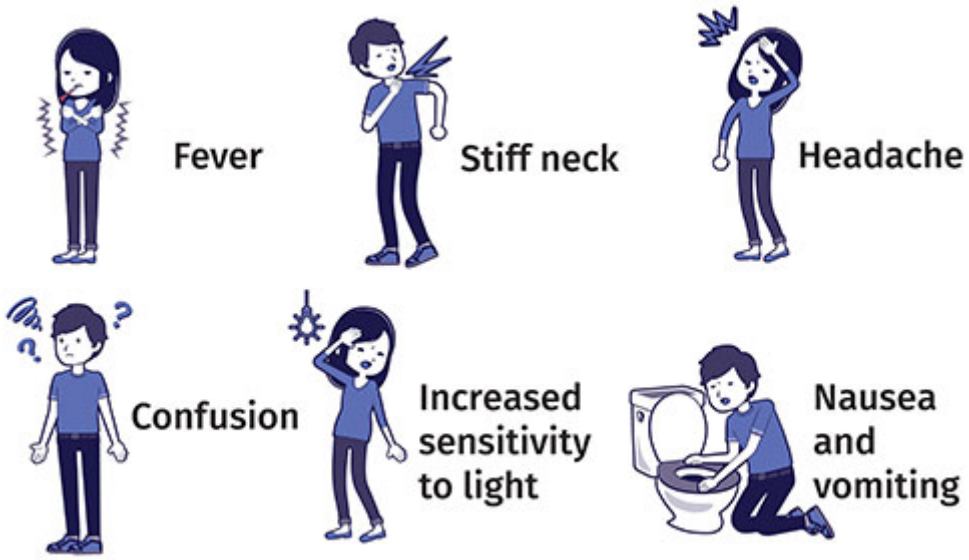
2. Clody cerebrospinal fluid in purulent meningitis.

3. Neurophilicpleocytosis, lymphocytic pleocytosis.

4. High level of protein more pronounced in purulent meningitis.

5. Decreases glucose in bacterial, fungal and proroxoa.

Meningitis symptoms.



Picture 5. Meningitis symptom *(https://www.cdc.gov)*

Table 1. ***Differential diagnosis cerebrospinal fluid syndromes***

Disease / Indicators	Normal	Changes in cerebrospinal fluid			
		Meningism	Serous bacterial meningitis (tbs meningitis)	Serous viruses meningitis	Purulent bacterial (meningococcal meningitis)
Transparency	+	+	Opalescent	Opalescent	Clody
Pussure (mm water column)	100/200/250		↑	↑	↑
Cytosis (in 1 mcl)	0-5	N	Lymphocytic pleocytosis 65-80-L; 20-30%-n	Lymphocytic pleocytosis 90-95%	70-90% (n)
Cytogram		N			↑ Neurophilicpleocytosis
Protein g/l	0,12-0,45		↑	↑	↑
Delicate fibrous membrane	-	-	+	-	-
Glucose m/ml	3,5 mmol/l 40-70 mg/l	N	<50% of blood glucose level	N or <<	<<

Chlorides	120-130		<<	N or <<	<
LF		N	+	+	-
HF		N	-	-	+++
2,5-4,4 (no less than 50% blood level)					

Treatment meningitis

Bacterial meningitis

1. Antibacterial therapy shold be started in the first hours of the disease (depending on the sensitivity of the pathogen). Intravenous's antibiotics infection before sanitation of the cerebrospinal fluid (10-14 days).
2. Fight against cerebral edema – manitol, laziks, dexametasone.
3. Deintoxication – reosorbilact or repolyglucin.
4. Treatment usually includes normalization of vital body function (blood pressure, respiratory function, regulation of water and electrolyte balance).
5. Symptomatic therapy.

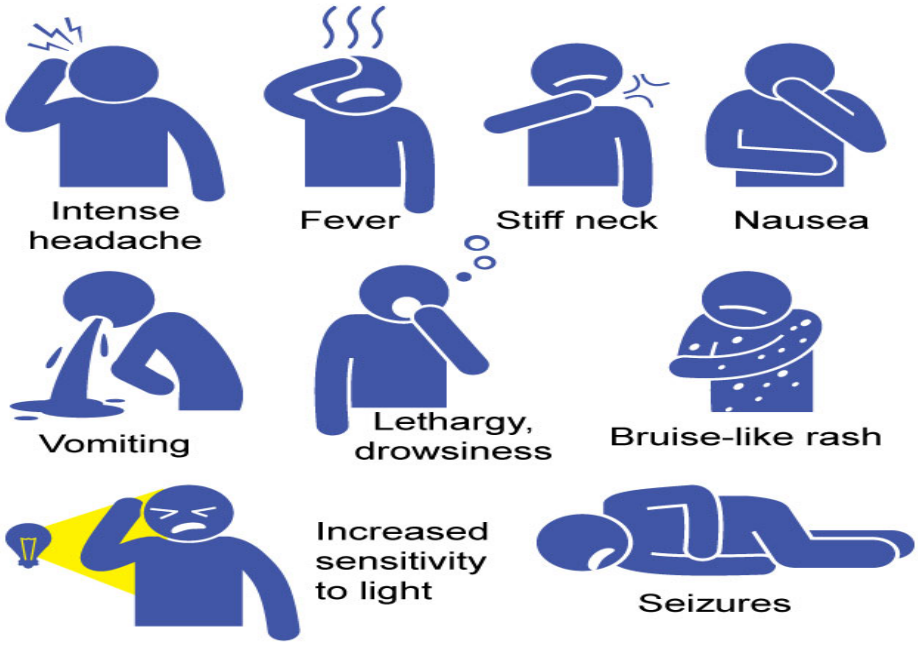
Viral meningitis

1. Etiotropic therapy: antiviral agents (Acyclovir, Zoviraks), immunomodulatory agents (human immunoglobulin).
2. Symptomatic therapy: correction of fluid and electrolyte balance (Ringer's solution, Trisol), control of intracranial hypertension (diuretics, glucocorticoid), correction of hypovolemia, seiures.

Clinical feature of meningitis

Purulent meningitis

1. Meningococcal meningitis.

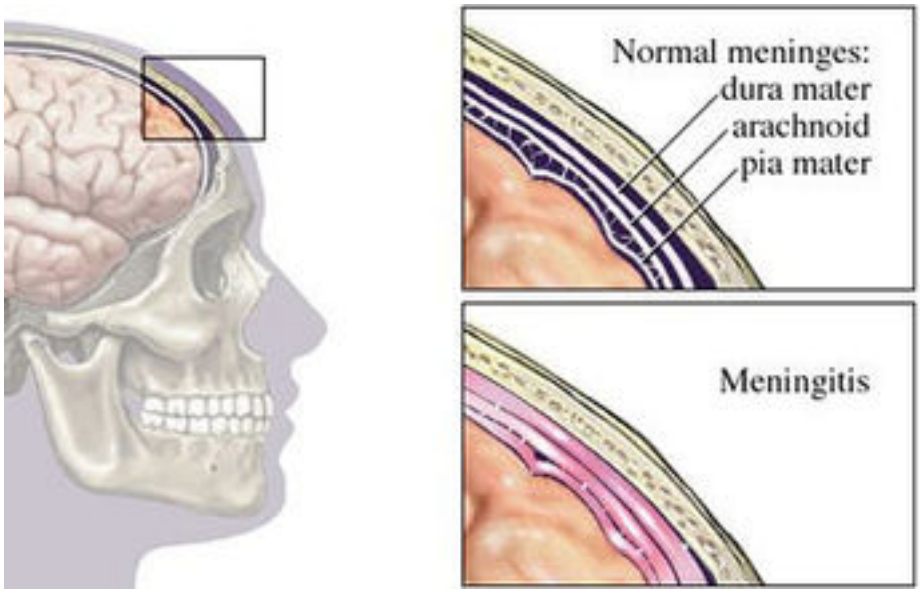


Picture 6. Symptoms meningococcal meningitis *(https://www.momjunction.com)*

The incubation period of meningococcal infection is 2-6 days.

Clinical presentation. The incubation period of meningococcal infection is 2-6 days. The disease begins acutely with fever up to 38-40° C, a sharp headache with irradiation to the neck and back, vomiting that does not bring any relief. During the first or second day meningeal signs are observed. At the beginning of the disease a patient is agitated, and then drowsiness, sopor turning into coma appears. From the 2nd day of illness herpetic eruptions on the lips and face are observed. In severe cases of meningococcal meningitis typical hemorrahagic rash develops. Neurological examination does not show any focal symptoms (in case of a hard course of the disease the signs of III, IV, VII, VIII pair of cranial nerves lesion are possible). A combination of acute beginning, fever, headache with vomiting in the first hours of the disease makes a doctor suspect meningitis.

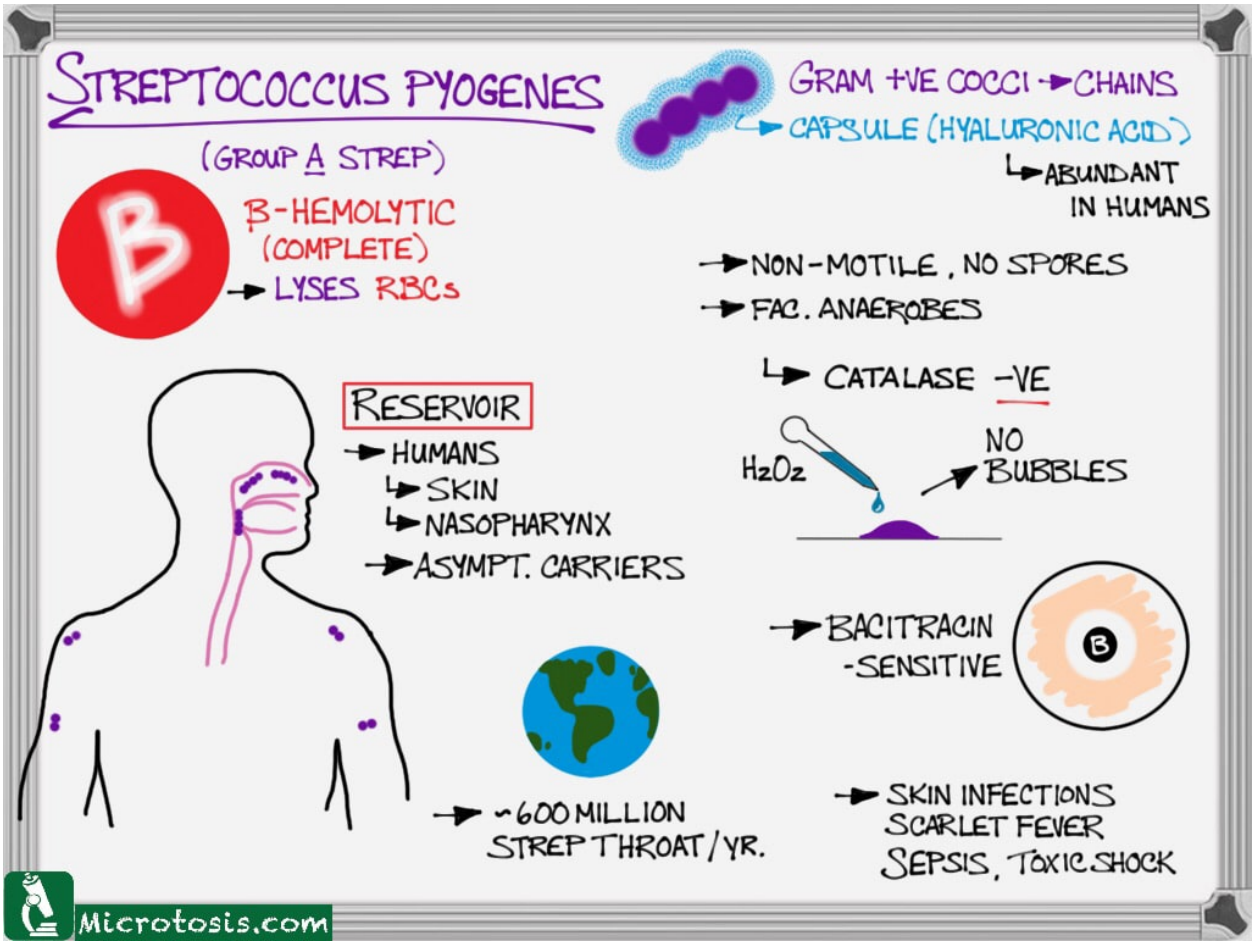
2. Pneumococcal meningitis.



Picture 7. Pneumococcal meningitis *(https://microbewiki.kenyon.edu)*

The pathogen of the disease is gram-positive diplococcus — extracellular pneumococcus. This meningitis may be either primary or secondary. Most often the disease occurs as complication after pneumonia, otitus, sinusitis, mastoiditis. The clinical course is hard. The brain tissue is often affected, the picture of purulent meningoencephalitiscan be observed. The typical sign is loss of consciousness with the development of sopor or coma, convulsions. There are signs of cranial nerves lesion, focal neurological symptoms (mono-, hemiparesis). A frequent phenomenon is swelling of brain tissue with possible signs of wedging. Bradycardia and low blood pressure are observed. In the peripheral blood inflammatory changes take place. Cerebrospinal fluid is very cloudy, has a greenish color, its pathological changes are the same as in case of other purulent meningitis.

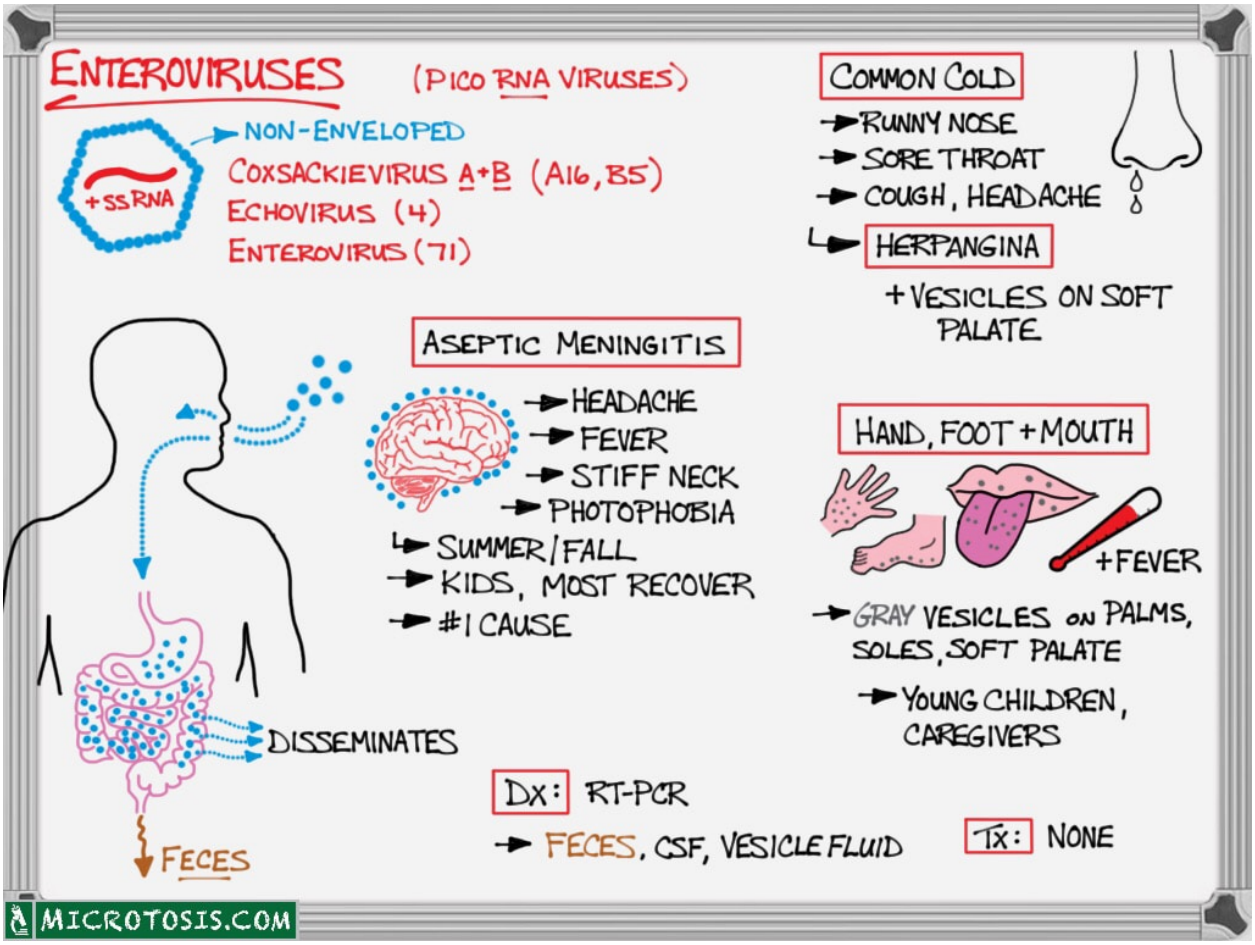
3. Staphylococcal and streptococcal meningitis.



Picture 8. Staphylococcal and streptococcal meningitis*(https://microtosis.com)*

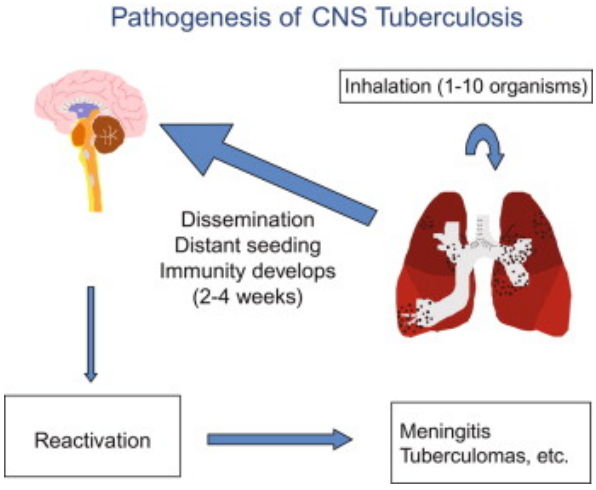
This meningitis occurs as complication of acute and chronic purulent processes or traumatic brain injuries. The onset is accompanied by chills and fever to 40°C. They characterized meningeal syndrome, impaired consciousness, and seizures. The peculiarity of staphylococcal meningitis is frequent abscess formation, blocking of spinal routes and mycosis.

Serous meningitis



Picture 9. Serous meningitis*(https://microtosis.com)*

1. Tuberculous meningitis.



Picture 10.Patohogenesis Tuberculous meningitis *(https://epos.myesr.org)*

Specific secondary serous meningitis occurs on a background of tuberculosis of lung, bronchus, internal reproductive organs, bones, kidneys and other organs. Mycobacterium tuberculosis penetrates into the subarachnoid space from the primary focus and affects base of the brain, the III-d and IV-th ventricular ependyma, choroidal plexus. Most often, tuberculous meningitis is a manifestation of hematogenous disseminated tuberculosis.

The clinical features.The disease develops gradually: headache, dizziness, nausea, fever (subfebrile, seldom – high temperature).

Two phases of the disease are typical:

1 phase – typical harbingers are general exhaustion, pallor, anorexia, drowsiness, weakness, irritability, tearfulness. It lasts 2 weeks.

2 phase – the appearance of typical meningitis symptom: nausea, headache, constipation, neck muscle stiffness, Kernig's and Brudzinski's signs.

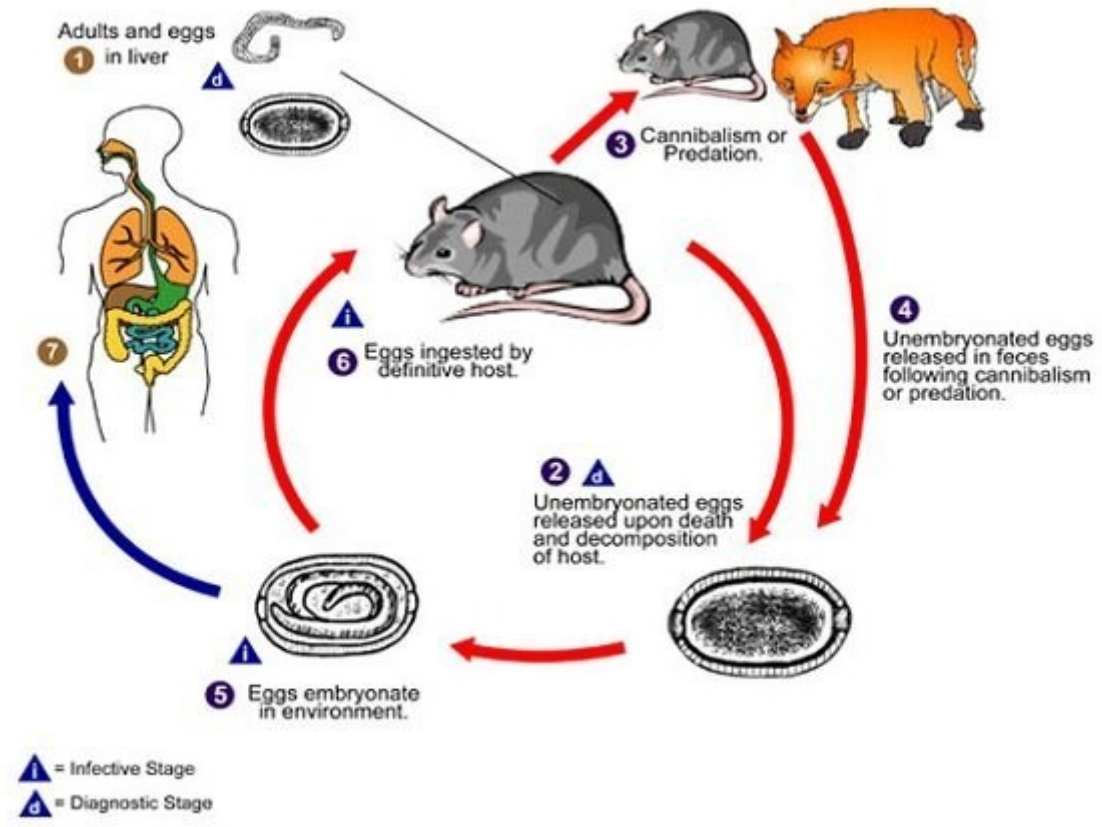
The manifestations of meningitis get more intensive gradually. As a result of the serous-fibrous exudates accumulation at the brain base, the irritation of cranial nerves may develop. The are following symptoms: vision disturbances, facial muscles paresis, strabismus, anisocoria, mydriasis, ptosis, deafness. Further, the impaired consciousness, clonic seizures, pelvic and autonomic disorders, signs of bulbar palsy, which are prognostical unfavorable symptoms for the patient, may develop too.

2. Serous meningitis caused by Coxsakie virus and ECHO (the group of enteroviruses).

In develops more frequently in children as epidemic outbreaks. The meningitis onset is acute with fever, muscle pain, gastrointestinal disorders and herpetic rash on the lips, sometimes – herpetic sore throat. Meningeal symptoms occur after 2-3 days of illness onset. Severe manifestation of intracranial hypertension is marked. Disease course prognosis is favorable.

3. Acute lymphocytic choriomeningitis (acute serous Armstrong's meningitis) is caused by a virus, which are the main reservoir of the virus. Human

contamination is a result of product use, which is contaminated by nasal mucus, urine and feces of mice. The virus spreads by blood throughout the body. The clinical features are typical. Perhaps there is the III-d and VII-th cranial nerves defeat.



Picture 11. Acute lymphocytic choriomeningitis*(http://www.histopathology-india.net)*

Additional examination methods

1. CSF research.
2. Bacteriological and serological testing of the cerebrospinal fluid reveals pathogen of purulent meningitis. It is importance to determine its sensitivity to antibiotics.

3. Detection of DNA of pathogens in cerebrospinal fluid by polymerase chain reaction (PCR) method: herpes simplex virus (type 1, 2), Epstein-Barr virus (type IV) and cytomegalovirus (V type), Mycobacterium tuberculosis.
4. EEG in patients with seizure to detected epileptic activity.
5. CT scan or MRI. The value of these techniques is limited in the acute period of meningitis. They are important for the diagnosis of meningoencephalitis, brain abscess.
6. While finding brain abscess a neurosurgeon consultation is indicated.
7. Ophthalmological research.
8. Otolaryngology research for identifying the primary focus of purulent meningitis.

Differential diagnosisis made with disease which occur with similar clinical symptoms; subarachnoid hemorrhage, hemorrhagic stroke, acute hypertensive encephalopathy, brain abscess, sinus thrombosis, brain tumor (extracerebral), membranes carcinomatosis of brain and spinal cord, membranes sarcoidosis, hypertensive syndrome in case of brain trauma; infection, which are accompanied with meningismus.

ENCEPHALITIS

Classification

I. The primary, virus encephalitis.

1. The encephalitis with the known virus.

1.1. Arboviral seasonal (transmissive).

1.1.1. Spring-summer:

- tick-borne
- scotish

1.1.2. Summer-autumn:

- Mosquito (Japanes)
- American
- Australian

1.2. The primary viral without precise seansonality.

1.2.1. Enteroviral (Koksaki and ECHO).

1.2.2. Herpetic.

1.2.3. The influenza encephalitis.

1.2.4. In the case of rabies.

2. The encephalitis with the unknown virus.

2.1. The epidemic encephalitis (Economo).

2.2. The polyseansonal encephalitis in children (combined group).

II. The encephalitis and encephalomyelitis: infection-allergic and allergic.

1. Parainfectious encephalitis and encephalomyelitis (secondary).

1.1. In the case of measles.

1.2. In the case of chiken pox.

1.3. In the case of rubella and other exanthematous infection.

2. The postvaccinal encephalitis and encephalomyelitis.

2.1. Vaccination against the smallpox.

2.2. Vaccination against DTP and ADTP.

2.3. In other vaccination.

3. Antirabic.

4. Demyelinating.

4.1. The leuko- and panenencephalitis.

III. The microbial and rickettsial encephalitis and encephalomyeltes.

1. Primary.

1.1. In the syphilis of the nervous system.

1.2. In the case of the tuphus.

2. Secondary.

2.1. Staphylococcal, streptococcal and other.

2.2. Malarial.

2.3. Toxoplasmosal.

3. When various microbial and rickettsial diseases.

IV. The infectious-genetic encepalites and encephalomyelitis.

1. Kuru.

2. The Viluj's encephalitis.

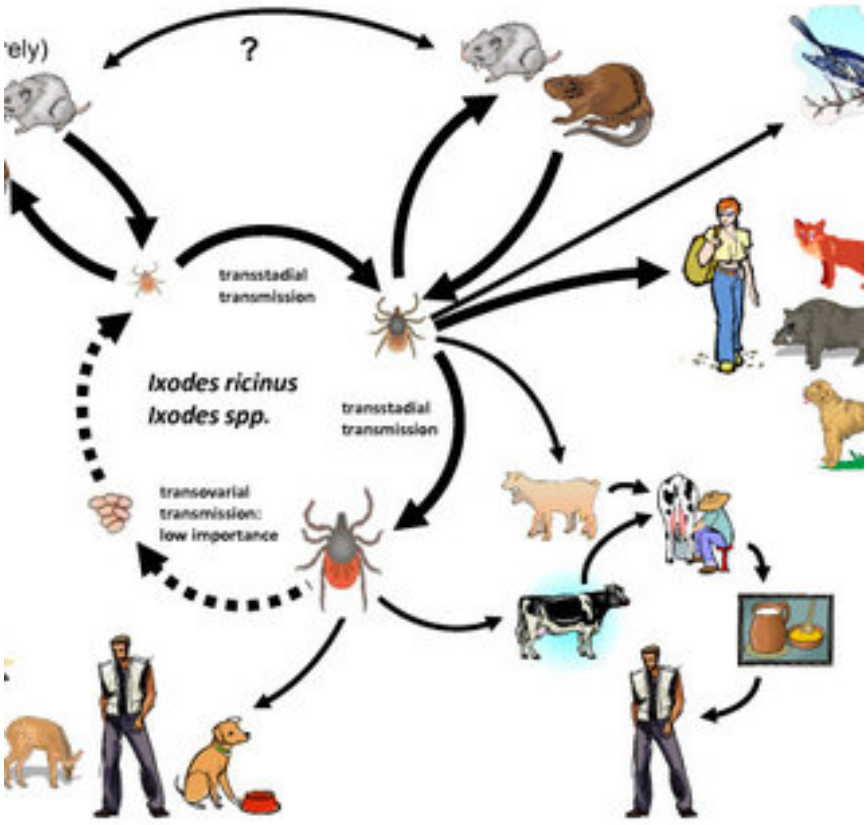
Table 2. Topical diagnosis of the encephalitis

Encephalitis	Localization of the lesion
Tick-borne (spring-summer)	Motor neuron of spinal cord, brain steam pyramidal cells.
Epidemic (Economo's, lethargic)	Gray matter (III-d ventricle walls), reticular formation, brain steam and hypothalamus, substrantianigra.
Mosquito	Brain cortex, basal ganglion, cerebellum, substantianigra, brain steam (medulla oblongata), and spinal cord.
Herpetic	Gray matter (frontal, temporal, parietal lobe), brain steam.

Table 3. Differential diagnosis of the primary encephalitis

Symptoms	Tick-borne (spring-summer)	Epidemic	Mosquito	Herpetic
Pathogen	Specific arbovirus with neurotic influence	Unknown pathogen	Neurothropic virus	Herpes simplex virus 1 type
Seasonality	Spring, summer	No clearly defined seasonality	Summer, autumn	Multi seansonal
Incubation period	7-14 days	4-15 days	2-4 days	8-20 days
By the following ways	Transmissible alimentary	Airborne and contact	Transmissible	Airborne hematogenous
Onsest (begins)	Acute	Acute	Acute	Acute
General infections symptoms	Pronounced	Pronounced	Pronounced	Pronounced
General cerebra symptoms	Pronounced	Pronounced	Pronounced	Pronounced
Focal symptoms	Characteristic	Characteristic	Characteristic	Characteristic
Changes of the CSF (lymphocytic, lymphocytic-neuyrophicpleocytosis, increased protein	Present	Present	Present	Present

Tick-borne encephalitis



Picture 12. Tick-borne encephalitis*(https://www.researchgate.net)*

MCB (A 84.0, A 84.1, A 84.8, A 84.9)

1. A 84.0 – Far East tick-borne encephalitis.
2. A 84.1 – Central Europe tick-borne encephalitis.
3. A 84.8. – Other tick-borne encephalitis.
4. A 84.9 – Tick-borne viral unspecified.

Tic-borne encephalitis is determined with specific arbovirus with neurotropic influence. Tic-borne encephalitis is a natural focal zoonosis that is found in the Far East, Siberia, the Ural Mountains, Eastern Europe, Transcarpathian region, the Volga region (Russia), Belarus ant the Baltic countries. Ixodid ticks are transmitters of viruses.

Infection occurs by the following ways:

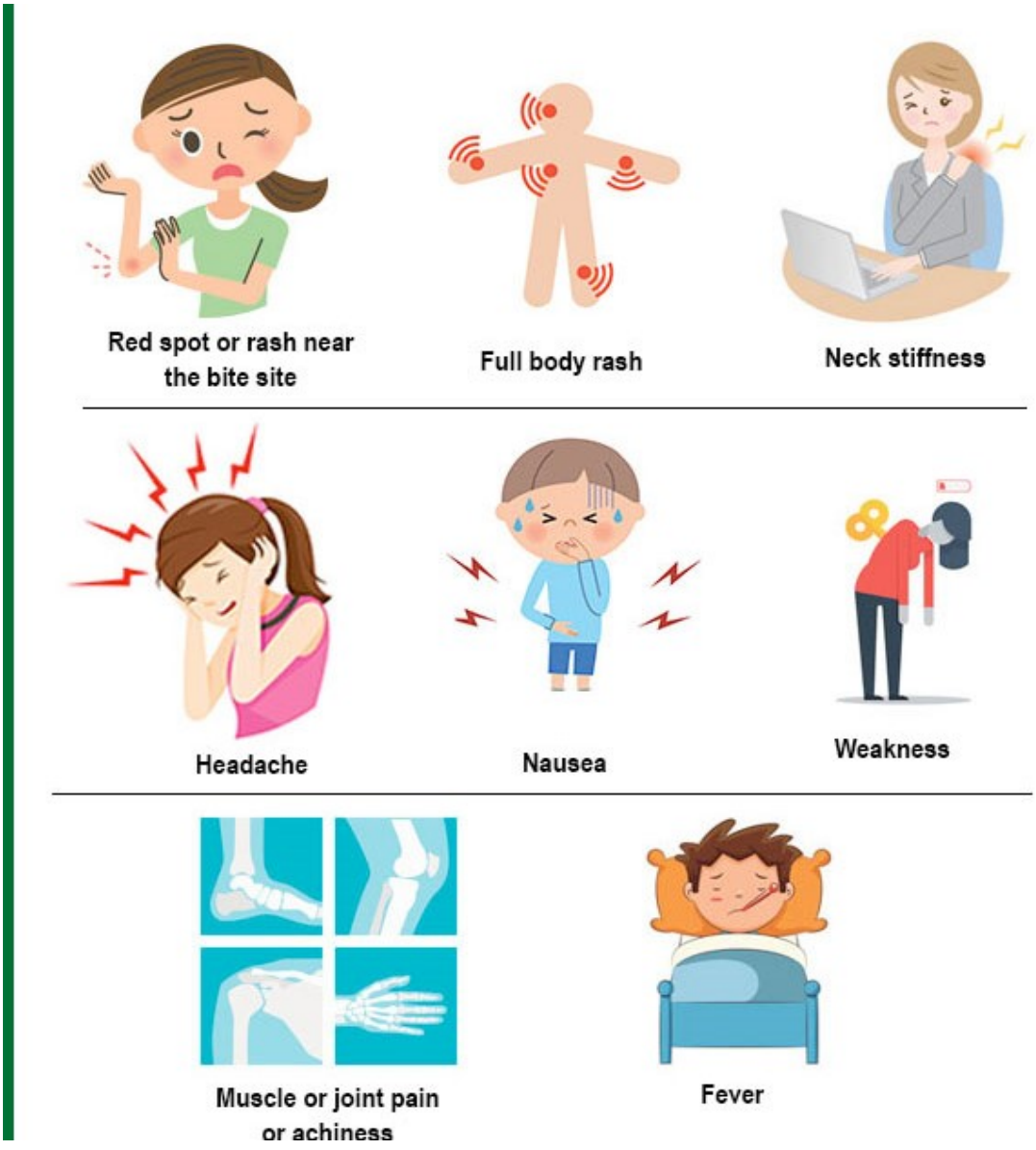
- transmissible (via a tick bite in park, forest);

- alimentary (while consumption raw of milk of goat or cow, which are infected with virus that enters the body of the animal after the tick bite).

In nature the virus reservoir are rodents (field mice, hedgehogs, rabbits, etc), birds. A tick, biting them, sucks the blood of animals with the virus and then biting a person or a goat transfer the parasite.

The disease is seasonal. Person can get sick tick-borne encephalitis in last spring is early summer months, due to the period of tick's activity.

Symptom tick-borne encephalitis.



Picture 13. Symptom tick-borne encephalitis*(https://www.uab.edu)*

The clinical classification.

I. The acute form:

1. Undetermined (febrile).

2. Meningeal (syndrome of serous meningitis).

3. Meningoencephalitic (cerebral).

4. Polyomyelitic.

5. Polyencephalomyelitic.

6. Polyradiculoneuritic.

7. Mixed.

II. The chronic form:

1. Amyotrophic:

1.1. With the polyomyelitic syndrome.

1.2. With the syndrome of the lateral amyotrophic sclerosis.

2. Hyperkinetic:

2.1. With the syndrome of Kozhevnikov's epilepsy.

2.2. With the syndrome of myoclonus-epilepsy.

2.3. With the chorea-epilepsy syndrome.

2.4. With the myoclonia syndrome.

3. Encephalomyelitic.

3.1. With the syndrome of the disseminated encephalomyelitis.

Clinical features.The disease begins suddenly with general infectious symptoms (raising the temperature up 39-40^{0}C, myalgia, fever) and cerebral (headache, vomiting, impaired consciousness) symptoms.

Do to predominant of nervous system structure disturbance there are the following forms of encephalitis.

*Poliomyelitic form*is characterized by flaccid paralysis of the muscles of neck, shoulder girdle and proximal upper extremities. Patients cannot raise their

hands up, in sides, flex, and extend the arm in elbows. A typical sign is “handing head symptom” because of neck muscles weakness. Bulbar syndromes with dysarthria, dysfagia, atrophy of tongue muscles are marked.

Meningeal form: there are meningeal syndrome (stiff neck, Kernig’s and Brudzinsky’s symptoms) and cerebral symptoms.

Meningoencephalitic form: focal cerebral syndromes are joined to meningeal form signs: cerebral paresis, hyperkinesis.

Repairing of motor functions can be complete. Nevertheless, weakness and atrophy in the muscles of neck, shoulder girdle may be persisted.

Poliradiculoneuritis form: focal symptoms are represented by radiculoneuritis.

The reconvalescent period is 2 years, transition to the chronic progradient form (preservation of the active virus in the nervous system is observed infrequently).

Chronic course:

1. Kozhevnikov epilepsy – 1-6 month after acute illness occurs myoclonus in certain group of muscles and pereodic epileptic attacks.

2. Poliomyelitic form – manifects itself during the acute period: peripheral paresis.

3. Amyotrophic lateral sclerosis (ALS) syndrome: signs of ALS and “Handing head” syndrome.

Treatment.

1. Early admission to a specialized neurological or infection hospital is compulsory, compliance a strict bed regime for a period of fever and 7 days after normalization of body temperature.
2. In the acute period:

- in the first 3 days serotherapy with Unencephalitic human donor immunoglobulin in amount of 3-6 ml 2-3 times s day, convalescents serum

(people who had tick-borne encephalitis), specific hyperimmune gamma-globulin, placental gamma-globulin are applied;

- prednisolone 1 mg per 1 kg of the patientdepending on the severity of disease;
- antibiotics – according to indications;
- antiedemic drugs (mannitol, Lasix), Ringer's solution, antihistamines, pain relievers;
- complex B vitamins, vitamin C;
- in severe respiratory failure and bulbar disorders – intensive care, according to indications – the false lung ventilation.

3. In recovery period: anabolic hormones, biogenic stimulants, nootropics. Cerebrolysin et.al. If it is necessary, anticholinergic, massages, treatment with body position are applied. In epileptic syndrome – anticonvulsants.

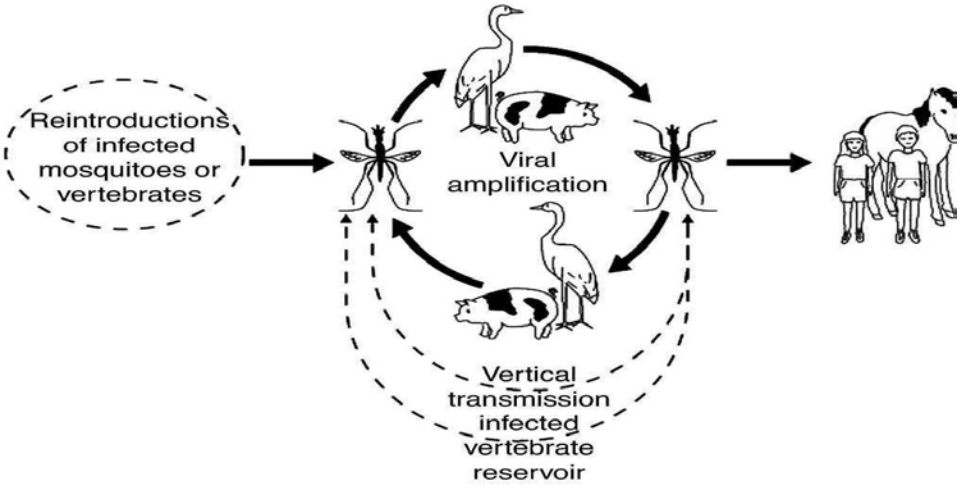
Prevention.Vaccination is used as a specific prevention. To form immunity in early epidemical season, the first dose is administered in autumn, the second – in winter. Urgent scheme (two injection of interval in 14 days) is used for unvaccinated persons who come in endemic foci in spring-summer period. Immunity develops within 2-3 weeks.

Economo's epidemic encephalitis (lethargic encephalitis)

Is the primary encephalitis with unknown pathogen.

In the course of the disease,there are two stages: acute and chronic.

The acute stage is characterized by inflammatory changes localized mainly in the gray matter of the 3-d ventricle walls, reticular formation, hypothalamic area and the area of oculomotoror nerve nuclei. In chronic stage, the morphological changes are degenerative in nature and often localized in nucleus striatum, nuclei of the hypothalamus, brain stem and substantianigra.



Picture 14. Economo's epidemic encephalitis*(https://www.ava.com.au)*

Acute phase. Its duration is weeks – month or 1 year. The typical Economo's triad includes the following:

1. Feverish state.
2. Oculomotor disorders: diplopia, ptosis, strabismus divergent, convergence and accommodation paralysis, paralysis of vertical gaze, Argyle-Robertson reserve symptom (maintaining the pupils reaction to light in absence of pupils reaction to convergence and accommodation).
3. Hypersomnia (lethargy or sleep formula violation: the patient sleeps during the day and at night he has insomnia).

There are some clinical forms of acute stage:

- Classic (oculolethargic)
- Vestibule-ataxic (vertigo, nystagmus)
- Hyperkinetic
- Abortive

There are following autonomic disorders: hypersalivation, hhyperhydroosis and facial greasiness. Hyperkinesias, consciousness impairment, delirium, hallucination, depression may occur.

Chronic phase.

1. Parkinsonian syndrome: bradykinesia, general stiffness, tremors of hands and lower jaw, flexed posture, slowed speech.

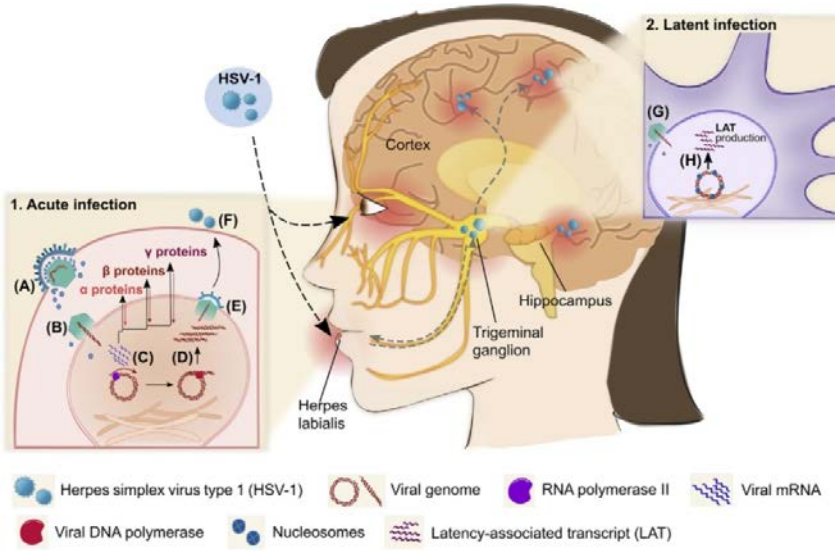
2. Hyperkinetic syndrome: myoclonus, athetosis, muscle dystonia phenomenon.
3. Neuroendocrine disorders.

*Treatment.*There is no specific treatment. Detoxication, desensitizion therapy, nootropic agents are applied. In case of parkinsonian syndrome – antiparkinsonial drugs.

Herpetic encephalitis

Herpetic encephalitis caused by herpes simplex virus type.

Damage to the nervous system by herpesovirysesis primarily represented by herpetic encephalitis (HSV1), herpetic meningitis (HSV2), ganglionnitis, polyradicoloneuritis, myelitis (VHZ).



Picture 15. Herpetic encephalitis*(https://simdos.unud.ac.id)*

Table 4.

Pathogen.	Herpes siplex viral
Seasonality	Polyseaonality
Route of infection	Hematogenous, neural (retroaxonal)
Incubation period	8-20 days during primary infection (30%)
The presence of herpetic eruption	Only in 10-15% cases
General infection symptoms (fever and over)	Are pronounced
General cerebral signs	Expressed

Typical focal symptoms	Aphatic disorders, behavior changes, impaired sense of smell and taste, hemiparesis, epileptic reizures
Changes of CSF	Moderate lymphocytic of lymphocytic neurophilicpleocytosis. Moderate protein increased

Clinical picture.The symptomatology of the herpes encephalitis develops acutely and includes the febrile body temperature, headache, weakness, and vomiting. Sometimes there are the signs of the upper respiratory tract infection. The febrile period can last some days or weeks. In certain cases the fever can have two-phase character. The neurologic symptomatology develops acutely or gradually depending on localization of the foci in the temporal or frontal lobe. The focal symptomatology is characterized by the wide polymorphism and includes the aphasia, amnesia, disturbance of behavior,olfaction, taste, vegetative implications, and also complex partial seizures. Often there are the hemiparesses, hemianopsia, lesion of the cranial nerves, meningeal symptoms, and papilledema. In an early stage most of patients has epileptic attacks and disturbances of consciousness, which can lead to the coma. In the absence of treatment, and if the process progresesse, the coma causes death in 50-70% of cases.

Diagnosis: lumbar puncture with detection of herpes virus DNA in cerebrospinal fluid by PCR (Polymerase chain reaction) method. MRI of brain detects the foci of hyperdensity with brain tissue edema.

Treatment. The pathogenetic and symptomatic therapy of the herpes encephalitis is aimed to maintenance of function of the external respiration, cardiovascular activity, water and electrolytic balance, prophylaxis and treatment of the secondary bacterial and trophic complications, and deep vein thrombosis of the lower extremities. For depression of the intracranial hypertension they use osmotic diuretics, barbiturates, carry out the APV in the hyperventilation regimen; in the epileptic attacks they prescribe antiepileptic preparations. The efficiency of corticosteroids in the case of the herpes encephalitis is doubtful. The specific

preparation of choice is the acyclovir (virolex, zovirax), which is used IV in a dose of 10 mg/kg 3 times a day for 10-14 days. If the PCR in the course of the treatment is positive, the therapy with acyclovir they continue for 3 weeks. At relapse of the disease it’s recommended to enlarge the dose to 15 mg/kg 3 times a day for 3 weeks.

SECONDARY ENCEPHALITIS.

The secondary encephalitis are specific complications occurring against the basic disease.

Classification

There are the following forms of the secondary encephalitis:

1. Parainfectious (postexantemic): in the measles, chicken pox, rubella, scarlet fever, and poliomyelitis.
2. Postvaccinal: after the ADPT, antirabic, polimyelitic, and diphtheria vaccinations.
3. Pheumatic.
4. Influenzal.
5. Toxoplasmic.
6. Fungic.

Clinicopathogenetic signs of the secondary encephalitis

1. The infectious-allergic nature of occurrence; the virus plays a trigger role for start of immunopathological reactions and formation of autoantibodies against proteins of the myelin and microglia, nevertheless the immediate cytopathic effect of the virus is possible.

2. The pathomorphologicpictury is characterized by the perivenous macro- and microglial infiltration, the demyelination foci; it testifiles to the secondary (para- or postinfectious) character of encephalitis and important role of immune reactions in its pathogenesis.

3. The diffuse lesion mainly of the white substance of the brain, especially the brain hemispheres, rarely of the brain stem and spinal cord, and in the chicken pox – of the cerebellum, parenchymatous – subarachnoidal hemorrhages are also possible.

4. There is no dependence between severity of the main infection course (exantemic or ARVI) and enchephalites, the last can hardly occur and have the severe course both in severe and mild forms of the main disease.

5. The acute beginning of development of the secondary encephalitis takes places more often on 3-8 day after the development of basic disease (with the rash in the exantemic encephalitis). The encephalitic symptoms often appear a short-term (some hours) improvement of the condition.

6. For clinical picture of the secondary encephalitis the triad of symptoms is characteristic: disturbance of consciousness (from the somnolence to coma), seizures (mainly generalized), and focal symptoms (pareses, paralyses).

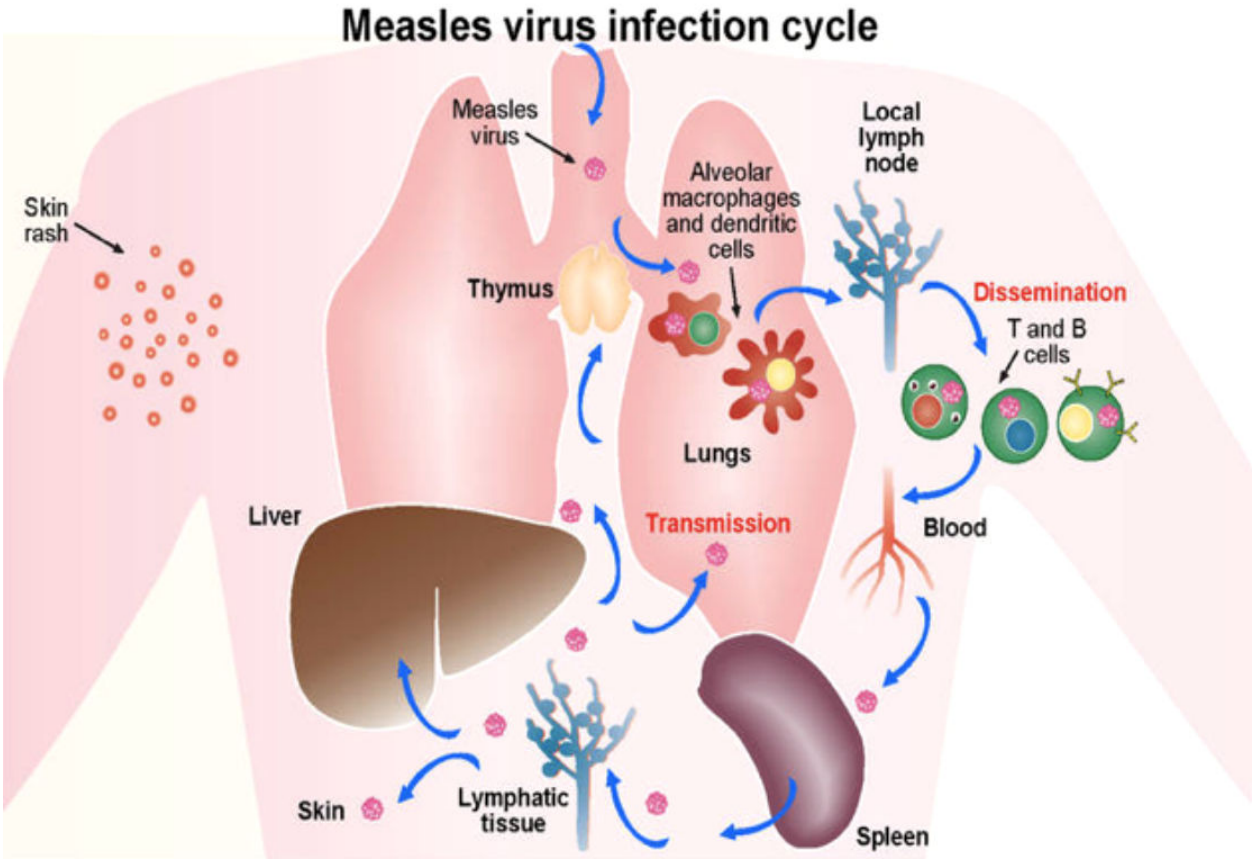
7. The general principles of laboratory diagnostics of the secondary encephalitis are based on determination of the virus DNA or RNA, specific antibodies of the class M and G in the cerebrospinal fluid by the ELISA method. Of clinical importance is the increase in the titer of intrathecal antibodies (IgG) in the dynamics of the disease and increased intrathecal antibody index in the ratio of the CSF/serum.

Encephalitis in the Measles

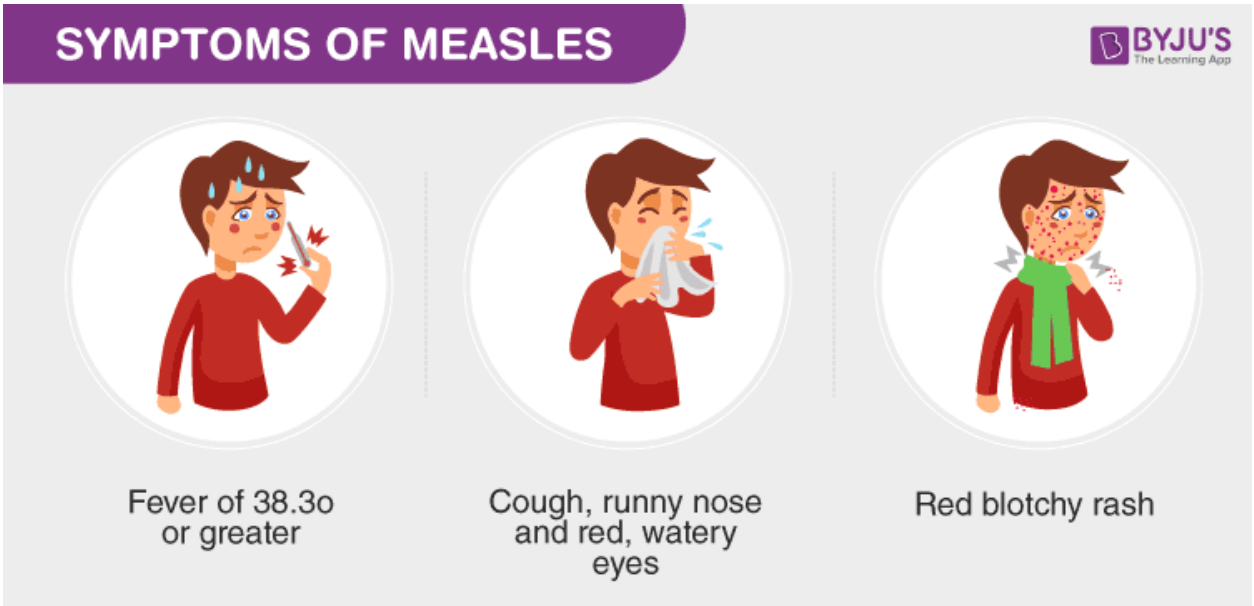
The lesion of structure of the CNS in the measles occurs more often than any other infections, and by the nature it belongs to the infectious allergic encephalitis. The viral causing this disease and organism allergization with change of its immunobiological reactivity is the reason of development of neurologic complication in the measles.

Clinical picture. The encephalitis in the measles can develop at any stage and in any severity of the measles, mainly on the 5 day after rash appearance. Clinically the disease is characterized by the high temperature (to 39-40^{0}C), bad headache, vomiting, and fast increase of the cerebral and focal neurologic symptoms. It may be the disturbances of consciousness - from the somnolence, disorientation,confusion up to the spoor and coma. Often there are neurological symptoms, such as the retrobulbar neuritis, nystagmus, signs of lesion of the optic, oculomotor, and abduncent cranial nerves, ataxy, agnosia, aphasia, focal convulsive paroxysm, pareses, and paralyses. The character of focal neurologic symptomatology depends on localization of the demyelination foci in the brain,

which are formed in the encephalitis in the measles. Also the white substance of the spinal cord is affected, and there is the transformation of measles encephalitis into encephalomyelitis, which is shown by the lower spastic paraparesis or paraplegia, disturbance of sensitivity on the conductive type, and dysfunction of organs of the small pelvic.



Picture 16. Encephalitis in the Measles*(https://asm.org)*

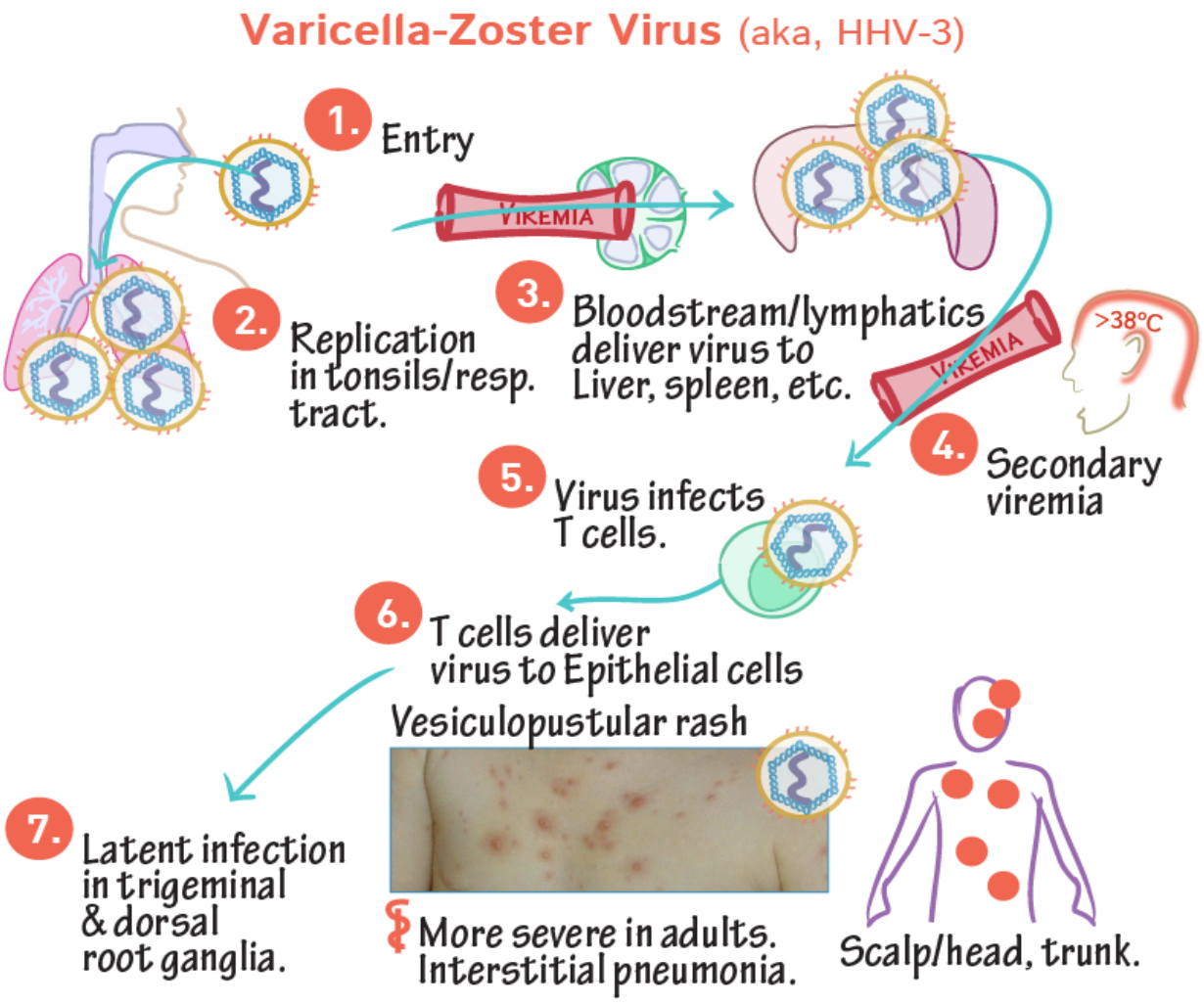


Picture 17. Symptoms of measles *(https://www.franciscanhealth.org)*

Diagnostics. The etiological diagnostics of neurological lesions in the measles is carried out by means of the following methods: isolations of RNA in the CSF and blood, the serological method – identification of specific antibodies to the pathogen of class M and G in the cerebrospinal fluid and blood, and also the method of pared serum (diagnostic significance is the increase in antibody titer in 3-4 times in the dynamics of the disease).

Treatment. There is no specific treatment of the encephalitis in the meales today. They carry out the pathogenetic and symptomatic therapy with use of glucocoticoids, hyperosmotic, vascular, and nootropic preparations in high doses.

Encephalitis in the herpes zoster



Picture 18. Varicella-Zoster virus*(https://quizlet.com)*

Encephalitis in the herpes zoster is the complication resulting from the unifocalvasculopathy is some weeks (3-8 weeks) and even months (to 6 months) after the herpes zoster reactivation. The encephalitic symptoms can gradually accrue or suddenly appear: with the fever, headache, cognitive disturbance, and generalized cramps. The disease can be characterized by different courses: monophasic, relapsing, progressing with death in the 25% patients. In the type of encephalitis in a part of patients are determined the moderate lymphocytic pleocytosis (100-200 cels in 1 μl), insignificant increase of the protein level. On

the brain CT and MRT there are areas of infarct. According to the angiography the damage of the middle and anterior cerebral arteriesmost often is found. In the case of trigeminal spread of the virus retinal arteries are mainly affected.

Multifocal vasculopathy

Multifocal vasculopathy (the encephalitis of the small vessels) is, as a rule, observed in the immunocompromised patients (HIV-positive people) and occurs without precursors – the pevious rash. The neurologic symptomatology is developed slowly, without intoxication; in includes the hemiplegia, aphasia, and lesion of the cranial nerves.

In other cases the disease begins acutely with high body temperature, intense headache, disturbance of consciousness, vomiting, cramps, and lesion of the cranial nerves. The herpetic rash apperars earlier than the neurologic implications.

Results of CSF study testify to the insignificant lymphocytic pleocytosis, normal or moderately increased level of proteins, and normal level glucose. The ischemic and hemorrhagic infarcts in the cortex and subcortical area (the gray and white substance), demyelination foci, the sizes of which depend on prevalence of damage of small vessels, are determined on the CT and MRI.

Diagnostics. The etiological diagnostics of neurologic lesions in the HIV-infection includes:

1. Determination of DNA of a virus in the cerebrospinal fluid by the PCR method. Nevertheless, if the HIV is reactivated in the spinal ganglia, which anatomically aren’et connected with the subarachnoid space, the PCR can be negative, and then diagnostics value is got by connection of the herpetic rush with the neurological symptomatology.
2. Identification of antibodies to the pathogen: IgM (during the acute period) and IgC (from the 14 day of the disease) in the cerebrospinal fluid. Increase of the titer of IgC antibodies of the disease dynamics, and the increase in the index of intrathecalIgC antibodies in the ratio of the CSF/serum have a certaion meaning.

3. Identification of virus antigens by the immunofluorescence assay (quiktesr) in smears from the vesicles.

Treatment. The causal treatment of the chickenpox encephalitis provides using of acyclovir, (valtrex, zovirax). At resistence of viruses to acyclic=over the teenagers and adults are prescribed foacarnet in the initial dose – 180 mg/kg a day IV within 1,5-2 hours every 12 hours. The maintenance dose is 90-120 mg/kg a day up to the clinical recovery. The course of the antiviral therapy last from 7 to 21 days depending on the disease severity and immune status of the pftient. As a control of efficiency of the antiviral therapy the DNA level of virus and toter of IgG antibodies is cerebrospinal fluid serves.

Influenza encephalitis

The lesion of the nervous system in the flu is taken place rather seldom and occurs, as a rule, against the serious course of this infection.

Clinical picture. The clinical picture of the influenza lesion of the nervous system is characterized by the neurotoxic syndrome, namely the vegetative disorders in the form of tachycardia, cyanosis, dryness of the skin and mucosa's, and increased arterial pressure. In some hours (or days) these symptoms are changed on the contrary – the parasympathetic dysfunction dominates. Such symptoms mainly disappear after the normalization of body temperature, but in some patients with flu they are kept and once the clinical manifestations of infectionaquire the character of unfluenzakcomplicarion: the postinfluezalvegetovascular dystonia occurs. The dystonic symptoms (the weakness, sweating, sleep disorder, loss of apperite, and sleeplessness) are combined with lability of the pulse, unstable blood pressure, and tachycardia. Disturbances in the emotional sphere (the tearfulness, irritability) are often observed.

Diagnostics of complications of the flu is based on the epidemiological data, symptoms of the disease, and results of the laboratory studies (virologic, serological test). The neurologic disturbances develop during the flu epidemic, in

the presence of close contact to a sick person. The diagnosis is confirmed by the immunologic and virusologic studies (isolation of a virus, positive hemagglutinationingibition test or compliment fixation test, high antibody titers to influenza).

Treatment. In the period of the acute course of severe forms of lesion of the nervous sustem due to the flu it is necessary to use the specific anti-influenza serum or hyperimmune gamma globulin for 3-4 days intramusculary in a dose of 3-6 ml with intervals of 6-12 hours. Also human leucocytic interferon is used (5-8 time per day).

Postvaccinal encephalitis

The postvaccinal encephalitis can develop after introduction of vaccines of ADTP, DT, antirabic, and most often after introduction of the measles vaccine. The leading cause in development of the postvaccinal encephalitis are autoimmune mechanisms. The poatvaccinal encephalitis occurs at any age, but mainly in 5-15 years against an interccurentdic=sease, hypothermy, overload, in 5-20 days after introduction of the ADTP, or in 10-20 day after carrying out the antirabic vaccination. Very seldom the postvaccinal encephalitis oe encephalomyelitis is observed after use of other vaccines. Pathomorphologically it is characterized by the expressed vascular disorders (hemorrhages, stasis), and during the remote period – with the dystrophic changes of neurons.

Clinical picture. The lesion of the nervous system after vaccination against the radiesis characterized by occurrence of the infrequent, but extremely severe secondary demyelinating multifocal meningoencephalomyelopolyradiculoneurites. The symptom, as a rule, occur on the 12-13 day after the inoculation (sometime on the 8-25 day) under the influence of provocative factors (overheat, intercurrentdiseases, overfatigue) acutely or subacutely. The main implications ara the pain radiculineuritic syndrome, headache, paresthesias, hallucination, seizures with the subsequent development of the myelitis (the transversal myelitis, ascending Landry’s paralysis) and encephalitis. In the CSF the lymphocytic

pleocytosis and in blood the neuriphilicleucocytosis is determined. In the fulminant course of the encephalomeningitisas a result of antirabic vaccination the fatal outcome on the 8-10 day is possible. Th ascending Landry's paralysis is dangerous because of respiratory disturbance. The transversal myelitis is characterized by a long course, which is complicated by the resistant lower paraparesis, malfunction of pelic organs.

The encephalomyelitis after vaccination against the chickenpox also develops under the influence of provocative factors, more often than the interccurent infections. It has an allergic character on the introduction of foreign protein, and its course is similar to the meningoencephalitis or encephalitis, rarely to the myelitis.

When carrying out the vaccination against the pertussis the complication are observed quite seldom; sometimes they are characterized by development of generalized attacks, seizures of consciousness. In some children they are severe residual implications in the form of the polymorphic epileptic seizures.

Treatment they use glucocoticoids, immunoglobulin G, carry out detoxication, prescribe antihistamine preparation, and symptomatic therapy. In sevrecases they apply the intensive care measures. The absolute recovery after the carried-out desensitizing therapy comes rarely.

Parainfectious encephalitis

Parainfectious encephalitis occurs alongside with childhood infections: measles, rubella, chickenpox, as infectious, allergic process.

In occurs in 3-5th days after eruption.

Clinical manifestationsare hyperthermia, consciousness impairment, meningeal signs, seizures. Cerebellar and vestibular disorders, paresis and dysfunction of the cranial nerves may occurs.

Changes in cerebrospinal fluid are non-specific. They are lymphocytic pleocytosis, a slight protein increase. The disease course is severe. Measles encephalitis mortality is 25%. Chickenpox encephalitis course is benigner.

Treatment.Corticosteroids, desensitizing and detoxification therapy.

Rheumatic encephalitis

The main forms of rheumatic brain lesions are:

- acute rheumatic meningoencephalitis
- chronic rheumatic meningoencephalitis
- rheumatic encephalopathy
- rheumatic vasculitis
- Sydenham's chorea (chorea minor)

There is diffuse lesion of cerebral cortex, subcortical nodes, brain steam and meningitis.in brain, there are vascular changes in forms of endarteritis, vasculitis, periarteritis.

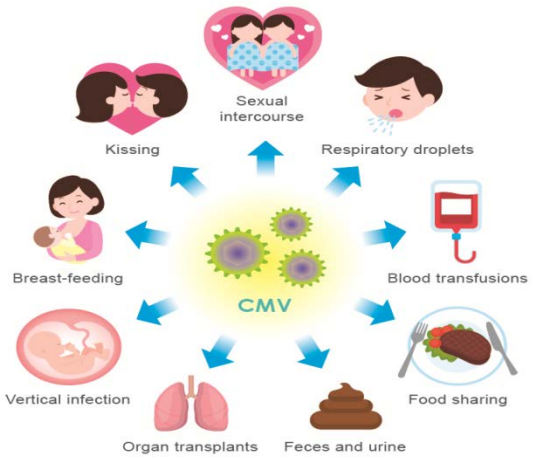
The clinical features of acute rheumatic meningoencephalitis are polymorphic, different focal symptoms (hemi- or tetraparesis, cranial nerve dysfunction, ataxia, aphasia, sensitivity disturbances, hyperkinesis) are characteristic.

Sydenham's chorea occurs in case of subcortical nuclei lesion. School-age children, mostly girls may get sick. There are psyche disorders (protervity, irritability), choreichyperkinesis: involuntary movements in forms of grimaces, excessive swinnding while walking. These children cannot maintain a given position for a long time (protruded tongue, closed eyes), speech is fragmentary, frequent eyelids blinking is marked. Proof of rheumatic carditis nature is arthritis, rheumatoid nodules.

Cytomegalovirus encephalitis

MCB 10 – Cytomegalovirus disease – B 25.

Cytomegalovirus – is the largest of the group herpes viruses. Infection can be carried out through contact with. An already infected person (the virus is found in saliva urine, semen, breast milk), as well as blood transfusion and organ transplantation. The disease caused by the virus can be associated with both primary infection and reactivation of latent infection.



Picture 19. Cytomegalovirus encephalitis*(https://www.cureus.com)*

Clinical features.Cytomegalovirus infection may present with infectious mononucleosis syndrome. Encephalitis of cytomegalovirus may present with fever, headache, drowsiness, impaired attention, memory, personality changes, epileptic seizures, dementia, focal neurological disorders.

In patient with AIDS may develop various variants cytomegalovirus infection than affect the central and peripheral nervous system (meningoencephalitis, myelitis, poly radiculopathia) 5-10%patients have retinitis, which initially leads to a unilateral decrease in vision in the absence of treatment to complete blindness.

Diagnostic.Is based on the isolation of the virus from urine, blood, CSF as well as serological tests that detect antibodies to the virus. CSF – lymphocytic pleocytosis high protein. CT often does not reveal pathology, MRI can defect defuse or multifocal lesions of the white matter. The most decorate diagnostic method at present in PCR which defect virus DNA in CSF.

Mosquito encephalitis (Japanese)

MCB 10 - Mosquito encephalitis – A 83.

Mosquito encephalitis - acute infections disease, with transmissible transmission is caused by a neurotropic virus, carried of which is a mosquito.The onset of neurological symptoms may be sudden or follow a prodrome lasting 3-4

days, characterized by headache, myalgia, fever, pharyngitis, gastrointestinal manifestations.

Etiology.RHA-contaningflavivirus – Encephalophilis japonicas.

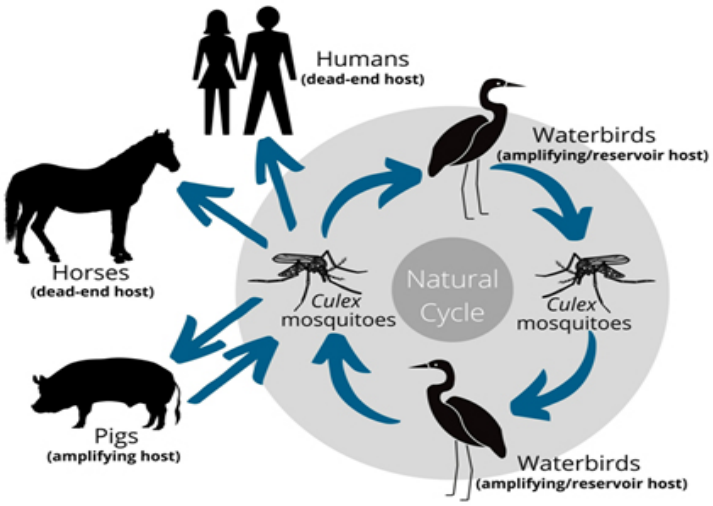
Raute of infection: transmissible mosquito bite.

Reservoir in nature: wild and domestic.

Seansonality – summer-autumn period.

Clinical features.The most acute period with general infectious, mental and cerebral syndromes, against which various focal symptoms are observed.

Incubation period – 4-14 days.



Picture 20. Mosquito encephalitis (Japanese)*(www.ava.com)*

The prodromal period is rase, has no characteristic features: headache, malaise, chills, loss of appetite, nausea.

Headache gradually increases, develop of stiff of neck, deep reflexes are brisk or reduced tremor of fingers, lips, of the tongue, ataxia, lesion of pelvic organs, damage to the cranial nerves. In severe cases delirium and depression of consciousness develop,fleash to coma. The resulting fever from the first day is resolved after 10-14 days. Bradycardia is rare.

General infectious syndrome: fever to 39,0-40,0^{0}C from the first days of illness lasting 7-10 days, chills, flushing of the face and conjunctiva, bradycardia, replaced by tachycardia, tachypnes, leucocytosis, lymphopenia.

Convalescent stage – 3-8 weeks.

Meningeal syndrome positive.

Characteristic of cerebral derangement are: consciousness disorders: spoor, phychomotor agitation in the acute period, remaining in the subacute phase and the period of convalescence partial and generalization clonic – monicparaxim and epileptic status.

Blood test – leukocytosis, CSF – lymphocytic glucose – normal, pleocytosis sometime – hyponatrieia (inadequate secretion of antidiuretic hormone). The disease is more common in children, possible severe residual neurological defect mental retardation.

Diagnosis:the diagnosis can be verified by isolating the virus from the blood, **CSF brain tissues.**

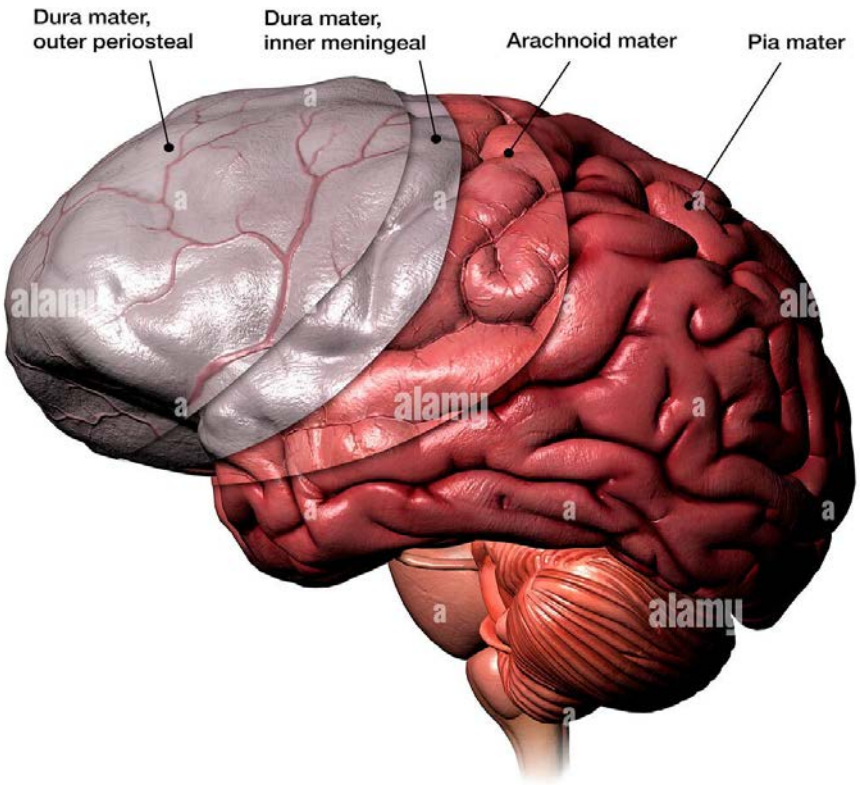
Differential diagnosis: epidemic encephalitis, tick-borne encephalitis, hemorrhagic fever.

Treatment:corticosteroids, specific j-globulin, detoxification, dehydration and symptomatic therapy.

Prevention:complex of anti-mosquito measures.

CEREBRAL ARACHNOIDITIS

The cerebral arachnoiditis is the inflammatory disease of the brain meninges, mainly arachnoid. The right using of the term “arachnoiditis” has still remained a debatable issue, however pathoanathomical changes in the brain as well as modern diagnostics techniquies (the CT, MRI) don’t deny its right to exit. The course of the arachnoiditis can be acute, subacute, and chronic. As a rule, the chronic arachnoiditis is determined more often.



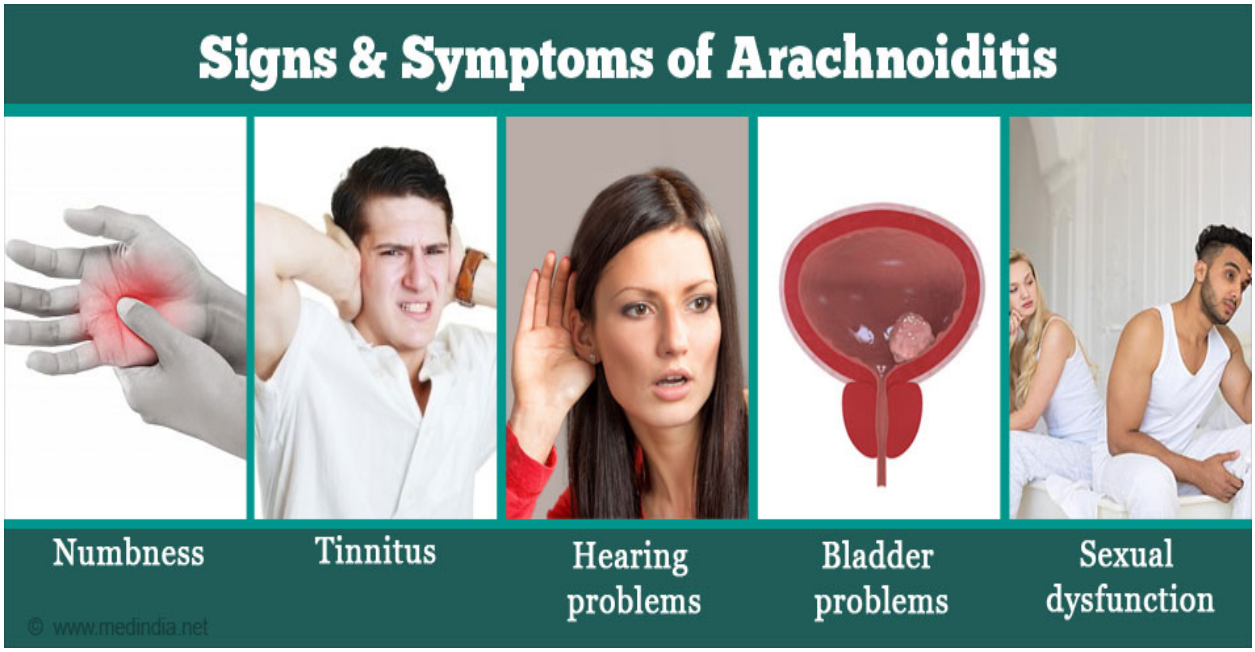
Picture 21. Brain *(https://www.brainandlife.org)*

Etiology. The most common causes of the cerebral arachnoiditisare: the local and general infections (consequence of the postponed serous of purulent meningitis, flu, rheumatic disease, measles, scarlet fever, and pneumonia); the disease of the paranasal sinuses (the adenoid disease, rhinosinusitis), or the ear (otitis). This list

may be added with the cranicerebral trauma, hemorrgages into the subarachnoid space; rarer the chronic intoxications occur.

Classification. There are the diffuse (rare) and focal form, which depend mainly on the localization of pathological process: the convexital, basal arachnoiditis (optico-chiasmal, intercrural, arachnoiditis of the transverarsalcistem, or of the pontine-cerebellar angle), arachnoiditis of the posterior cranial fossa, or the large occipital cistern.

Clinical picture. The clinical picture of the cerebral arachnoiditisis formed by the general cerebral disturbances and focal symptoms. The general cerebral disturbances depend on the intracranial hypertension or CSF hypotention; the focal symptoms depend on the localization of the pathological process. The cerebral symptoms first of all include the continuos or paroxysmal squeezing headache radiating to the eyeballs, neck, teeth, aggravated by a physical activity, and fast movements (a jump symptom); dizziness, often with nausea and vomiting. There is the irritability, weakness, fatigue, memory loss, sleep disorders, and fluctuation of the BP.



Picture 22. Signs and Symptoms of arachnoiditis *(https://www.medindia.net)*

Focal arachnoiditis

The convexitalarachnoiditismost often occurs after the craniocerebraltraumatorinfectionandis characterized by prevalence of implication of an irritation over the signs of function loss. Together with the general cerebral (the headache, dizziness, sleep disorders, memory loss, and weakness) symptoms there are signs of the vegetovascular (the sweating, fluctuation of the arterial pressure, acrocyanosis, and hypersensibility to the weather changes) and pyramidal signs (the anisoreflexia, depression of the abdominal reflex, and indistinct pathological signs), disturbance of functions the VII and XII pairs of the cranial nerves, and sensitivity disorders. One of the main symptoms of the arachnoiditis of this localization is the focal Jackson's and/or secondary-generalized epileptic seizures.

The basal (lesion of the meninges of the brain basis) is shown by the cerebral symptoms and lesion of the cranial nerves. One of the clinical forms of this localization is the optico-chiasmatearachnoiditis, which occurs usually as a result of the viral infection, after the acute stage of influenza, and sinusitis. It is characterized by the headache, mostly in the forehead, nose, eyeballs, feelling of the net in form of the eyes, reduced visual acuity, changes in visual field, and scotomas. They observe gradual changes of the olfaction; the vasculomotor and hypothalamic disturbances develop (the dermographism, theobesity).

The arachnoiditis of intercrural localization is characterized by malfunction of the third cranial nerves (the diplopia, strabismus, restriction of movements of the eyeballs, anisocoria, and weakening of the pupils reactions). Rarely there are the pyramidal and meningeal symptoms, the mild degree failure of functions of the VII pair of the cranial nerves.

In the arachnoiditis of the transversal cistern covering the corpora quadrigemine the leading symptom is the headache, which irradiates to the eyeball, nasal bridge, eyebrows area; there are symptoms of lesion of the vestibulocochlear

nerve with disturbance of the vestibular and auditory functions (depression of hearing, rotator dizziness, and spontaneous nystagmus); quite often the expressed symptoms of lesion of the V and VII pairs cranial nerves are observed. The cystic arachnoiditis of this localization can remined the clinical picture of the acoustic neurinoma.

The arachnoiditis of the pontine-cerebellar angle (arachnoiditis of the lateral cistem of the pons) is characterized by the moderately expressed cerebral symptoms and more determined focal signs (the headache in the occipital area, tinnitus, hearing loss, dizziness, sometimes vomiting, staggering, and falling). The V, VI, VII, and VIII pairs cranial nerves are affected. There is the pyramidal and more expressed implications of the pyramidal signs.

In the case of ***the arachnoiditis of the posterior cranial fossa*** the general cerebral symptoms prevail (the headache of the hypertensive hydrocephalic character in the occipital area and eyeballs, which gradually increases, dizziness, vomiting, papilledema). The focal symptomatology consists of the cerebellar symptoms, the signs of lesion of the cranial nerves (pairs V, VI, VII and VIII) and mildly expressed implications of the pyramidal signs.

It is possible to allocate ***the arachnoiditis of the large occipital cistern,*** where the arachnoidal cysts or fibrous tissue can be present. In this condition the outflow of the cerebrospinal fluid is broken; the clinically severe hypertension develops (with the headache increasing at turn of the head, fast movements, and cough). There are the meningeal, cerebellar, pyramidal symptoms and the signs of lesion on the IX, X, and XII pairs cranial nerves.

The diagnosis of the cerebelararachnoiditis is confirmed by data of the anamnesis (the craniocerebral trauma, inflammatory cerebral diseases, disease of the adnexal sinuses of the nose, ear, or ARVI) and the result of clinical examinations and modern additional methods of study (the CT, MRI of the brain, EEGand ophtalmoscopy).

The differential diagnostics of the cerebral arachnoiditis is carried out first of all (especially in the case of localization of process in the posterior cranial fossa) with the tumor, hematoma, and brain abscess. It is necessary to carry out the differential diagnostics with functional diseases of the nervous system, in particular neuroses.

The treatment is conservative and surgical. It's necessary to specify the etiologic, factors, which caused the disease (inflammatory or traumatic). They prescribe antibiotics of the broad spectrum of activity, corticosteroids, desensitizing, antiepileptics, dehydrational agents, and preparations for better microcirculation. The medicinal treatment of the arachnoiditesalso includes the vitamin therapy, immunocorrecting preparations, and neiroprotectors. In the cystic and adhesion arachnoiditis the surgery is appointed.

Picture 22. Treatment for arachnoidities *(https://www.icliniq.com)*

BRAIN ABSCESS

Brain abscess – local purulent inflammation of the brain, which occurs secondarily as a result of an untreated primary purulent lesion.

Etiology.

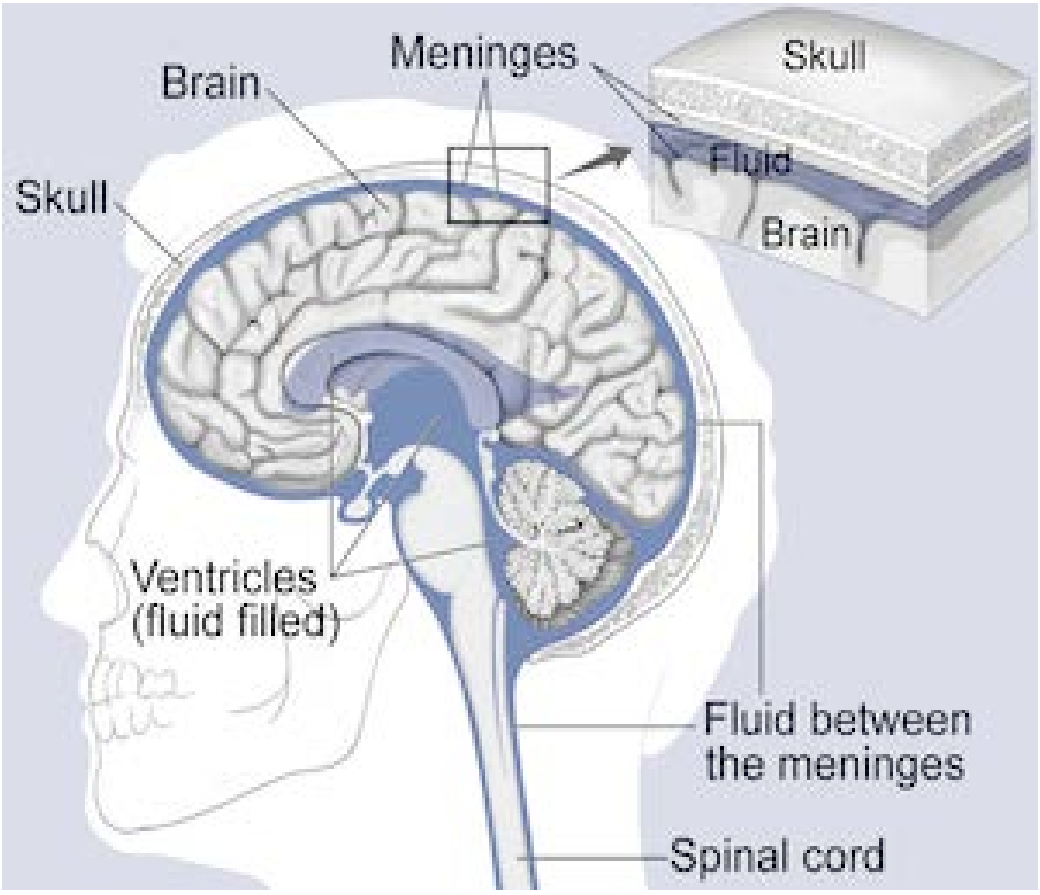
Direct Local Spread

A brain abscess can originate from infections in head and neck sites: otitis media (5%)and mastoiditis (secondarily cause inferior temporal lobe and cerebellar brain abscesses), paranasal sinus infection (approximately 30% to 50% as the reported cause), infection from frontal or ethmoid sinuses spreads to the frontal lobes, dental infection usually causes frontal lobar abscesses. Facial trauma, even from neurosurgical procedures, can result in necrotic tissue, and brain abscesses have been reported afterward. Metal fragments or other foreign bodies left in the brain parenchyma can also serve as a nidus for infection.

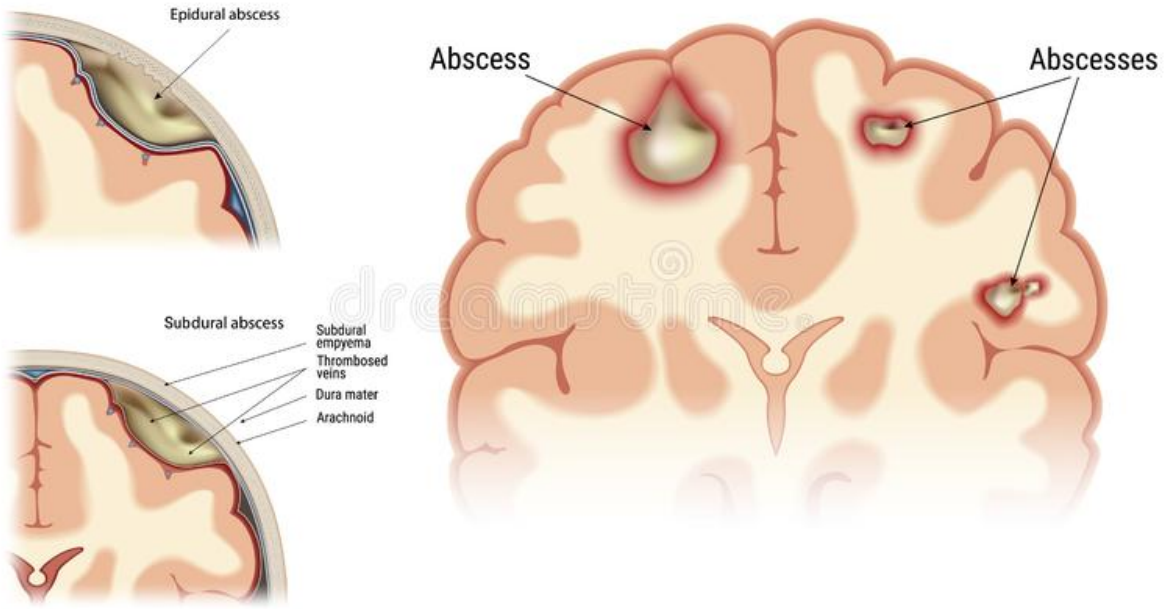
Generalized Septicemia and Hematogenous Spread

Various conditions can cause hematogenous seeding of the brain. Some common ones include lung, the most common associated organ; pulmonary infections, such as lung abscess and empyema, often in hosts with bronchiectasis; or cystic fibrosis from an important encountered cause. Others include pneumonia, pulmonary arteriovenous malformation, and bronchopleural fistula. Cyanotic congenital heart diseases in children are associated with more than 60% of cases. Bacterial endocarditis, ventricular aneurysms, and thrombosis are also among the causes. Skin, pelvic and intraabdominal infections also have been reported frequently as risk factors. Brain abscesses associated with bacteremia commonly cause multiple abscesses, mostly in the distribution of the middle cerebral artery and usually at the gray-white matter junction.

The most frequent microbial pathogens isolated from brain abscesses are Staphylococcus and Streptococcus. Among this class of bacteria, Staphylococcus aureus and Viridian streptococci are the commonest.



Picture 23. Brain abscess *(https://study.com)*



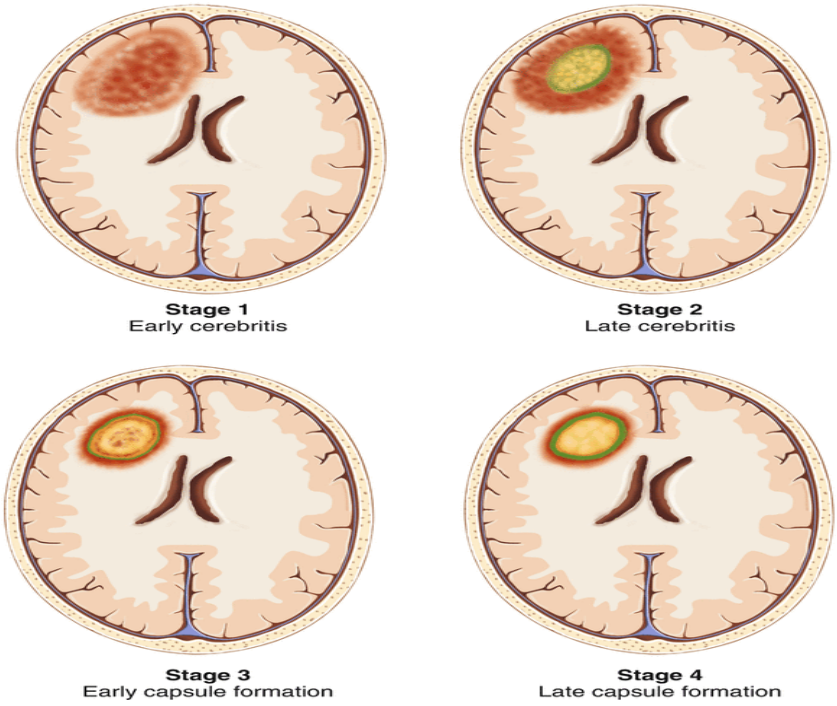
Picture 24. Brain abscess *(https://study.com)*

In about two-thirds of cases, symptoms are present for 2 weeks or less. The diagnosis is made at a mean of 8 days after the onset of symptoms. The course ranges from indolent to fulminant. Most manifestations of brain abscess tend to be nonspecific, resulting in a delay in establishing the diagnosis. Most symptoms are a direct result of the size and location of the space-occupying lesion or lesions. The triad of fever, headache, and the focal neurologic deficit is observed in less than half of patients. The frequency of common symptoms and signs is as follows:

- A headache the most common medical symptom.
- Mental status changes lethargy progressing to coma is indicative of severe cerebral edema and a poor prognostic sign.
- Focal neurologic deficits occur days to weeks after the onset of a headache.
- Pain is usually localized to the side of the abscess, and its onset can be gradual or sudden in nature. The pain is most severe in intensity and not relieved by over-the-counter pain medications.

- Fever
- Seizures can be the first manifestation of brain abscess. Grand mal seizures are particularly common in frontal abscesses.
- Nausea and vomiting are mostly seen with raised intracranial pressure
- Nuchal rigidity is most commonly associated with occipital lobe abscess or an abscess that has leaked into a lateral ventricle.
- Third and sixth cranial nerve deficits.
- Rupture of abscess usually presented with suddenly worsening headache and followed by emerging signs of meningismus.

Clinical features:the development of the disease usually has a gradual course, but it can also be acute.



Picture 25 Clinical featuresbrain abscess *(https://study.com)*

General infections symptoms: general weakness, fever feeling of heat, tachycardia. General symptoms are characteristic of the onset of disease, an increase in the focus contributes to an increase intracranial hypertension: there may be meningeal signs (stiff neck muscles, symptoms of Kerning, Brudzinsky,

confirmation of severe intracranial hypertension there may be congestive optic discs, consciousness).

Focal symptoms: depend on the localization of process:

- Subtentorial – hemyparesis, sensory lesion, aphasia, visual field loss, nystagmus, forsed head position.
- Brain steam – sings damage to the cranial nerves.

Differential diagnosis.

The main differential diagnoses include:

- Bacterialmeningitis
- Braintumors
- Demyelination
- Epidural/subduralabscess
- Encephalitis
- Fungal or parasitic infestations: Cryptococcosis/Cysticercosis
- Mycoticaneurysm
- Septicduralsinusthrombosis

Diagnostic: history taking, physical examination, detailed CNS assessment.

Routine tests: Complete blood count with differential and platelet count, erythrocyte sedimentation rate, serum C-reactive protein, serologic test, blood cultures (at least 2; preferably before antibiotic therapy).

Lumbar puncture: Rarely required and only should be performed with a prior CT and MRI scan after ruling out increased intracranial pressure because of the potential for cerebrospinal fluid (CSF) herniation and death. In circumstances of acute presentation of patients or suspicion of meningitis, blood cultures can be used for the initiation of antibiotic therapy. The results are mostly nonspecific, consisting of an elevated protein level, pleocytosis with the variable neutrophil count, typically a normal glucose level, and sterile cultures. A lumbar puncture in

the case of rupture when white blood cell (WBC) count becomes high in addition to elevated CSF lactic acid and abundant red blood cells (RBCs) in the CSF.

Stereotactic Computed Tomography (CT) or Surgical Aspiration

Samples obtained can be employed for culture, Gram stain, serology, histopathology, and polymerase chain reaction.

Computed Tomography

Imaging findings depend on the stage of the lesion. Early cerebritis often appears as an irregular low-density area that does not enhance or may show infrequent patchy enhancement. As cerebritis evolves, a more conspicuous rim-enhancing lesion becomes visible. Enzmann et al. reported that CT findings of patchy enhancement in early cerebritis evolve to a rim of enhancement in late cerebritis which later on forms the brain abscess. A key histopathologic difference is that rim enhancement of late cerebritis is not associated with collagen deposition as seen in an abscess where it surrounds a purulent cavity. Serial CT examinations in patients with late abscesses show progressively decreasing edema and mass effect. Brain abscess wall is usually smooth and regular with 1 mm to 3 mm thickness with surrounding parenchymal edema. The ring of enhancement may not be uniform in thickness and can be relatively thin on the medial or ventricular surface in the deep white matter, where vascularity is less abundant. Edema and contrast enhancement is suppressed by the administration of steroids. Multi-location with subjacent daughter abscesses or satellite lesions is frequently seen. Gas if presently is suggestive of gas-forming organisms.

Magnetic Resonance Imaging (MRI)

MRI is the imaging modality of choice for diagnosis as well as follow-up of lesions. It is more sensitive for early cerebritis and satellite lesions particularly those present in the brain stem as well as estimating the necrosis and extent of the lesion. It allows for greater contrast between cerebral edema and the brain and is

also more sensitive for detecting the spread of inflammation into the ventricles and subarachnoid space.

Conventional spin-echo imaging with contrast

Classic MR imaging findings of an abscess include a contrast-enhanced rim surrounding a necrotic core. Rim is T1 isointense to hyperintense relative to white matter and T2 hypointense. On MRI characteristic smooth tri-laminar structure of the rim on T2W imaging proves helpful in differentiating from other ring-enhancing lesions. Central necrosis shows variable hyperintensity on T2 depending upon the degree of protein content and hypointense on T1.

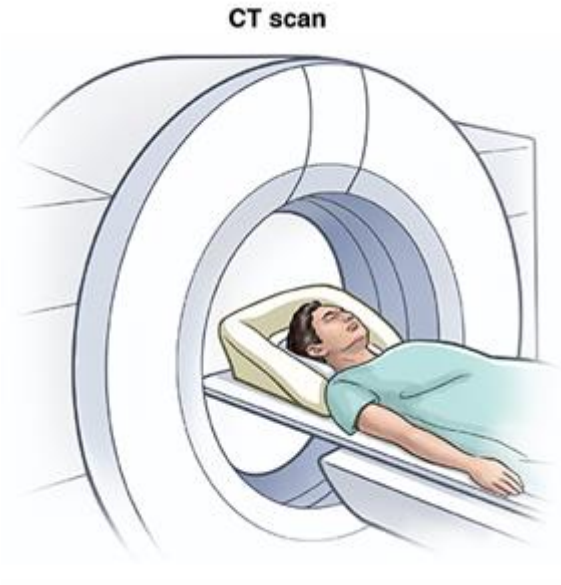
Diffusion-weighted magnetic resonance imaging

Diffusion-weighted imaging is capable of distinguishing brain abscesses from other ring-enhancing brain lesions. Abscesses are typically hyperintense on DWI (indicating restricted diffusion, characteristic of viscous materials, such as pus), while neoplasms like glioma as lack restricted diffusion appearing hypointense or variable hyperintense much lower than an abscess.

Diffusion-Tensor Imaging is based on three-dimensional diffusivity and commonly employed for the evaluation of white matter tracts. Fractional anisotropy, a quantitative variable is calculated by diffusion-tensor imaging. This variable reflects the degree of tissue organization and quite higher in abscess supposedly due to organized leukocytes in the abscess cavity.

Proton MR Spectroscopy probe tissue metabolism. Spectral analysis reveals elevated succinate, although not commonly seen is quite specific for an abscess. Other significant metabolites include elevated acetate, alanine, and lactate signals. Amino acids from neutrophil-driven protein breakdown suggest a pyogenic abscess. MR spectroscopy may be used to further differentiate anaerobic from aerobic metabolism by elevated succinate and acetate peaks which are only observed in anaerobic infections due to glycolysis and subsequent fermentation.

Also, lactate peaks are lowest in strict anaerobes owing to metabolic lactate consumption.



Picture 26. Stereotactic Computed Tomography (CT)*(https://www.hopkinsmedicine.org)*

Treatment

A brain abscess can lead to elevated intracranial pressure and has significant morbidity and mortality. Management can be divided into medical and surgical approaches.

Medical management can be considered for deep-seated, small abscesses (less than 2 cm), cases of coexisting meningitis, and few other selected cases. Usually, a combination of both medical and surgical approaches is considered.

CT and MRI brain guides in management by localizing the abscess and delineating details including dimensions and a number of abscesses. Usually, large abscesses (more than 2 cm) are considered for aspiration or excision based on the surgical skills of the operator. The approach for multiple abscesses includes a long course (4 to 8 weeks) of high-dose antibiotics with or without aspirations, based on weekly CT scanning.

The selection of an antibiotic regimen should be wisely made based on microorganisms isolated from blood or CSF. Certain antibiotics are unable to cross the blood-brain barrier and are not useful in treating brain abscess; these antibiotics include first-generation cephalosporins, Aminoglycosides, and tetracyclines.

Steroids can be considered in select cases, especially to reduce the mass effect and improve antibiotic penetration and cerebral edema.

The surgical approach has a pivotal role in the management of brain abscesses. The choice of the procedure depends on operator skills and preference. Approaches include ultrasound, or CT-guided needle aspirations via the stereotactic procedure, bur hole, and craniotomy for loculated multiple abscesses. Intravenous or intrathecal agents against specific microorganisms are considered with surgical therapy.

Complication

The complications that can occur secondary to a brain abscess are:

- Meningitis
- Ventriculitis
- Increasedintracranialpressure
- Brainherniation
- Seizures
- Septicemia
- Neurologicaldeficits
- Thrombosisofintracranialbloodvessels
- Death

MYELITIS

Myelitis is an inflammation of the spinal cord, in which both while and gray matter are affected.

Etiology, pathogenesis

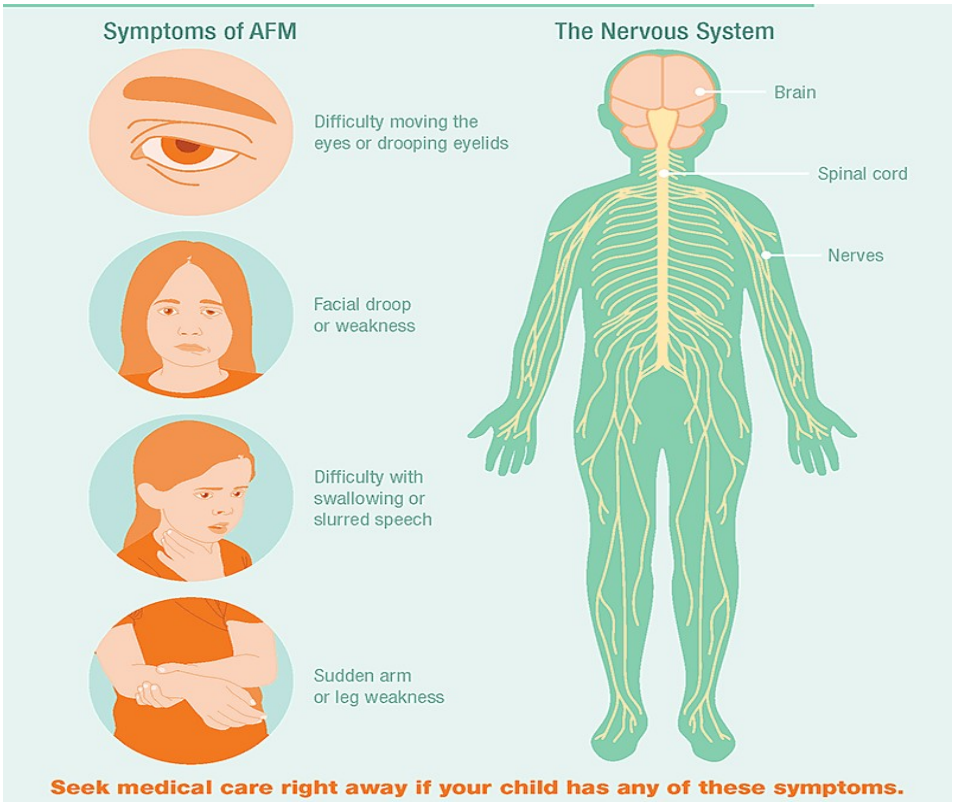
– Traumatic myelitis occur in the case of open and closed injuries of spine and spinal cord with joined of secondary infection.

– Secondary infectious myelitis. In their pathogenesis the autoimmune reactions and skidding of hematogenous infection from the infection focus to spinal cord play the main role.

– Post-vaccination myelitis.

– Due to severe exogenous poisoning or endogenous intoxication.

The clinical manifestation



Picture 27. Symptoms myelitis *(https://medizzy.com)*

The clinical manifestation of myelitis occur acute or subacute on the background of temperature increasing up to 38-39^{0}C, chils, malaise. Neutologic manifestations of myelitis begin with moderate pain and paresthesia in the lower limbs, back and chest, with radicular nature. Then motor, sensory and pelvic disorders appear, grow and reach their maximum within 1-3 days. The neurological symptoms character depends on the pathological process level.

Myelitis of uppercervical level of the spinal cord is characterized by spastic tetraplegia, phernic nerve disturbance with respiratory disorder, sometimes bulbar disorders. Sensitivity disorders are in form of conductive hypesthesiaor anesthesia with the upper limit, which corresponds to the level of theaffected segment. Pelvic disorders occur firstly (retention of urine and feces) passing into the central type of incontinence.

In case of spinal cord injuries on the cervical enlargement level the lower spastic paraplegia with conductive anesthesia develops. Pelvic disorders by central type.

Myelitis on the thoracic spinal cord levelis characterized by spastic paraparesis with conductive sensitivity lesion, pelvic disorders (retention of urine and feces passing into the central type of incontinence).

Myelitis on the lumbar spinal cord levelis characterized by peripheral lower paraparesis, anesthesia, the level of which begins from the groin. Pelvic disorders occur (retention of urine and feces passing into the central type of incontinence).

In more rare cases, the inflammatory process covers only the half of the spinal cord, which clinically is manifested as Brown-Sequard symptom.

In case of sudden transverse myelitis developing the muscle tone may be low for some time according to the foci location due to diaschisis phenomenon.

Bed sores develop quickly on the sacrum, trochanter and feet areas.

Subacute necrotizing myelitis is described. It is characterized by the lumbosacral spinal cord lesion, followed by pathological process spreading up with bulbar syndrome development and lethal outcome.

The course of myelitis is acute. The process reaches its greatest seventy in a few days, and then for a few weeks it is remained stable. The recovery period lasts from several months to 1-2 years. Often the paralysis or paresis of the extremities remain. Cervical myelitis is the most severe form because of tetraplegia, vital centers proximity, respiratory disorders.

Diagnostics.MRI of the spinal cord. In cerebrospinal fluid high protein content and pleycytosis are detected. Among the cells there may be neutrophils, and lymphocytes. While carring out liquorodynamic tests the block is absent. In blood there is leukycytosis and erythrocyte sedimentation rate is increased (leukocyte formula offsets to the left).

Treatment. Antiviral treatment needs to be tailored to the specific causative virus, when know. If Epstein-Barr virus, varicela-zoster virus, or HSV-1 or HSV-2 is suspected, acyclovir (10 mg/kg intravenous every 8 hours) should be administered. If cytomegalovirus is suspected, ganciclovir (5 mg/kg intravenously every 12 hours) or foscarnet (90-120 mgkg/day), or both, should be administered. There is no evidence supporting the use of glucocorticoids for vital myelitis; however, their use is indicated when the pathogenesis is unknown and immune-mediated processes are considered in the differential. Spasticity that typically ensues in the chronic phase can be alleviated with baclofen, benzodiazepines, and tizanidine.

POLIOMYELITIS

Poliomyelitis is an acute infectious disease characterized by general toxic symptoms and damage to the nervous system in type of peripheral paralysis.

Etiology.Virus Poliovirus hominis. According to antigenic properties, it is divided in to 3 types. They share a complement-fixing antigen. It is stable in the environment tolerant to cold, dies out by boiling and under the influence of ultraviolet irradiation.

Clinical features.The incubation period is 5-35 days.

Polio is divided into 2 large forms:

1. Without damage of the nervous system (inapparent, abortive, meningeal forms).
2. With damage of the nervous system (paralytic form).

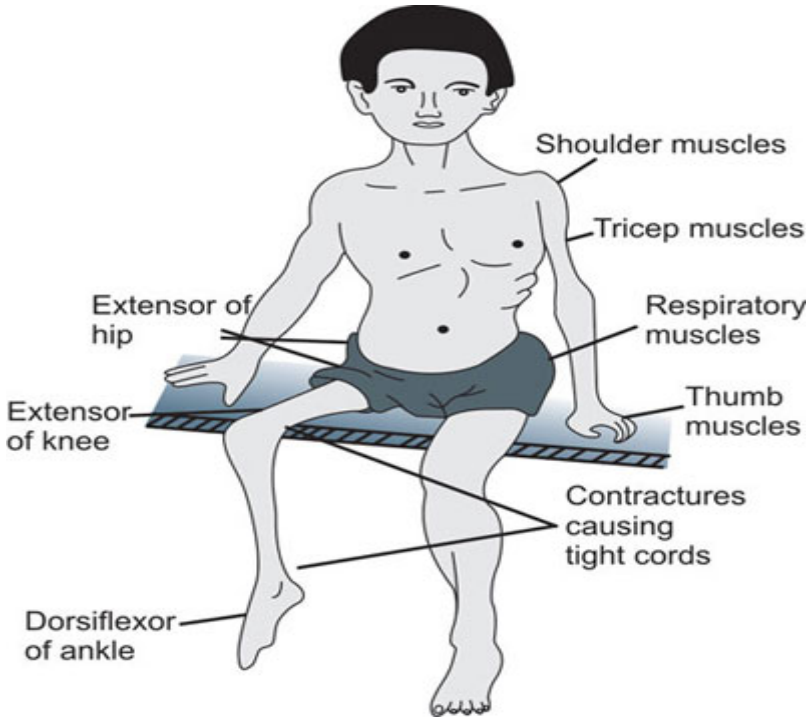
Poliomyelitis with damage of the nervous system is called typical, without such damage is it atypical one.

Paralytic poliomyelitis (typical)

Depending on the pathological process localtion – spinal, bulbar, pontine, mixed (bulbospinal). Spinal form is the most common.

Polio is distinguished by the periods.

Preparalytic period: acute onset, temperature is 39C, catarrhal phenomena, abdominal pain, pain while pressuring the spine, while flexing head and back. Forced position with head thrown back, positive meningeal symptoms are typical for patient. Lumbar puncture data: high pressure, other parameters are within normal data. The duration is 2-5 days.



Picture 28. Paralytic poliomyelitis (typical) *(https://www.jaypeedigital.com)*

Paralytic period. Duration is 5-6 days. Its forms are:

- Spinal form: a peripheral paralysis of hands and feet alongside with normal temperature. Areflexia, muscular atonia are common. Affected extremities are cold, pale, and cyanotic. Lumbar puncture data: a slight lymphocytic pleocytosis, elevated level of protein and sugar.
- Pontine form: the disturbance of the facial nerve nucleus with the paresis is facial muscles.
- Bulbar form: dysphagia, dysphonia, respiratory failure, paralysis of the diaphragm and respiratory muscles.
- Encephalitic form: loss of consciousness, seizures, speech disorders, meningeal symptoms, vestibular disorders.

The recovery period: the disappearance of intoxication symptoms and pain. Recovery of function is slow (long-term atonia, areflexia). The duration is 1-3 years.

Residual phenomena: flaccid paralysis, muscular atrophy, deformation and contractures, a shortening of the limbs.

Atypical poliomyelitis

Insidious form: due to virus carrier state within the pharyngeal ring and bowel it occurs without clinical manifestations. Diagnostics of this form is possible only based on virological test.

Abortive form: is characterized by malaise, appetite decrease, mild catarrhal symptoms, intestinal disorders. Neurological disorders are absent. The course is favourable.

Meningeal form: intoxication, severe headache, vomiting, twitching of muscles, horizontal nystagmus, meningeal symptoms are observed on the 2nd-3th days. CSF data: lymphocytic pleocytosis, a slight increase of protein and glucose. There are no paralysis. The course is favourable.

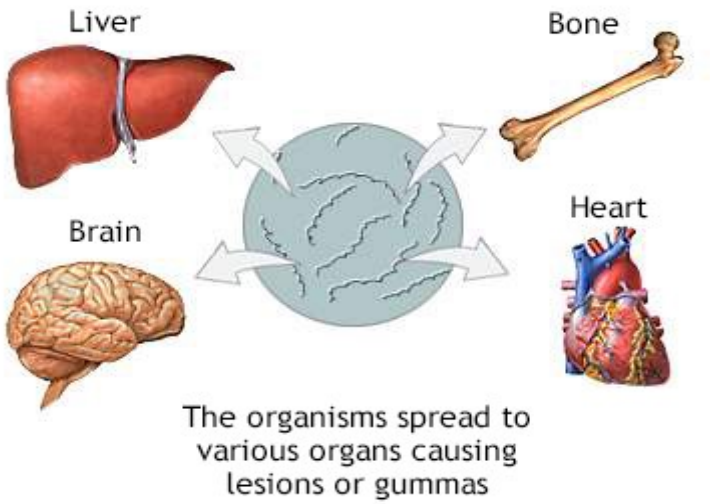
Laboratory diagnostics.Diagnostics is based on the poliovirus allocation from faces, cerebrospinal fluid, nasopharyngeal swabs and blood on the 3rd-7th day of illness. A week after the poliovirus infection the IgM and IgG appear in the serum of infected persons. The level of IgV is 2-8 times as high as the titers of IgG. After 2 weeks, the level of IgM reaches its peak, after 2 month they disappear. IgG titers gradually increase, antibodies may persist throughout human life. In some cases, serum IgA may appear in blood serum. Secretory intestine IgA-antibodies have a crucial role in protection against polio. Children may be resistant to poliovirus reinfection, even if they have no serum antibodies but have secretory antibodies in high titres.

Treatment.Hospitalization to infectious disease department. Therapy is supportive. Bed rest, fever and pain control (heat therapy is helpful), and careful attention to progression of weakness (particularly of respiratory muscles) are important. No intramuscular injections should be given during the drainage of the

bladder may be needed. Assisted ventilation and enteral feeding may also beneeded.

NEUROSYPHILIS

Neurosyphilis it is damage of a nervous system with Treponema palladium. It can occur at any stage of syphilis, and depends on the penetration of the exciter into the brain tissue.



Picture 29. The organism spread to various organs causing lesions or gummas *(http://www.histopathology-india.net)*

Classification.

I. The early neurosyphilis:

1.The latent (asymptomatic) syphilitic meningitis.

2. The acute generalized syphilitic meningitis.

3. The meningoneuritic form of the syphilitic meningitis (the basal meningitis).

4. The hydrocephalus.

5. The early meningovascular syphilis.

6. The syphilitic lesion of the optic nerves.

7. The syphilitic lesion of the vestibulocochlear nerves.

8. The syphilitic neuritis and polyneuritis.

9. The syphilitic meningomyelitis.

II. The late neurosyphilis.

1. The late diffuse meningovascular syphilis.

2. The syphilis of the cerebral vessels (the vascular syphilis).

3. The tabesdorsalis.

4. The progressive paralysis.

III. Brain gumma.

Clinical picture. In development of the latent, clinically asymptomatic syphilitic meningitis there are no cerebral symptoms, but it may be headache, dizziness, and general hyperesthesia. Such neurologic implications may be present: depression of the bone conduction due to affecting of tne cochlear nerve, weakening of reaction of the pupils to light, hypalgesia on the radicular type in the region of the segments T4-T6, T8-T10 (the hypesthesia in the area of Hitzing), and also depression of the vibratory sensitivity of the lower limbs. The meningeal symptoms are absent or poorly expressed. The diagnostics is based on changes of the cerebrospinal fluid: the high pressure of the liquor and increased protein level, pleocytosis (lymphocytic) to a hundred/thousands of cells. The Wassermann's reaction is positive in the blood and CSF, reaction of immune-fluorescence (RIF) gives positive results on the 2 week of the disease, and the reaction of immobilization of Treponema (RIT) appears only on the 10 week of illness. Identification of the latent forms is important action to prevent the development of late forms of the neurosyphilis.

Classification.

- Early neurosyphilis (meningovascular or mesodermal form) develops in the first 3-5 year after infection. Affects mainly vessels and meningesalongside with the secondary period of syphilis.
- Late neurosyphilis (parenchymal or ectodermal form) develops within 7-25 years after infection. Characterized by inflammatory and dystrophic lesions of the brain parenchyma; i.e. nerve cells and fibers, glia.

Form of neurosyphilis.

Early (mesodermal) neurosyphils.

Asymptomatic meningitis. Mid headache, dizziness, nausea, general hyperesthesia, pain while moving the eyeball, passing dysfunction of the cranial nerves. Malaise, insomnia, irritability, depression are observed sometimes. The inflammatory changes in the cerebrospinal fluid are present (slightly expressed lymphocytic pleocytosis) on which the diagnosis is set. In blood and cerebrospinal fluid the positive serological tests for syphilis (Wassermann test, the TPIT – the treponemapallidumimmobolization test) are marked.

Syphilitic meningitis (meningovascular or mesenchymal syphilis) involves inflammation of the sheaths and small blood vessels in the brain. It is also called the basilar form, because the process mainly covers base of the brain and blood vessels. In these cases the process is gradual, continues during weeks and even month, and in the clinical picture the meningeal syndrome is slight and does not come to the fore.

The damage of cranial nerves is quite typical. The damage of:

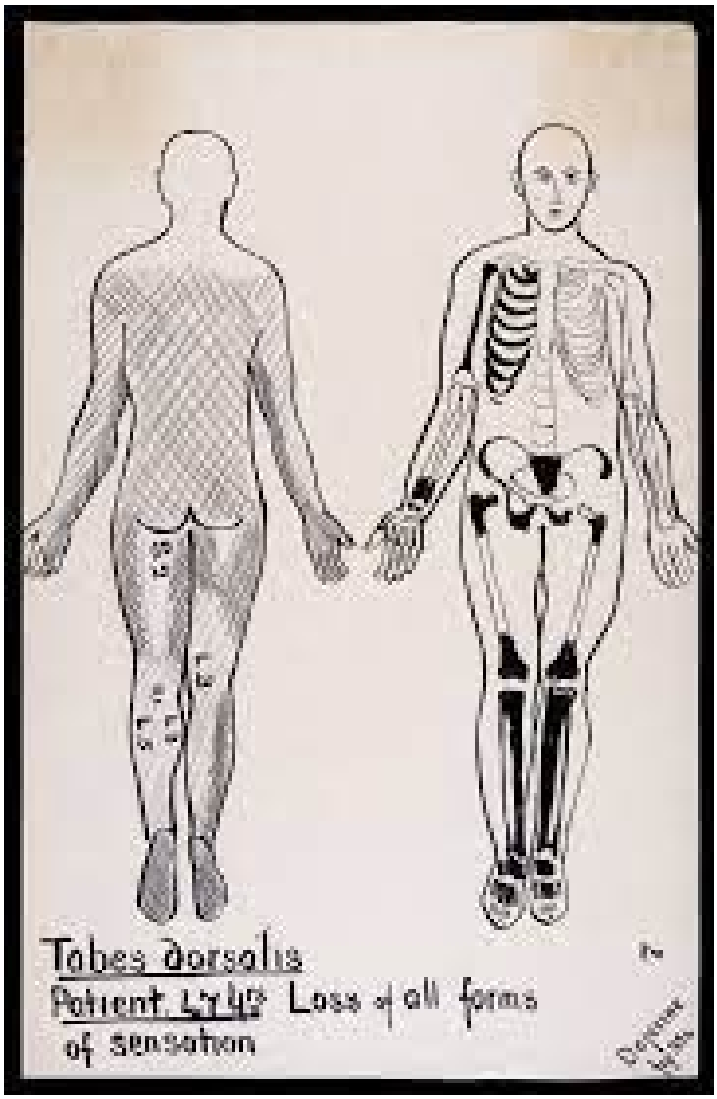
- oculomotoroaabducens nerves lead to diplopia
- facial nerve – to paresis of mimic muscles
- cochlear nerve – to hearing loss, dizziness
- optic nerve – to reduceof visual acuity, optic atrophy.

It can result in a stroke because of the defeats of small blood vessels.

A direct Argyle-Robertson symptom occurs: loss of reaction of pupils to light with preservation of their reaction to accommodation. The symptom is not specific enough (can be observed in multiple sclerosis, chronic alcoholism and diabetes), as a consequence of optic atrophy and uvelis, but the small size of the pupils and deformation of contours alongside with other clinical and paraclinical data may be evidence in favor of syphilis.

Late (parencimatous)neurosyphis.

Tabesdorsalis. The disease develops within 5-10 years after the initial infection. At the pathogenesis, probably the autoimmune processes play a certain role. However, the predominant mechanism remains the neurotoxic effect of spirochetes that persist chronically (continue to live in membranes for year, affecting the nervous elements by its metabolic products). This effect is a unique characteristic for neurosephilis, unikle other infections (except perhaps prions). The increased degeneration of posterior roots of the spinal cord, sensory and autonomic ganglia, and later the ascending sensory fibers, mainly the Gaulle and Burdach pathways are found on autopsy.



Picture 30. Tobes dorsalis *(https://utmb-ir.tdl.org)*

The first stage in the neuralgic. It is characterized by sudden attacks of pain in the appropriate dermatomes (from several seconds to minutes, hours or even several day) and splanchnotomes: heart, liver, stomach, bladder or rectum, etc, though pain may have a shingle character (as in case of intercostals neuralgia). The pain is strong, unbearable; “stabbing” and can somulatr an attack of angina pectoris or myocardial infarction, perforated gastric ulcer, biliare colic and other diseases. Although there are not appropriate changes of blood or ECG, such patients find themselves on the operating table. At the same time lower extremities may be revealed. Disorders of urination (retention, incontinence), and sexuao function, constipation, are joined.

The pathology of cranial innervations in from of primary optic atrophy, direct Argyle-Robertson symptom, pupils deformation (sometimes a quick change of their diameter, “jumping pupils”), oculomotor dysfunction, face asymmetry and hearing impairment are detected. Loss of achilles and knee reflexes are joined. The areas of hypoesthesia by segmental type on the trunk or extremities are revealed sometimes.

The second stage is ataxia: the process extends to the dorsum column of the spinal cord. Sensitive ataxia joints the above-mentioned complaints. This ataxia means istability and loss of coordination during walking, which significantly increase in the dark with closed eyes. The patients notice the loss of sense of surface (feel like walking on the “rubber carpet”), they walk placing the feet widely and leaning mostly on the heel. Muscular hypotonia, especially in the legs, results in genu recuvatum.

Trophic disorders develop. They are painless arthropathy with deformation of the joints (ankle, elbow and other joints) with their swelling and increase in size. Fractures occur as a result of local osteoporosis. Trophic disorders may be manifested in the form of a painless ulcer on the foot (malumperforanspedis), tooth loss, disturbance of nail growth and skin trophic.

The third stage of the disease is paralytic stage. Paresis and paralysis are absent, but owing to ataxia, patients lose the ability to walk, stand and sit.

Progressive paralysis or general paralysis of insane is manifested by progressive mental isorders until deep dementia, accompanied by somatic and neurological disorders. In the intial stage symptoms of neurasthenia dominate. They are fatigue, irritability, reduction of working capacity. Later personality changes are joined. Memory disorders, the lements of intellectual disabilities, the fomer skills and knowledge are lost. On the base of progressive dementia absurd delusions of grandeur, good or bad mood, hypochondria, changing states of depression and manic excitement may occur. Disorders of speech, movement, writing, epileptic seizures can develop. Characteristic neurological syndromsare:deformation of pupils, direct Argyle-Robertson symptom, paresis of eye muscles, hyperreflexia, hand tremor.

Unspecified neurosyphilis – Gumma (syphilitic) of central nervous system (granulomas). Gumma (from gumma-glue) may be quite small, looking like grayish nodules resembling military tubercles, but more common ara solitary gumma with size up to 30 mm in diameter. The clinical course is the same as a tumor of the brain (general cerebral syndrome and focal signs depending on the location). As they are formed mainly in pia mater of the brain base, focal manifestations include cranial nerve roots lesions. The clinical picture of gumma in spinal cord is the same as extramedullary tumors (lesions of the spine root, Brown-Sequard syndrome) with probable compression of the spinal cord.

Diagnosis

Diagnosis of neurosyphilis is set on the base of 3 main criteria:

- clinical picture
- changes in the CSF and positive test result for syphilis
- the evaluation of neurosyphilis of neurosyphilis clinical features is possible only after a neurologist’s and an ophthalmologist’s examination (an overview of the pupils, ocular fundus).

Laboratory tests for syphilis are applied comprehensively and repeatedly if it is necessary. RPR-test (Rapid Plasma Reagin – detects IgG and IgM antibodies to lipid and lipoprotein materials released from damaged cells of patients with syphilis). IFR (immunofluorescence reaction), TPIT (Treponemapallidum immobilization test).examination of cerebrospinal fluid reveals Treponemapallidum, elevated protein, lymphocytic pleocytosis. Tne result of the IFR is positive.

MRI and CT scan of the brain or spinal cord in case of neurosyphilis find mostly non-specific pathological changes in from of thickening of the meninges, hydrocephalus, atrophy of the brain matter and the localization of syphilitic gumma that allows setting a differentiated diagnosis of neurosyphilis and other similar in clinic disease.

Differential diagnosis. The differential diagnostics of early forms of the neurosyphilis needs to be carried out with meningitis and meningomyelites of other etiology, vascular lesions of the brain (the TIA, stroke) and spinal cord (the spinal stroke), or due to other causes. The locomotor ataxia should be distinguished from the Friedreich's disease, which is characterized by the mixed sensitivity and cerebral ataxia, signs of dysraphia, the early beginning (age of 20 years) and existence of a hereditary factor; from the funicular myelosis, when the posterior and lateral funiculi of the spinal cord are affected, the malignant anemia, deficiency of vitamin B12 become perceptible; from the Adie's syndrome, when there is weak reaction of a pupil to light (nevertheless the pupil is enlarged in dark; this syndrome is unilateral; the wide pupil is characteristic), depression of the Achilles and knee reflexes caused by the myelodysplasia. The amyotrophic spinal syphilis is resemble to the spinal muscular atrophy of adults and the ALS. The differential diagnosis of the Erb's spastic paralysis is carried out with the Strimpell's paraplegia. The gummae of the brain and spinal cord they must differentiate with the tumors and other mass processes of this localization.

Treatment of neurosyphilis.Basic treatment of all forms of neurosyphilis includes penicillin G prescribed by a dermatovenerologist. The result is assessed on the base of normal level of cells in the cerebrospinal fluid (increased protein can remain longer, for several months). Serological reactions in cerebrospinal fluid may remain positive for years or during all lives, despite the positive clinical effect. Symptomatic treatment: complex B vitamins, ascorbic acid, physiotherapy treatments (massage, balneotherapy).

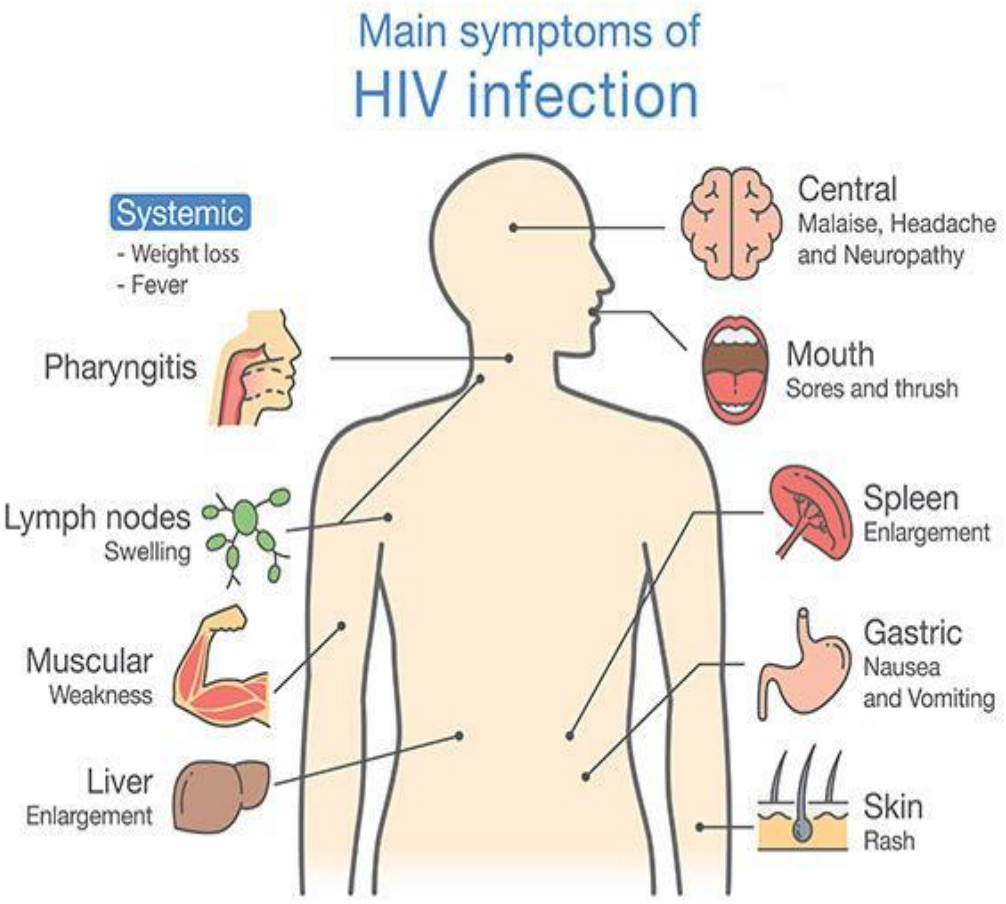
NERVOUS SYSTEM LESION IN HIV-POSITIVE PANIENTS

Neuro-AIDS

Etiology.Nervous system lesions occur of 90% patients, although clinical neurological complications are detected only in 50-70% of cases. The causative agent of the disease is human immunodeficiency virus (HIV), which belong to the retroviruses family. Certain body fluids can transmit the virus (blood, scmen, pre-seminal fluid, rectal fluid, vaginal fluid, breast milk).

Classification.

- Early neuro-AIDS
- Late neuro-AIDS

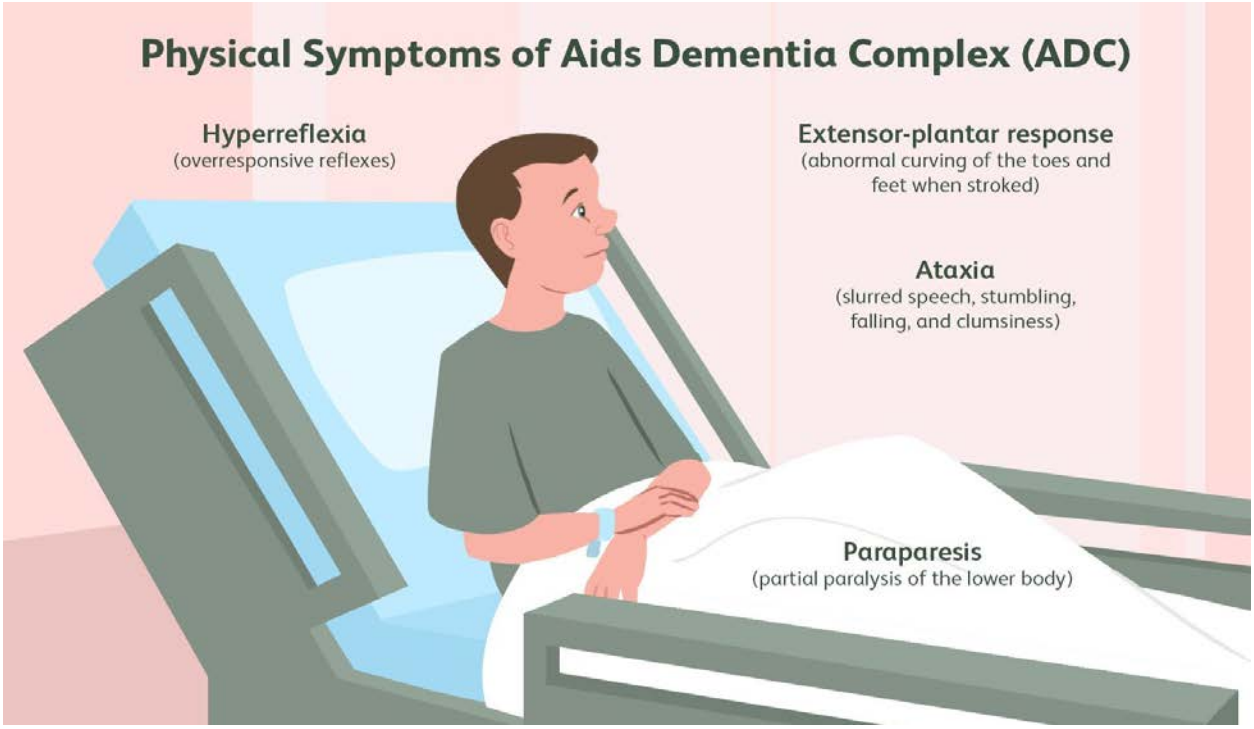


Picture 31. Main symptoms of HIV infection *(https://www.alamy.com)*

The main nosological forms of early neuro-AIDS

AIDS dementia (HIV encephalopathy).

Morphological substrate of HIV encephalopathy is a primary cerebral hemispheres white matter lesion of the inflammatory and demyelinating nature, mainly in subcortical structures, which is caused by multifocal giant cells encephalitis and progressive diffuse leukoencephalopathy.



Picture 32. Physical symptoms of AIDS dementia (*https://www.verywellhealth.com*)

Characteristic features.

Cognitive-mnestic disorders: reduced memory, impaired concentration, slowing and reduction of intellectual activity occur at an early stage of the disease. In the neurological status hyperreflexia, oral automatism symptoms, tremor and mild ataxia, hypomimiyaare observed.

Changes in behavior: gradually arise cognitive function violations, drowsiness, untidiness and apathy.

Movements disorders: stiffness, central paresis, ataxia, hyperkinesis, seizures, pelvic organ dysfunctions.

Akineticmutism (loss of contact with reality, loss of interest in reality, the lack of desire to communicate with other, poor emotional expressions), abulia, lower spastic paraplegia. Incontinence of urine and feces, generalized seizures, psychotic disorders occur at the last stage of the disease.

Diagnostic criteria according to CT scan or MRI:

- Cortex atrophy with subarachnoid space and brain ventricles extencion.
- Subcortical foci in the frontal and parietal lobes.

HIV-associated meningitis.

The meningitis diagnosis is based on the presence of three common syndromes: general infectious, meningeal syndrome inflammatory changes in the cerebrospinal fluid. However, atypical variants of HIV-associated meningitis are observed in most cases. Seizures, mental disorders, consciousness disorders are possible in the case of severe meningoencephalitis.

Diagnostic criteria for HIV-associated meningitis are slight, but persistent lymphocytic pleocytosis, HIV infection and antibodies to it in CSF, while their absence in blood is possible.

Vascular neuro-AIDS

In case of neuro-AIDS virus-induced brain and spinal cord vasculitis development is possible. Therefore, 20% of patients may experience a stroke. HIV-infected patients have vascular wall infiltration with leukocytes, edema and proliferative changes of the internal layer. This leads to vascular obctryctions, thrombosis with subsequent development of cerebral infraction, blood vessels rupture with hemorrhage occurrence.

HIV-associated vacuolar myelopathy

It may be isolated or combined with AIDS dementia. Spinal cord dymalization and spongy degeneration are morphologically determined mainly in the lateral and posterior columns in the middle and lower thoracic segments level. Vacuolar myelopathy is characterized by slowly progressive spastic paraparesis with high tendon reflexes, pathological plantar signs, sensitive ataxia, conductive type sensitivity disorders with the upper limit of the body skin corresponding to the affected segment. Central type pelvic organ disorders are also characteristic. MRI detects atrophy and stretch enhanced signal T2-weighted mode at the thoracic level of the spinal cord involving the cervical level or not.

Inflammatory polyneuropathy

It may occur at any stage of HIV infection. In case of the distal symmetric polyneurophathy patients are concerned with numbness, buring, paresthesia in the lower limbs that strengthen by the slightest touch. Pain increase at night and its reduction when putting the lower limbs in cold water is typical. In neurological status “gloves” and “socks” hypoesthesia types are detected, vibration sensitivity is decreased and reduction of Achill es reflexes occurs.

Late neuro-AIDS

Progressive multifocal encephalopathy with subcortical hyperkinesis and progressive dementia is a demyelinating disease of the nervous system, which is caused mainly by papovavirus JC. Its replication occurs as a result of immunosuppression. Clinical manifestations are headache, dementia, aphasia, hyperkinesis, progressive paresis, sensitivity disturbances and epileptic seizures. The clinical course is rapidly progressive, death occurs within 6-9 months.

Toxoplasmosis encephalitis is the most common cause of tumor processes in case of AIDS. Disease onset may be acute or subacute in the from of focal or generalized seizures. Attention is drawn to focal neurological symptoms: aphasia, cranial neuropathy, hemiparesis, sensory and coordinate disorders on cerebral symptoms background – headaches, disorientation and confusion. Optical lesions (uveitis, focal necrotic chorioretinitis, papillitis) are prior or simultaneous to the

central nervous system disorders that occur without oblious inflammatory reaction. Diagnosis of toxoplasmosis encephalitis is based on CT scan or MRI data and detection of Toxoplasma DNA in blood and cerebrospinal fluid by polymerase chain reaction (PCR).

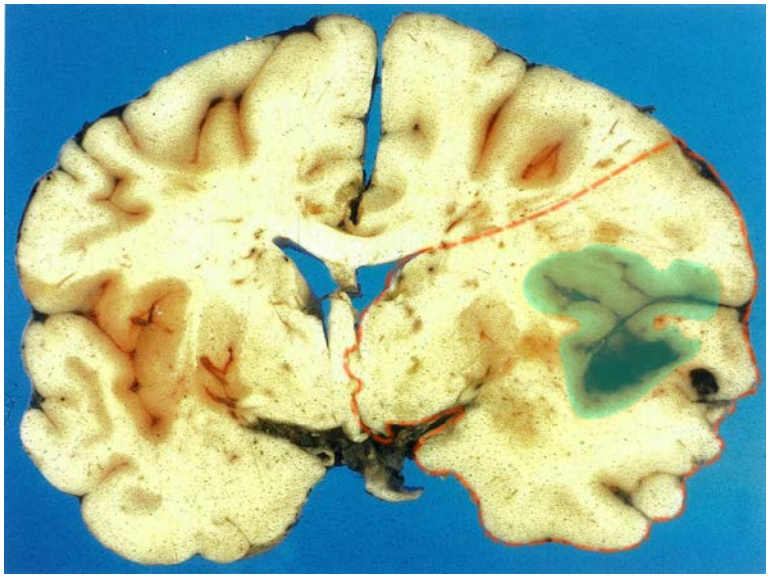
Cryptococcus meningitis usually occurs on the base of severe immunodeficiency due to the fungus Cryptococcus neoformans, which is acquired via the respiratory tract. The disease begins acutely or subacutely with headache, fever, nausea, photophobia, confusion, weight loss, temperature up to 37.5-38.0^0C. Local granulomas development is possible in brain tissue in the form of accumulated. Cryptococcus that clinically resembles a tumor. The CSF examination show a slight pressure, increase mild lymphocytic pleocytosis, a moderate increase in protein content, decreased glucose levels.

Picture 33. Cryptococcus meningitis*(https://www.slideshare.net)*

Herpetic encephalitis. In clinical course the following stages are distinguished:

- Early stage
- Clinical manifestations
- Stabilization stage
- Symptoms regression stage

The disease may begin with meningeal type at the early stages: general infection symptoms, expressed general cerebral symptoms with consciousness disorders are observed. Sometimes the disease begin with cortical (delirious) type: inappropriate behavior, disorientation in time and place, amnesia. A stroke-like start is possible: sudden generalized seizures, loss of consciousness with seizures and such changes of behavior as disorientation in place and time, hallucinations, agitation will be admitted to a psychiatric department. Rarely brain stem type lesions occur, characterized by diplopia, dysarthria, ataxia, alternating syndromes. At late stage of the disease, there may be general syndrome and liquorice-induced symptoms, pyramidal and extrapyramidal system syndromes, seizures, severe cognitive violation. Diagnosis is based on a CSF study, in which moderate lymphocytic pleocytosis, high IgG levels and positive PCR to herpes simplex viruses are detected.



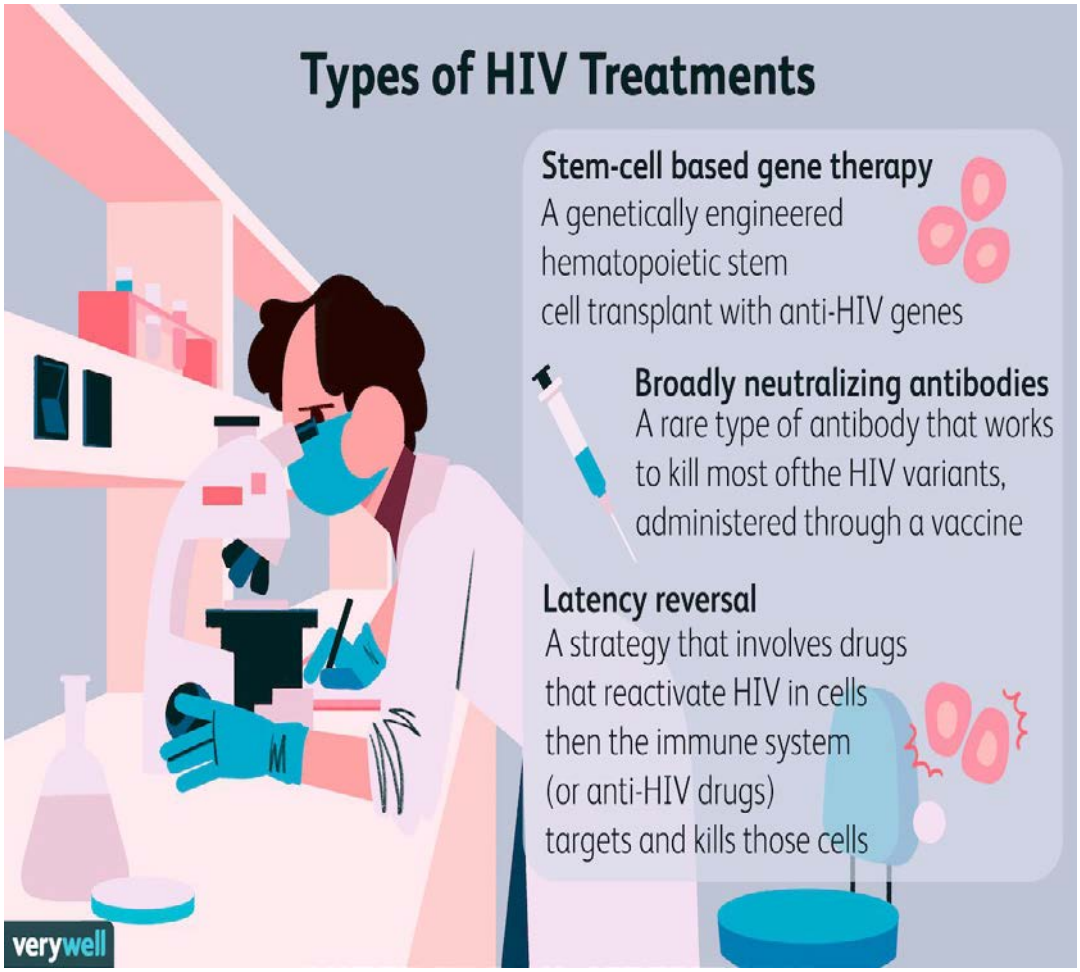
Picture 34. Herpetic encephalitis*(https://pubs.rsna.org)*

Vasculitis and cerebral circulation lesion. Most frequently necrotic vasculitis in localized in the medial parts of the temporal lobe. Aggression, negativism and seizures are characteristic. The development of parenchymatous and subarachnoid hemorrhage is possible due to mycotic aneurysm rupture (sustainable local expansion of blood vessels), bleeding is possible in the brain tumor, Kaposi sarcoma. Along with of signs vascular pathology vascular change in the skin, myocarditis and optic lesions are observed.

Neoplasms of the CNS: primary lymphoma is the most common. It is found in 2% of cases, usually as a result of profound immunosuppression. The initial manifestations reflect single or multiple lesions of the brain parenchyma and intracranial hypertension. The variety of clinical symptoms is determined by the process location. Confusion, stupor, decreased memory. Behavior disorders, cranial nerve lesions, hemiparesis, aphasia and seizures are typical.

Picture 35. Neoplasms of the CNS*(https://pubs.rsna.org)*

Treatment of the neuro-AIDS



Picture 35. Types of HIV treatments *(https://www.verywellhealth.com)*

Neuro-AIDS treatment should be comprehensive and pathogenic. In case of primary neuro-AIDS specific highly active antiretroviral therapy prescribed which slows down the disease progression and temporarily stabilizes the patients condition.

There are two groups of drugs:

- HIV reverse-transcriptase inhibitors (zidovudine, abacavir, stavudineetc);
- different viral enzyme protease inhibitors (indinavir, ritonavir).

The positive clinical effect is observed in patients with neuro-AIDS when the recombinant interleukin-2 is added to the treatment. It significantly improves the celluar immunity indices.

The treatment of the patients with secondary neuro-AIDS is based on the application of specific treatment of nervous system lesions caused by oppottunistic infections.

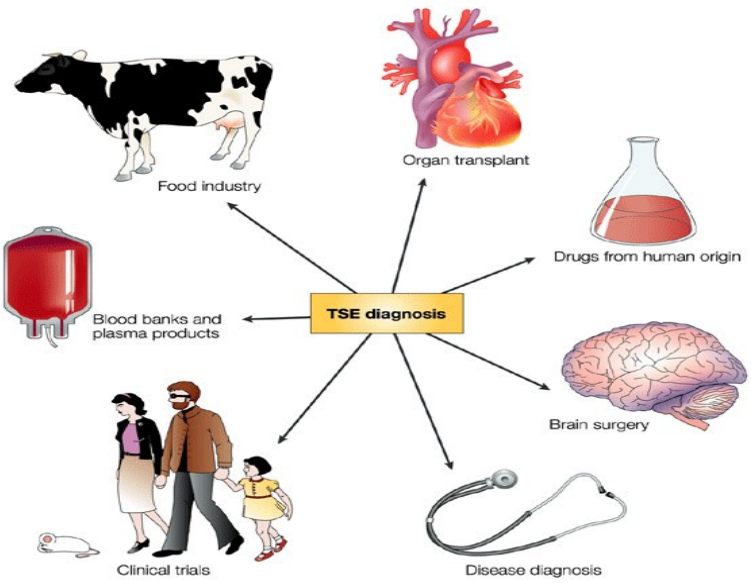
If cytomegalovirus in the cause of the nervous system lesion, a combination of ganciclovir and foscarnet is used; if toxoplasmosis in the cause, induction treatment of pyrimethamine, sulfadiazine, leucovorin are prescribed. For patients with cruptococcal meningitis, induction treatment with amphotericin B and flucytosineare given followed by fluconazole.

PRION DISEASE

The prion disease belong to the group of neurodegenerative disease of the people and animals caused by infectious proteins (prion). The diseases are connected with the disturbance of metabolism and accumulation of the prion proteins in the CNS cells. There is the prion protein in a human body in norm: it is coded by the gene located on the chromosome 20. The concentration of protein in the brain neurons is especially high. In the pathology its modified form resistant to action of proteases collects.

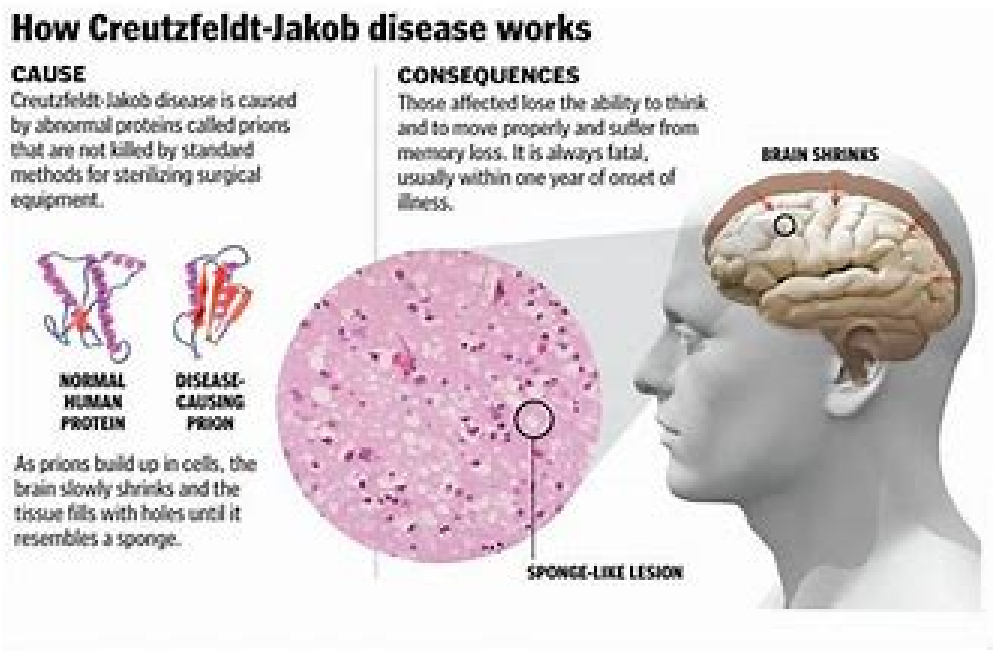
Today four human diseases caused by prion are studied: the disease of Creutzfeldt-Jakob, Kuru, syndrome of Gerstmann-Straussler-Sheinker, and fatal insomnia.

They are subdivided into sporadic (the disease of Creutzfeldt-Jakob, fatal insomnia), acquired or infectious (Kuru, iatrogenic disease of Creutzfeldt-Jakob, new option of disease ofCreutzfeldt-Jakob), and hereditary (disease Creutzfeldt-Jakob, syndrome of Gerstmann-Straussler-Sheinker, and fatal insomnia). On the result of numerous researches a part of the family prion disease is about 10%.



Picture 36. Prion disease *(https://www.nature.com)*

Clinical form prion disease. The frequency of the disease Creutzfeldt-Jakob in different regions is identical and is 0,3-1 case on a million of people annually. Ratio between men and women is 1,5:1. Some clusters who have the disease more often are taped, for example, in the Libyan Jews living in Israel. The peak of a case rate o this disease is about 60-65 years; average life expectancy of the sick people is about 8 months, 90% of patient die within the first year.



Picture 37. Creutzfeldt-Jakob disease*(https://microbe-canvas.com)*

The main clinical syndrome of the diseaseCreutzfeldt-Jakob is the fast-progressing

multifocal dementia with myoclonus. The distinguish five stages of development of disease ofCreutzfeldt-Jakob:

1. The prodromal stage.
2. The stage of the first symptoms.
3. The development stage.
4. The final stage.

5. The stage of the prolonged life in the conditions of the intensive care unit.

The vegetative disorders (the asthenia, sleep and appetite disorders, decrease of a body weight, and loss of libido), disturbance of memory, attention, thinking, disorientation, hallucination, apathy, and paranoia belong to the prodromal symptoms.

The visual disorders, as a rule, belong to the first implication off a disease in the form of the diplopia, illegibility of vision, disturbance of the visual fields, and visual agnosia.

In the deployment stage the key syndrome are the subacutesclerosing dementia, myoclonias, typical periodic complexes on the EEG and normal composition of the cerebrospinal fluid. The patient develops the disturbance of memory and spatial orientation, depression, and emotional lability. Often there may be the cerebellar disorders, such as the ataxy of extremities, tremor, and dysarthia. In the patients they observe the disturbance of the visual fields, colour perception, visual hallucinations, and supranuclear motor disorders. The myocloniasare noticed in 75% of patients, and the extrapyramidal and pyramidal disorders – more than in a half of patients.

In the final stage of the disease the dementia, akineticmutism, disturbance of consciousness, spastic paralyses and hyperkinesias, decebration rigidity, widespread myoclonias, trophic disorders, accompanying somatic pathology, and disturbances of respiration on the central type, which lead the patient to death, dominate.

The resuscitation is ineffective in the case of such disease. In the stage of the prolonged life in the clinical picture the apalic syndrome, vegetative status, hyperkinesias, joint contractures, myoclonias, and loss of muscle mass is observed. The heat failure is the cause of death.

There are three forms of the course of the disease:

- the slow progressing mental and behavioral disturbance in a debut with the subsequent their increase;

- the persistent or step progressing without the stabilization periods;
- the fast deterioral within several month with the subsequent rather slow progressing in the terminal stage.

At the early stages there may be mood and psychological disorders (the anxiety, depression, and change of behavior), rarely – the pain and dysesthesia in the extremities and on the face. In some weeks or month the progressing cerebellar disorders join, and in late stages – the disturbance of memory and dementia, to a lesser extent – the myoclonias and chorea, pyramidal disorders, and akineticmutism. On the EEG there are no changes, characteristic for the Creutzfeldt-Jakob's disease. On the CT and MRI they find out the atrophy of the brain of different degrees. In the biopsy of the pharyngeal tonsils they determine PrP^{Sc}.

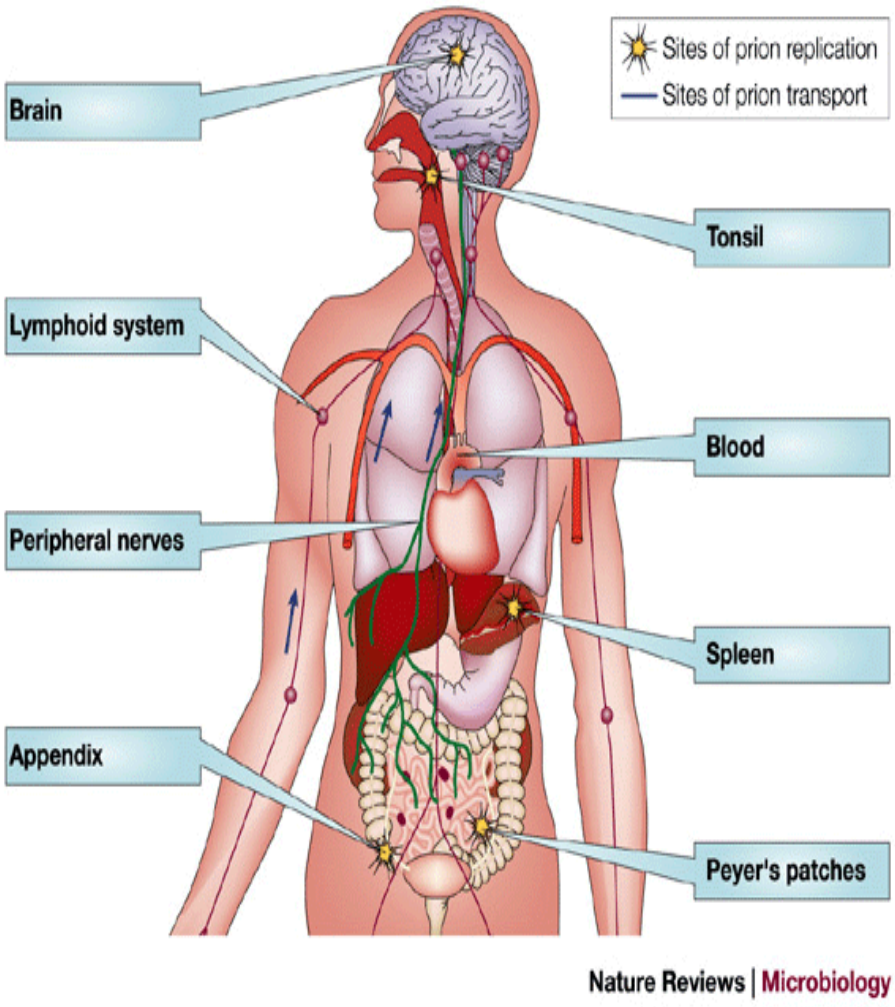
The sporadic fatal insomnia clinically and pathoanathomically is indential to the family fatal insomnia. Today about 100 observations on the family fatal insomnia were described.

Diagnostics.The diagnostics of the prion disease remains complicated. The diagnosis is made mainly on the basis of clinical data and result of the EEG. The morphological diagnostics of these diseases is carried out by result of research of the biopsy or autopsy material. Al the early stages of illness the retardation of bioelectric activity is observed. With the progression of the pathological process there are periodic two-phase or three-phase sharp waves with a frequency about 2Hz. At the same time these changes aren't specific and can accompany varios metabolic encephalopathies; nevertheless in other neurodegenerative diseases they aren't described. In the disease Creutzfeldt-Jakob's of some hereditary forms, as well as in its new option, the EEG-changes are absent. In the syndrome of Gerstmann-Straussler-Sheinker and the family fatal insomnia the retardation of the background activity is observed, but periodic sharp waves aren't determined.

In recent years in the CSF of patients with the disease of Creutzfeldt-Jakob's unusual proteins were identified, and one of them (14-3-3) is of great importance

in diagnostics of all PD of humans and animals. While studies it was shown that in human and animals (sick with the disease Creutzfeldt-Jakob's) in the blood high titers of autoantibodies to neurofilaments were determined.

The CT and MRI often allow detecting the sings of the progressing brain atrophy. On the MRI hyperdensive signals in a projecting of the basal ganglia and thalamus can be registered. Introduction of a contrast agent, for example gadolinium, strengthens this signal, which can precede the MRI-changes, are registered. During the magnetic resonance spectroscopy they find the depression of N-acetylaspartate signal with the progression of Creutzfeldt-Jakob's disease while reducing the number of neurons.



Picture 38. Diagnostics (*https://microbe-canvas.com)*

Different diagnostics. In the different diagnostics of the Creutzfeldt-Jakob's disease they consider neurologic implications on the systemic vasculitis, MELAS syndrome, hypoxemic encephalopathy, subacutesclerosingpanencephalitis, herpetic encephalitis, dismetabolicencephalpathies, and initial implications on the AIDS-dementia. In the initial stages the Creutzfeldt-Jakob's disease may resemble the Parkinson's disease, progressing suprenuclear paralysis, vascular encephalopathy, pfraneoplastic syndrome, ant lateral amyotrophic sclerosis. The clinical and electro-physiological implications of this disease can imitate the encephalopathy caused by the toxic action of preparations of lithium.

Treatment.There is no the effective etiological or pathogenetic treatment of the PD today. At the early stages hey use the symptomatic treatment, which corrects the behavioural disorders, disorders of sleep and myoclonia: amphetamines, barbiturates. Antidepressants, benzodiazepines, and neuroleptic. At the late stages they carry out the maintenance therapy.

NEUROBORRELIOSIS

Lyme disease (neuroborreliosis)

The causative agent in Lyme disease is the spirochaeteBorreliaburgdorferi, which is transmitted by the tick Ixodesdammini. The organism is prevalent throughout Europe and North America (e.g. Lyme, Connecticut, where the disease was first recognized).

Classification.

I. Form: latent, manifesting.

II. Stage: localized (a stage of the primary effect), disseminated (generalized), persistent (chronic), and residual.

III. Variant: the predominant lesion of the skin, nervous system, joints, heart, and mixed.

IV. Course: acute, subacute, and chronic (continuous, relapsing).

V. Course severity: mild, moderate, and severe.

VI. On the basis of infection: seropositive, seronegative.

VII. On existence of complications.

Clinical presentation. The clinical course of Lyme disease can be divided into three stages. Stage 1 begins 3-30 days after the tick bite and consists of a relapsing-remitting pyrexia and arthralgia, with a typical skin lesion (erythema chronicummigrans) developing at the site of the bite. This stage resolves after about 4 weeks.

Stage 2 occurs a few weeks or months after stage 1 and. consists of neurological (15 %) or cardiac symptoms (10 %), which can last up to 8 weeks. Neurologicalmanifestationsinclude:

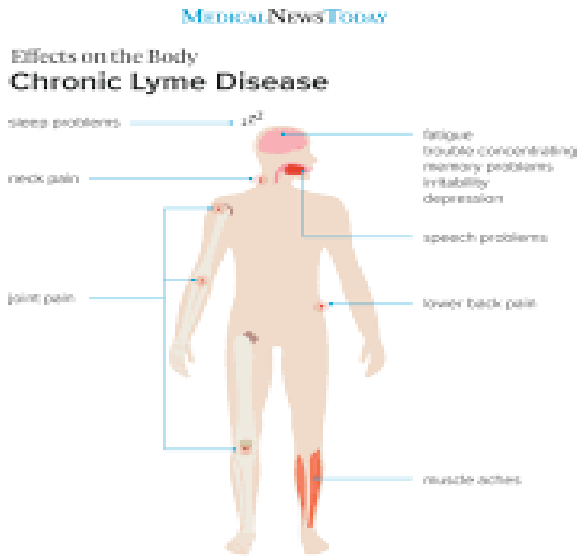
- Subacute lymphocytic meningitis: often mild and self-limiting but can recur if not treated.
- Subacute encephalitis: often mild and self-limiting.
- Cranialnerveinvolvement.

– Peripheral neuropathy with painful radiculitis.

A patient that is presented with a unilateral or bilateral peripheral palsy of VII cranial nerve, a rash and a systemic upset, e.g. fever should always be becon-sidered to have possible Lyme disease.

Stage 3 occurs several months or years later and consists of recurrent and often eiosive arthritis. Signs of diffuse CNS involvement may also develop, with focal encephalitis, seizures, behavioural disorder and a multiple-sclerosis-like illness.

Picture39. Lyme disease*(https://www.healthline.com)*



Picture 40. Chronic Lyme disease *(https://www.healthline.com)*

Diagnostics. The neuroborreliosisshould be suspected in patients with the serous meningitis, cranial neuropathy, isolated or multiple radiculitis, plexitis, meningocephalitis, and encephalomyelitis. In such patients it needs to clear up, whether the appearalitis of cutaneous manifestation (erythema migrans, lymphocytes, acrodermatitis) with a tick bite, especially in endemic regions and in the appropriate season (spring-summer).

Using the results of the clinicoserologic researches,it is possible to make the probable diagnosis of the neuroborreliosis in the following cases:

1. The patient develops (as a rule, after the tick bite) typical tick-borne erythema migrants in the form of a ring of a least 5 cm with the enlightenment in the center.

2. In the absence of the typical tick-borne erythema migrants the accurate diagnosis of the Lyme's disease is made in the case of development of one or several typical syndromes of illness based on the laboratory confirmation of the borrelious infection:

- the lesion of the nervous systm in the formed serous meningitis andencephalitis (with exception in the sick person the diagnosis of the tick-born virus encephalitis), meningoradiculitis, polyradiculoneuritis, and neuritis of the cranial nerves;
- the damage of joint on the type of the mono- or oligoartgritis of relapsing or chronic nature;
- the heart damage: the myocarditis (pericarditis) with disturbance of conduction on the type of the atrioventricularblocade of the II or III degrees;
- the single lymphocytoma of the ear lobule or nipple of the breast;
- the chronic atrophic acrodermatitis.

3. The clinical diagnosis has to be confirmed by definition of the specific antibodies to Borreliaburgdorferi (IgG, IgM) in the cerebrospinal fluid. The presence of antibodies in the blood serum, irrespective of a titer, doesn't allow diagnosing the borreliosis, because their certain level is constantly observed in the

population of the endemic zones and doesn’t show the activity of process. The Lyme’s disease is possible to diagnose only at the presence of all three criteria.

Treatment. Treatment comprises oral antibiotics (penicillin or tetracycline) at beginning of desease with following high-dose intravenous penicillin or cef-triacsone for 14 days in stage 2 and 3. This shortens the course of neurological illness and preventsfurther parenchymal damage.

NEUROLOGICAL MANIFESTATIONS OF COVID-19

General facts:

– The coronavirus is an enveloped positive-sense single-stranded RNA virus belonging to the coronoviradae family.

– An outbreak in the city of Wuhan in December of 2019 saw the introduction of a new coronovirus strain, severeacute respiratory syndrome coronovirus 2 (SARS-CoV-2), named coronovirus disease 2019 (COVID-19) by the Word Health Organization (WHO) in February 2019.

– Under electron microscope , the virus appears crown-like due to the small bulbar viral sprike peplomers on the surface envelope.

– SARS-COV has shown to have a zoonotic origin with bats being the primary reservoir adapted by humans.

– It has shown to spread via respiratory droblets, fomites, and person-to-person contact.

The mechanism by which SARS-Cov-2 enters the CNS could be enunciated as follows:

- Direct infection injury.
- Blood circulation pathway.
- Neuronal pathway.
- Immune mediated injury.
- Hypoxic injury.

Haematogenous Spread of SARS-CoV-2 to target CNS:

- It has been proposed that SARS-CoV-2 gains entry to the CNS by systemic vascular dissemination and, the virus invades neural tissue due to its properties of neurotropism.

- SARS-CoV-2 gains entry to cells via ACE 2, which it binds via spike proteins.

ACE 2 receptors are expressed:

- On glial tissues.

- Neurons.
- Alongside the capillary endothelium of the central nervous system, which make them a target for the attack by SARS-CoV-2.

Key neurological manifestations include:

- Headache.
- Dysfunction of olfaction and gustatory sensation:

anosmia – loss of the sense of smell,

hyposmia

- Dizziness.
- Neuropsychiatric complications:

altered consciousness

encephalopathy

- Cerebrovascular accidents:

hypercoagulable statesin patients with COVID-19 have been reported at alarming frequencies, with many resulting in the ocurrence of stroke, even in those without any cerebrovascular risk factors; this hypercoagulability is thought to be a result of the activation of inflammatory and thrombotic pathways from SARS-CoV-2 interaction with ACE-2 (Angiotensin-converting enzyme 2) on endothelial cells.

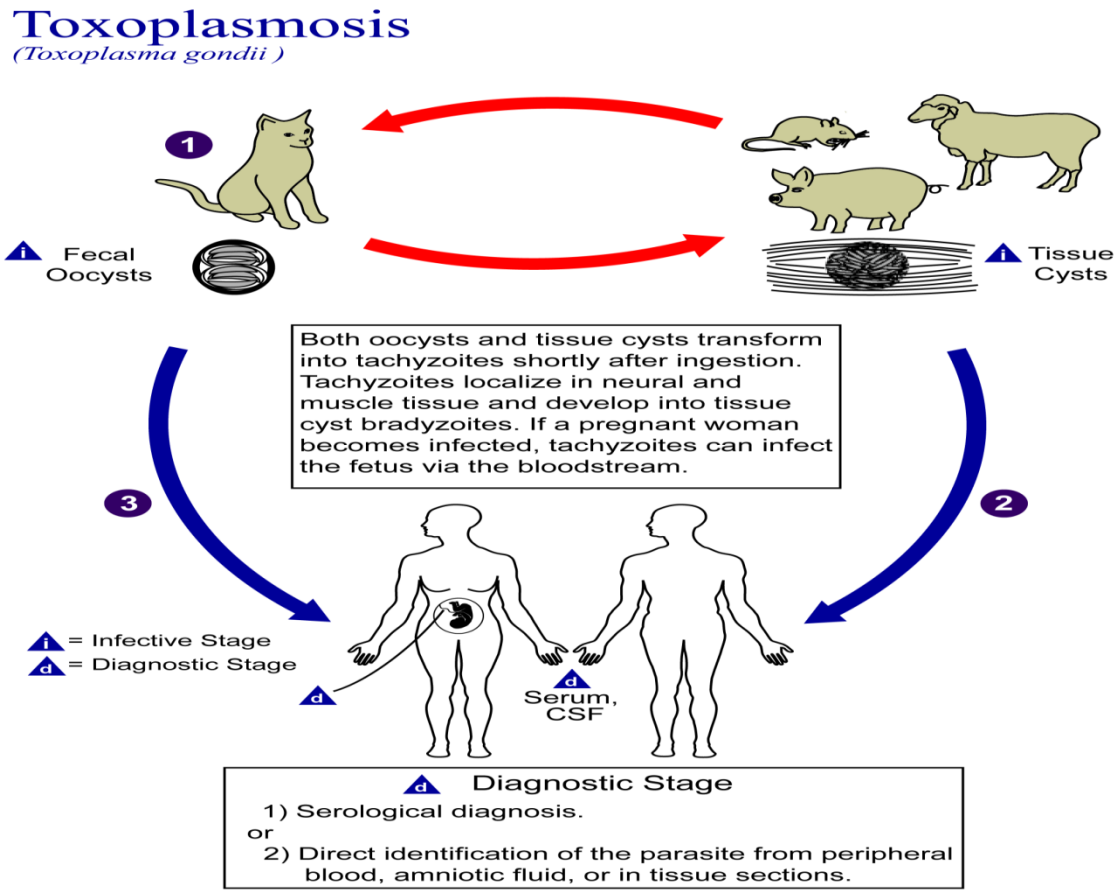
- Seizures.
- Guillain-Barre syndrome – an immune-mediated neurological complication that can occur following infection.
- Post-infectious acute disseminated encephalomyelitis / post-infectious brainsterm encephalitis.

PARASITIC AND FUGAL DISEASES OF THE NERVOUS SYSTEM

Diseases of the nervous system of this group are met infrequence and characterized by a nonspecific clinical picture.

TOXOPLASMOSIS

Etiology.The causative agent of the disease is the intracellular parasite of Toxsoplasmosis gondii, which belongs to the type of pronosoa, class of sporozola.



Picture 41. Toxoplasmosis *(https://www.wikiwand.com)*

Clinical picture.The disease is rather widespread and may have an acute, chronic, or latent form. In most cases the neurotoxoplasmosis is followed by the accompanying damage of other organs and systems.

The acute toxoplasmosis is characterized by the sharp beginning with chills and high temperature to 39-40^{0}C preceded by the prodromal stage (7-12 days). On the background of the generalized lymphadenopathy, the lesion of the cardiovascular system, parenchymatous organs, macullopapular rash (the variable symptom) the clinical picture of the encephalitis or meningocephalitis (the liquorhypertensive, meningeal syndrome, the pyramidal and cerebellar failure against the affected consciousness) with the lymphocytic pleocytosis in the cerebrospinal fluid develops. The neutologic symptomatology progresses, and without the adequate therapy it can lead to death.

The chronic toxoplasmosis has a certain clinical polymorphism. Mainly it's the chronic encephalitis or encephalomyelititis. Less often the peripheral nervous cyctem is affected. The symptom-complex can be presented by the subthalamic, hypertencive-hydrocephalic, hyperkinetic, vestibulocerebellar, epileptiform, amyostatic, and asthenoneurotic syndromes. Due to the high affinity of the parasite to cells of the system of mononuclear phagocytes their periventricular localization with the corresponding symptoms of irritation and loss of functions depending on the process phase is characteristic. On the nature of the course the disease is chronically relapsing with intensifying of neurologic deficiency from exacerbation to exacerbation.

The latent toxoplasmosis occurs against the high host's immune resistance to a parasite. There are no clinical symptoms at all, and it is determined only during the X-ray examination of the skull or during the laboratory diagnostics.

*The congenital toxoplasmosis*is marked separately in connection with feutures of the clinical picture. The acute form is shown from the first day of life by the weakness, fever, maculopapular rash, mainly in the lower part of the stomach and on the legs. The hemorrhages into the sclera and mucosa can be observed. The liver, spleen, and lymph nodes may be affected. As for the nervous system it may be the themeningocephalitis. The Sebin's triad is characteristic: the chorioretinitis, calcificats in the brain and hydrocephalus, when the epileptiform

syndrome often joints. The cerebrospinal fluid is transparent, xanthochronic, with the lumphocyticpleocutosis. Often the disease has the primary or secondary-chronic character. In the first case it is clinically show at the age of 10-15 years for the first time, in the second – due to conservation of live parasites after etiotropic specific treatment the exacerbation periods are observed.

Diagnostics. The suspicion on the neurotoxoplasmosis occurs when the patient has the neurological symptoms against the subfebrile body temperature of the unknown etiology, the widespread lymphadenopathy (the soft enlarged painful lymph nodes of pasty consistence (never exposing to necrotic disintegration); the small dense nodes soldered into conglomerates that testifies to the previous exacerbations); the enlarged liver and spleen, and the chronic relapsing course of the disease. For the diagnosis they use the skullroentgenography (numerous calcificats with a diameter of 1-4 mm, which are localized in the cortex and depth of gemispheres), sometime – the epidemiological anamnesis (domestic cats, the use of uncooked meat).

There are the following methods of verification:

1. Direct:
   - The microscopy of the cerebrospinal fluid, the biopsy of the lymph nodes with the corresponding staining of material;
   - The biological method is the infection of mice and identification of parasites in organs of the dead animals; infection of kittens and identification of oocysts in excreta.

The direct method are less informative due to significant difficulties of isolation of toxoplasmata in people.

2. Inderect:
   - The compliment binding reaction with the toxoplasmic antigen
   - The immunofluorescence reaction
   - The enzyme immunologic assay

– The intracutaneous test with toxoplasmin (the technique on the type of the Mantoux test).

The positive results of these tests testify to presence of antibodies to parasites; at the same time it isn't confirmation of the toxoplasmosis genesis of the disease. Increase of the titer of antibodies with development of the disease and regress of symptoms are of great importance during the specific treatment on the positive results of immunologic assays on the toxoplasmosis.

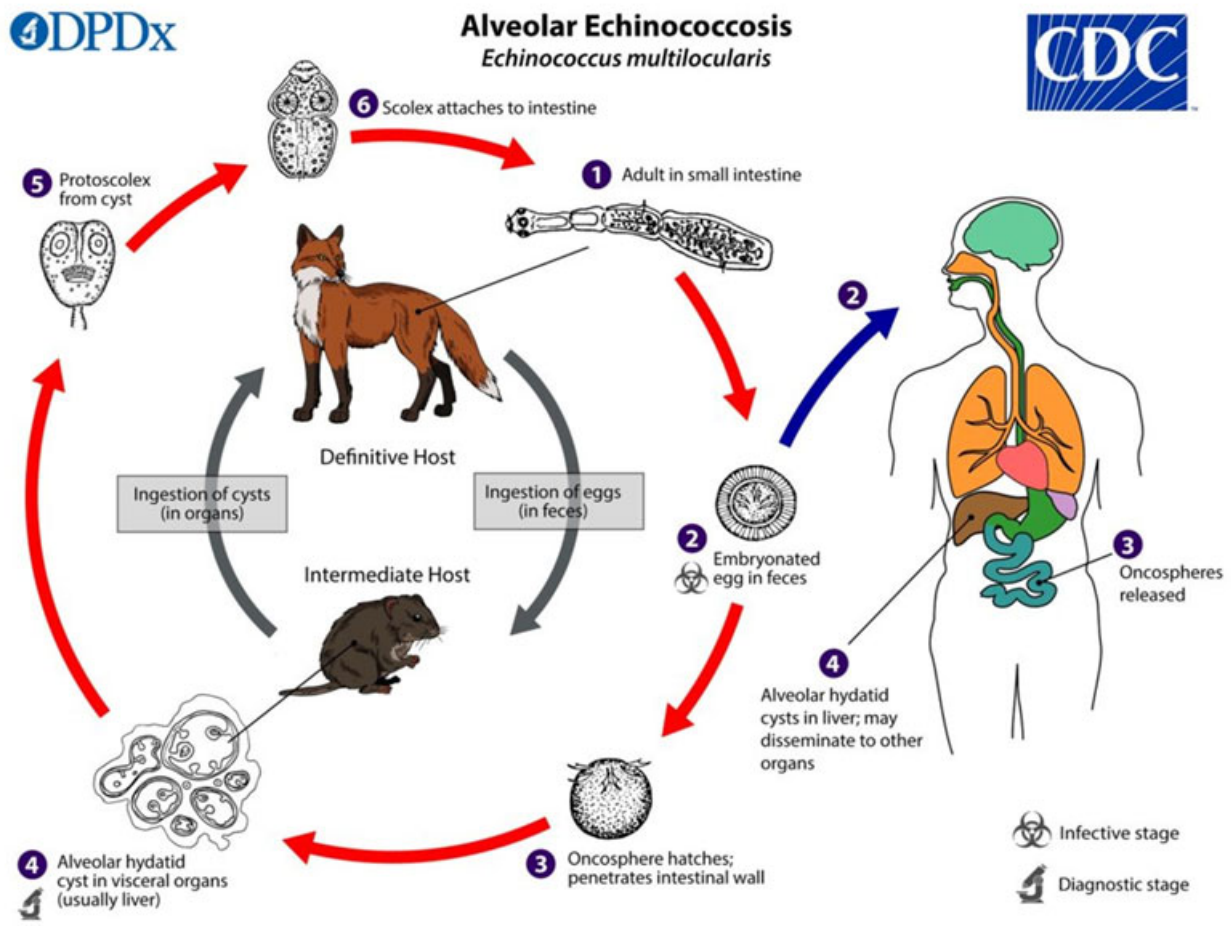
The differential diagnostics is carried out with the encephalitis and encephalopathy of other etiology. There is the less acute onset, the long period of fever, signs of generalization of infection (rash, hepatolienal syndrome, icterus). Result of compliment binding reaction and toxoplasmosis test chow increase of the titer of antibodies in the blood serum and cerebrospinal fluid. The antibodies to toxoplasmosis antigens appear on the 3-4 week of the disease, i.e. the positive reaction to the toxoplasmosis on the 3-5 day of the acute encephalitis denies the parasitic genesis of the disease. The differential diagnostics is carried ouy also with the brain neoplasms.

Treatment. In the acute and chronic toxoplasmosis they prescribe clindamycin 1.2 – 2.4 g for 2-4 injections daily for 10-14 days, or pyrimethamine by cycles for 5 days with an interval of 7-10 days (in combination with sulfa-preparations) 50 mg, then – 25 mg per day. In addition to the specific therapy they carry out the antibacterial, detoxication, and sedative treatment. If it's necessary, they prescribe glucocorticoids in the average therapeutic doses.

Prophylaxis. The congenital toxoplasmosis: the treatment of pregnant women with the expressed form of the disease is carried out to two cycles with an interval of 10 days, since the 16 week of pregnancy (not earlier than the II trimester). On requirement the 3 cycle can be started in a month. In the acquired toxoplasmosis people have to wash hands after contact to raw meat, pets, soil, vegetables, and they must eat only cooked meat.

ECHINOCOCCOSIS

Etiology. The causative agentsis a larva of the echinococcus (Echinococcusgranulosus). The main hosts of a parasite are dogs, wolves, foxed and other predators, as the echinococcus lives in their intestines.



Echinococcus multilocularis *life cycle*

Picture 42. Echinococcosis multilocularis life cycle *(https://www.wikidoc.org)*

Clinical picture. The clinical picture depends on localization of a parasite, quantity of bubbles, their localization concerning outflow of the cerebrospinal fluid, brain meninges and bones of the skull. There are the following stages: latent

(asympromatic); from the moment of invasion up to emergence of the subjective symptoms; obsolete sings; expanded picture; complication.

The early occurrence of the general or focal epileptic attacks with gradual development of transitional motor and sensitive disorders is characteristic. In50-60% of patients the bilateral tremor, lesion of the cranial nerves, muscle hypotonia, and slowly progressing central hemiparesis are observed.

The course of disease in children has some specific features: the illness begins with occurrence of the hypertensive syndrome, which has the two-phase course (increase of severity of clinical signs to the divergence of the cranial sutures with subsiding and returning of the symptoms on conditional of further growth of a cyst). The disease is characterized by the headache (according to localization of a parasite); it intensifying in horizontal position, especially in a prone position on the side of a cyst and at percussion. The skull bones are often involved into the pathological process: the pressure upon them causes the symptom of crackle of parchment in children and further, in certain cases, leads to formation of the bone defect and even the release under the skin. Due to the frequent localization of a parasite in the frontal lobe of hemispheres the psychoorganic syndrome is observed. Features of the echinococcosis of the brain ventricles are the development of hypertension-hydrocephalic syndrome with Brun's attacks. In the multiple echinococcosis the main focus is clinically shown at beginning, and in the process of development of the disease there are signs testifying to existence of other centers of lesion.

The complications of a disease are the rupture of a cyst into the liquor system and its suppuration. In the first case, when a rupture is into the subarachnoid space, the sudden attack of the headache, vomiting, high temperature to 40-41^{0}C with allergic dermal reaction (rash), and meningeal, epileptiform, and pyramidal syndromes are observed. When a rupture is into the brain ventricles, the hormetonic syndrome joint. The suppuration occurs as the abscess of the brain, its

rupture leads to the development of the acute meningitis or meningocephalitis, most often with a fatal outcome.

Diagnostics. The clinical picture of the echinococcusis deprived of specific signs. Data on the echinococcosis of other organs and result of additional studies (the transitional eosinophilia of the blood lymphocytic pleocytosis with positive albumonous reactions in the cerebrospinal fluid, rising of level of succinic acid in the blood plasma) can be the reason for assumption of the parasitic origin of the neurologic symptoms.

The specific assay is the Kasoni's test, which monitoring in 5-10 minutes and making the compliment binding reaction. The pseudo-negative results are observed in the cachexia, pyesis, or calcification of the echinococcus cyst. The CT and MRI allow detecting the cavitary formations; they are the most informative methods in diagnosis of a disease.

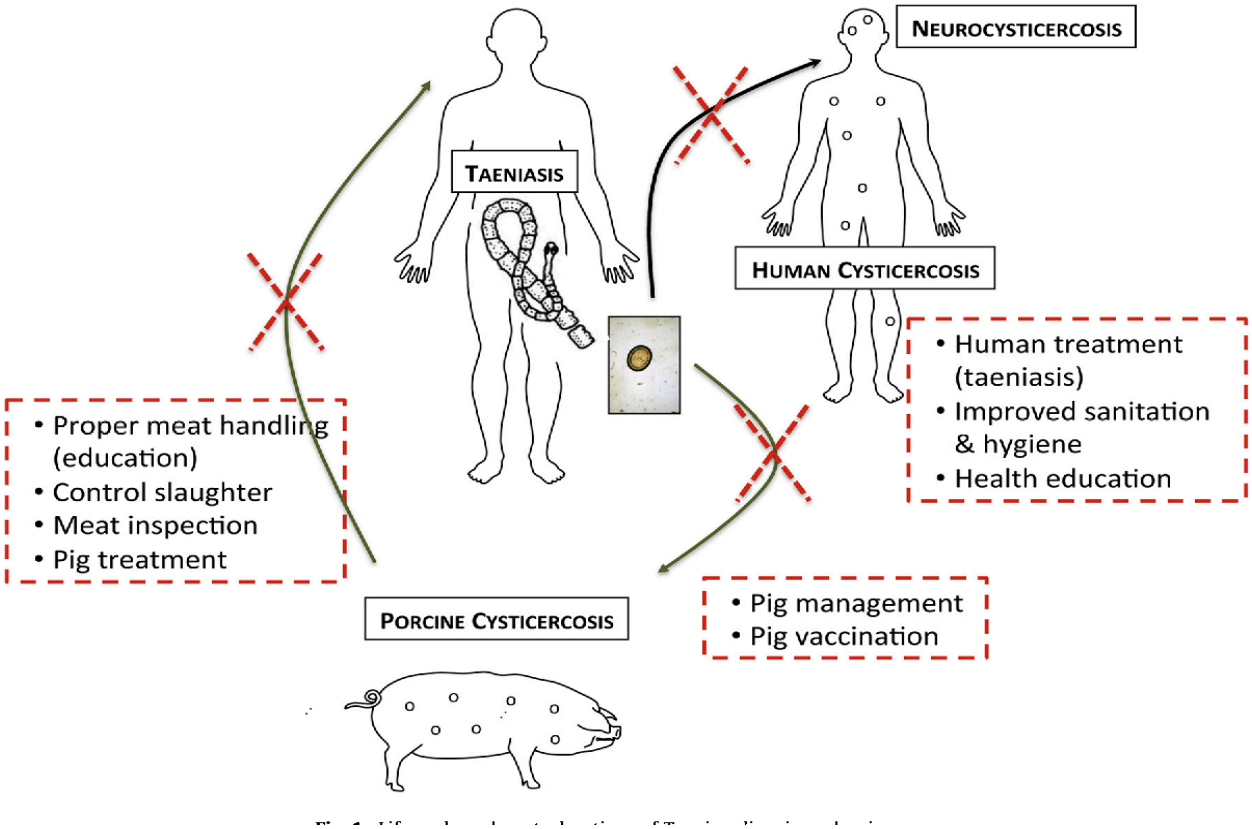
The differential diagnostics is carried out with the neoplasms, arteriovenous malformation, focal meningocephalitis, and cystricercosis.

Treatment.In the precence of the single echinococcosisbubble they carry out its enucleation. In the cases of the multiple unicameral or alveolar echonococcosis (ehich is multiple and secondary) the surgery has the palliative character and indications tj it are defined individually. The complications are treated symptomatically.

The prophylaxis includes veterinary supervision for farm animals, prevention of eating of organs of the dead of animals by dogs, keeping of rules of personal hygiene.

CYSTICERCOSIS

Etiology. Cysticercocosis are the larva stage of a pork tapeworm (Taeniasolium). The main host of the last is human; the mature form of helminthes lives in his (her) intestines. In the process of reproduction the tapeworm segments full of eggs (hexacanth) are excreted with faeces.

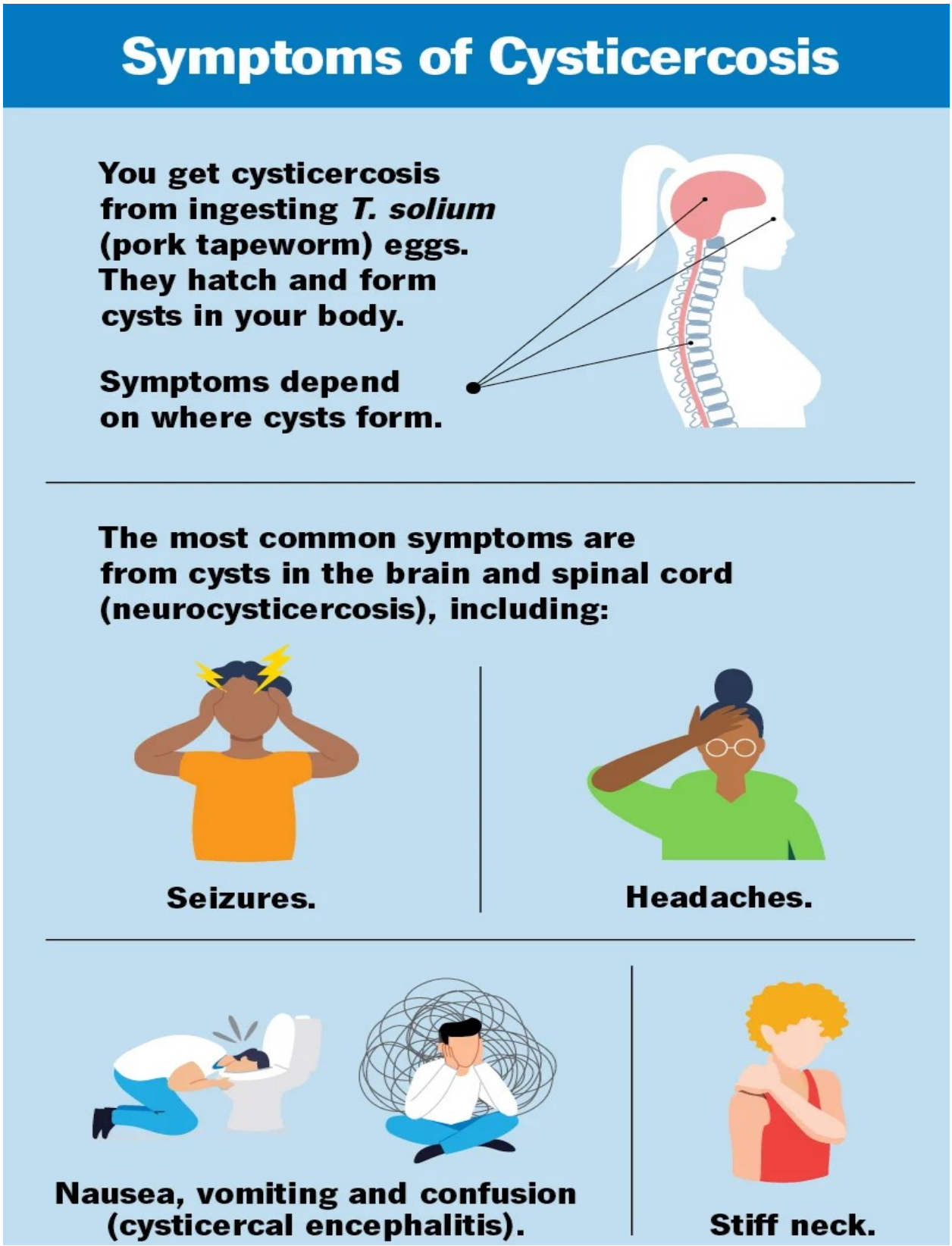


Picture 43. Cysticercocosis*(https://www.sciencedirect.com)*

Clinical picture. The disease has extremely dynamic clinical picture with labile neurologic symptoms. Almost in all cases against the main syndrome the focal remote symptoms caused by presence of parasites in other regions of a brain are always determined.

The symptom-complex can be presented by the epileptiform, pseudo-tumor, hypertension-hydrocephalitic, and meningeal syndromes.

Picture 44. Symptoms of cysticercosis*(https://my.clevelandclinic.org)*



The epileptiform syndrome is most characteristic for the brain cysticercosis. It is shown in the form of the focal or secondary-generalized attacks. Lack of the

resistant symptoms of abaissement after attacks, and also liability of the epileptiform syndrome with increasing and termination of attacks for a certain period, polymorphism of the clinical picture is characteristic. Sometime the seizures occur several times a day, the development of the epileptic status is possible.

The pseudo-tumor syndrome is shown by mild multifocal symptoms without the progredientcoursr and mental disorders. When the parasite is located in the cerebral hemispheres the central paresis of the mimic muscles and tongue, disorder of sensitivity without accurate localization become perceptible. In the case of the spinal localization of process the syndrome course has the progredient character, sometimes with the expressed neurologic deficiency.

The hypertension-hydrocephalic syndrome accompanies the cysticercosis of the brain ventricles. It is characterized by the sharp headache with womiting, the compelled position of the head, and dizziness. If the localization is in the third ventricle, the hypothalamic (diencephalic) syndrome caused by a secondary inflammation is observed. Often it is followed by the lesion of extrapyramidal nuclei and visual disorders. There are the Bruns's attacks (the sudden dizziness because of the head turns, sharp headache, vascular reactions, bradypnea, bradycardia, frequently with loss of consciousness and tonic cramps) caused by the parasitic irritation of the nuclei of the floor of the rhomboid fossa, and the acute liquor hypertension as a result of occlusion of the median foramen of the fourth ventricle.

The meningeal syndrome is a signs of the chronic relapsing leptomeningitis, mainly basal.

The diagnostics is based on the clinical picture of the brain inflammatory andspace occupying processes with prevalence of symptoms of an irritation, lack of the progredient course. Identification of cysticerci in muscles and hypodermic fatty tissues according to the clinical implications or during the X-ray examination allows assuming the parasitic genesis of the neurologic symptomatology. The CBR

(Complement Binding Reaction) of the cerebrospinal fluid and blood with the cysticercus antigen in dynamics is specific. The negative result isn't the criterion for excluding the disease. The CT and MRI allow visualizing the parasites in the form of the rounded encapsulated hypodensive formations.

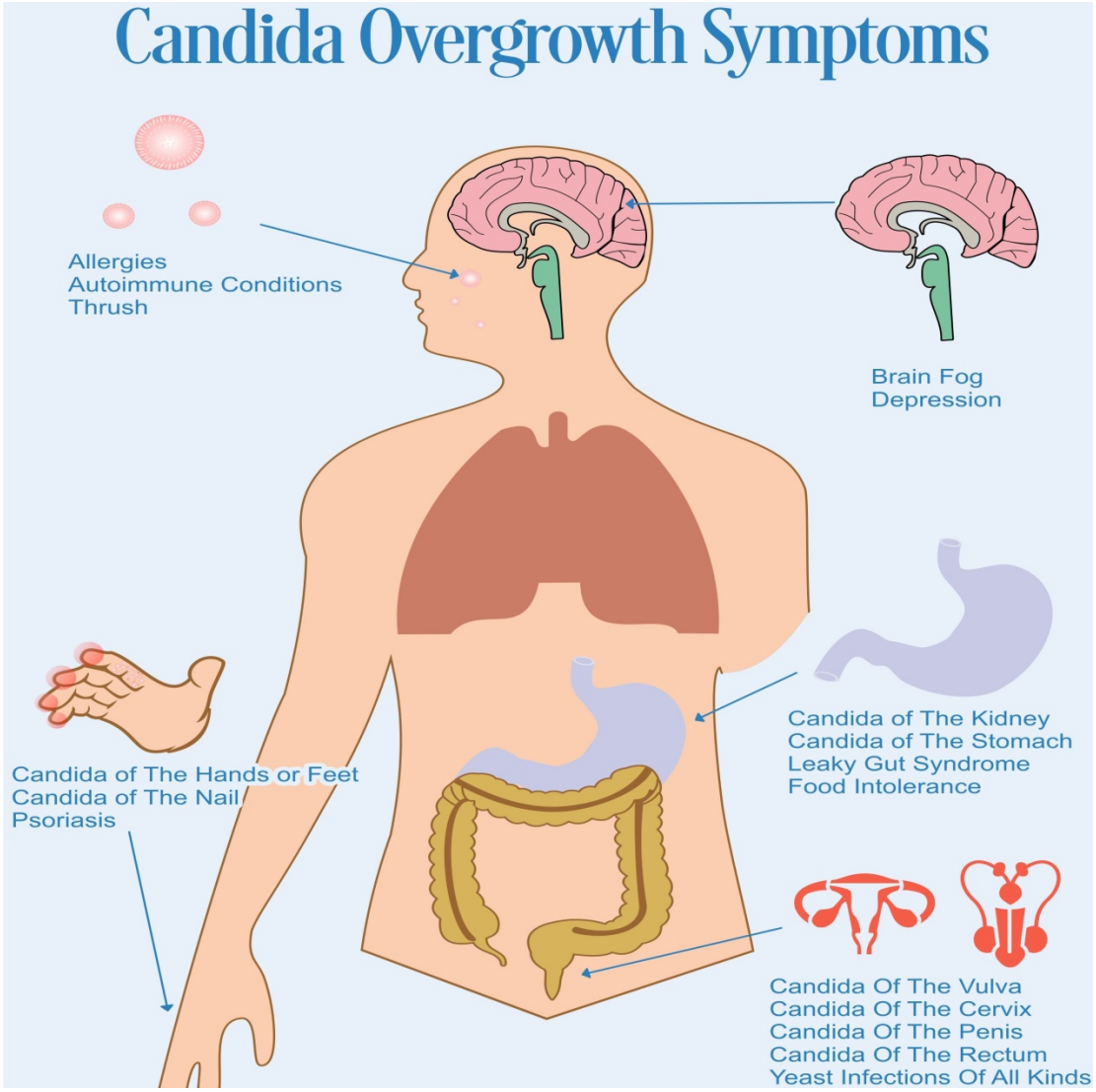
The different diagnostics is carried out with the symptomatic epilepsy, meningocephalitis of other etiology, multiple encephalomyelitis, neoplasm, and encapsulated hypodensive formations.

The treatment cysticercosis can be therapeutic and surgical. As for the medicinal preperations they prescribe praziquantel 50 vg/kg for 15-30 days, albendazol 0,4 g twice a day for 8-10 days. The surgical treatment is carried out in the presence of a single parasite in available to intervention place, which is rare due to its various focal placements.

The prophylaxis consists in keeping of rules of personal hygiene, medical observation of persons of group of risk.

CANDIDIASIS

Etiology. The causative agents of the disease are yeast fungi of the genus Candida (most often Candida albicans). These fuhgus-aerobes are considered opportunistic; their virulence considerably depends on the condition of the macroorganism as medium of their living. They can be observed more then in 20% of healthy people on the skin, mucosa of the mouth, intestines, or vagina.



Picture 45. Candidiasis symptoms *(https://www.creative-biolabs.com)*

Clinical picture. The lesion of the brain tissue is characterized by gradual increase of the focal symptoms with further spreading in the exhausted patients against the severe somatic pathology, which is followed by the low-grade fever or normal body temperature. The defeat of the meningitis occurs in the form of the diffuse cerebrospinal meningitis, meningocephalitis, or basal meningitis. The meningeal, liquor-hypertension syndromes, focal symptoms, disorders of consciousness, and dysfunction of the cranial nerves are characteristic. Presence of the septic and pulmonary forms of the candidiasis, especially in children and elderly people are of great diagnostic value.

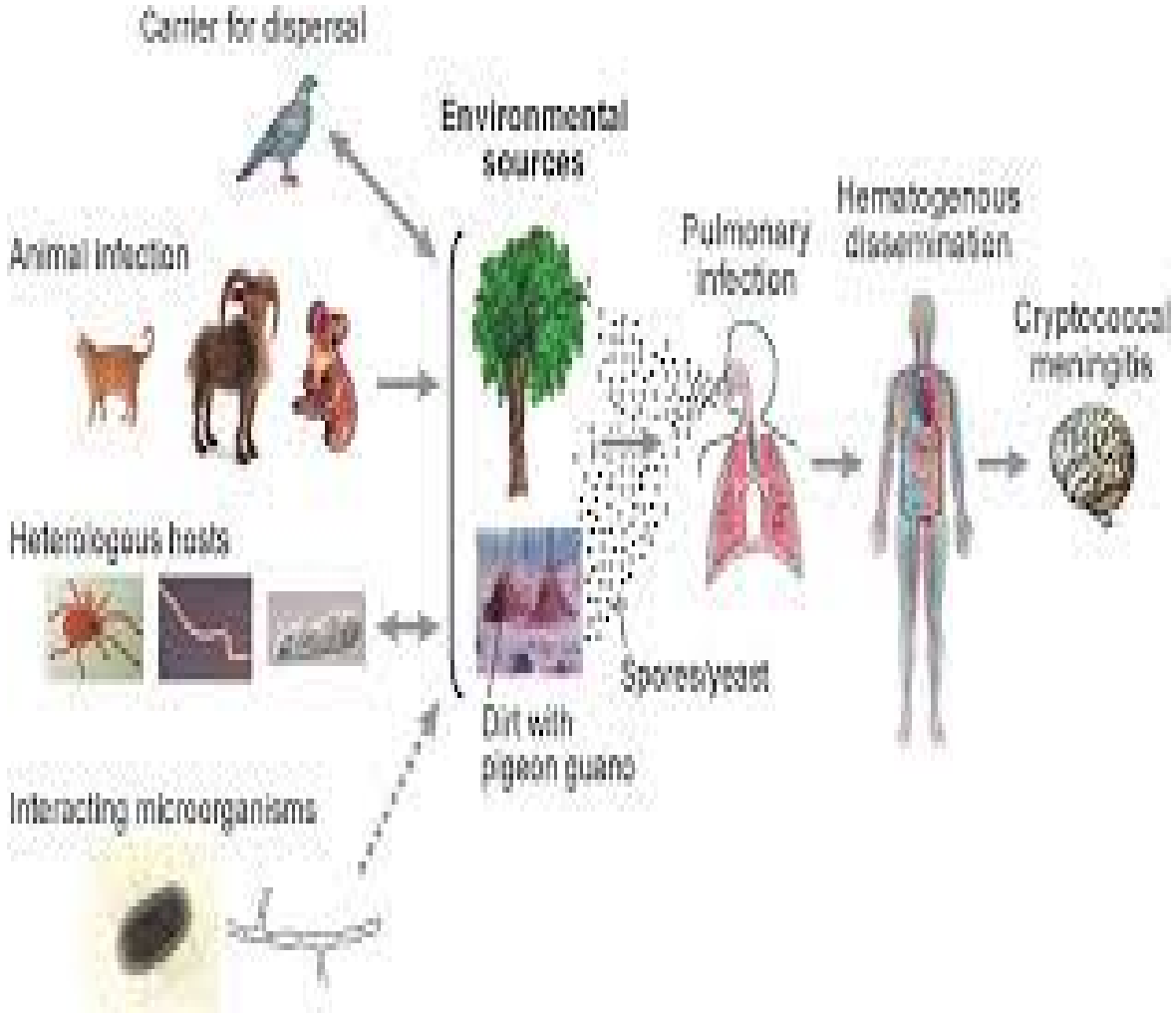
The diagnostics is based on identification of a fungus in the cerebrospinal fluid (slight increase of the liquor pressure, neutrophilicpleocytosis, rising of the protein level). The lergological and serologic diagnostics (CBR, RP – reaction of precipitation) is possible, but in the exhausted patients with the hyporeactivity of specific system the reactions may be negative even in the acute period of an illness. Certain diagnostic value has the increase in antibody titer.

The differential diagnostics is carried out with the tumors and brain abscess, nonspecific inflammatory lesions of the meningitis. Differentiation of these states is based on data of the anamnesis, features of the clinical picture (a course, existence of other forms of the candidiasis), result of mycology of the cerebrospinal fluid and blood, definition of the increased antibody titer to sort Candida fungi in the blood serum.

Treatment. The causal treatment consist in use of amphotericin B (250 UA/kg) iVdriply with intervals 2 days every 6 days and every 7-10 days after 20 injection has been made. The course of treatment is 4-8 weeks, and the course dose is 1500000-2000000 UA/kg. They also prescribe amphogluaminum, fluconazole intravenously – 200-400 mg daily for 7-14 days to a month. The pathogenetic therapy consist in desensitization and detoxification, restorative treatment, and stabilization of the hormonal bakance.

CRYPTOCOCCOSIS

Etiology.The causative agents of the disease are Cryptococcosisneoformans fungus-aerobes. On the antigenic structure they are the encapsulated and somatic antigents; the antibody formation to them is extremely low.



Picture 46. Etiology Cryptococcosis symptoms *(https://link.springer.com)*

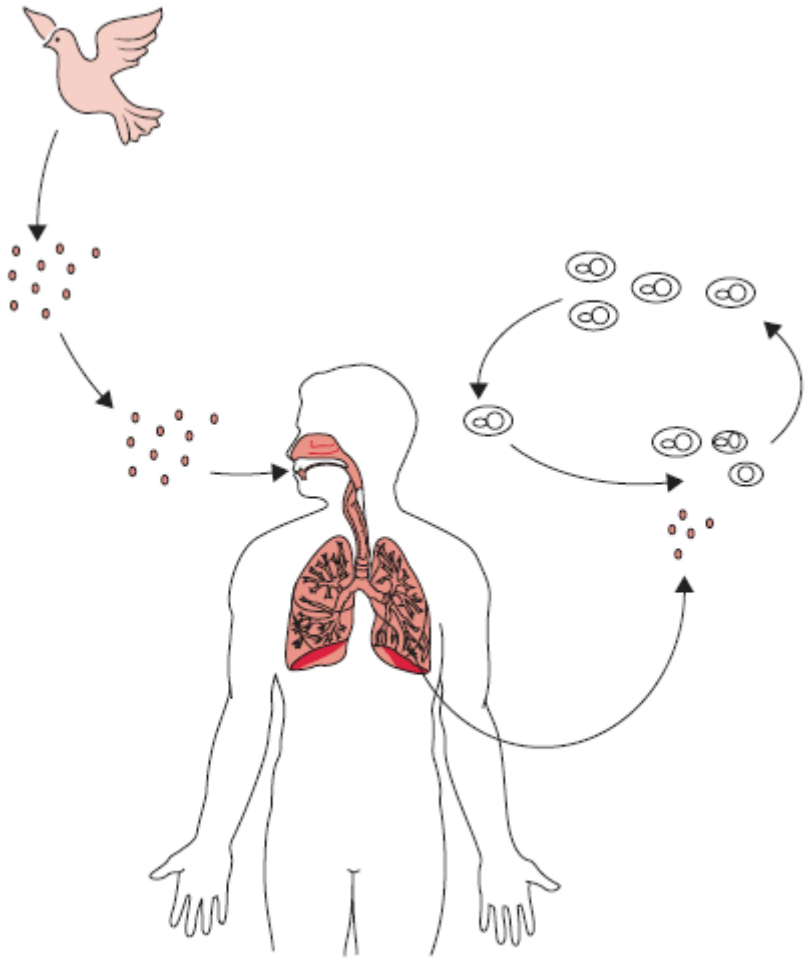


FIG. 73-1. Transmission of *Cryptococcus neoformans*.

Picture 47. Transmission of Cryptococcus neofarmans *(https://link.springer.com)*

Clinical piture. There are some kinds development of pathological process; the formation of giant cel granulomas with eosinophilic and lumphocytic infiltration, caseous necrosis with possible formation of cyst with parasites in perivascular spaces (the gelatinous form); granulomatous form with histiocytic reaction, epithelioid and fiant cells, and sign of the phagocytosis of fungi. The calcification is absent.

Diagnostics. For verification of the diagnosis they carry out the microscopy of the centrifugation of the cerebrospinal fluid without staining. Use of the agglutination test, reaction of precipitation, complement binding reaction, and

biological method with infection of animals is possible. Fetures of the antigenic structure of a fungus cause periodic negative reaction against the developed.

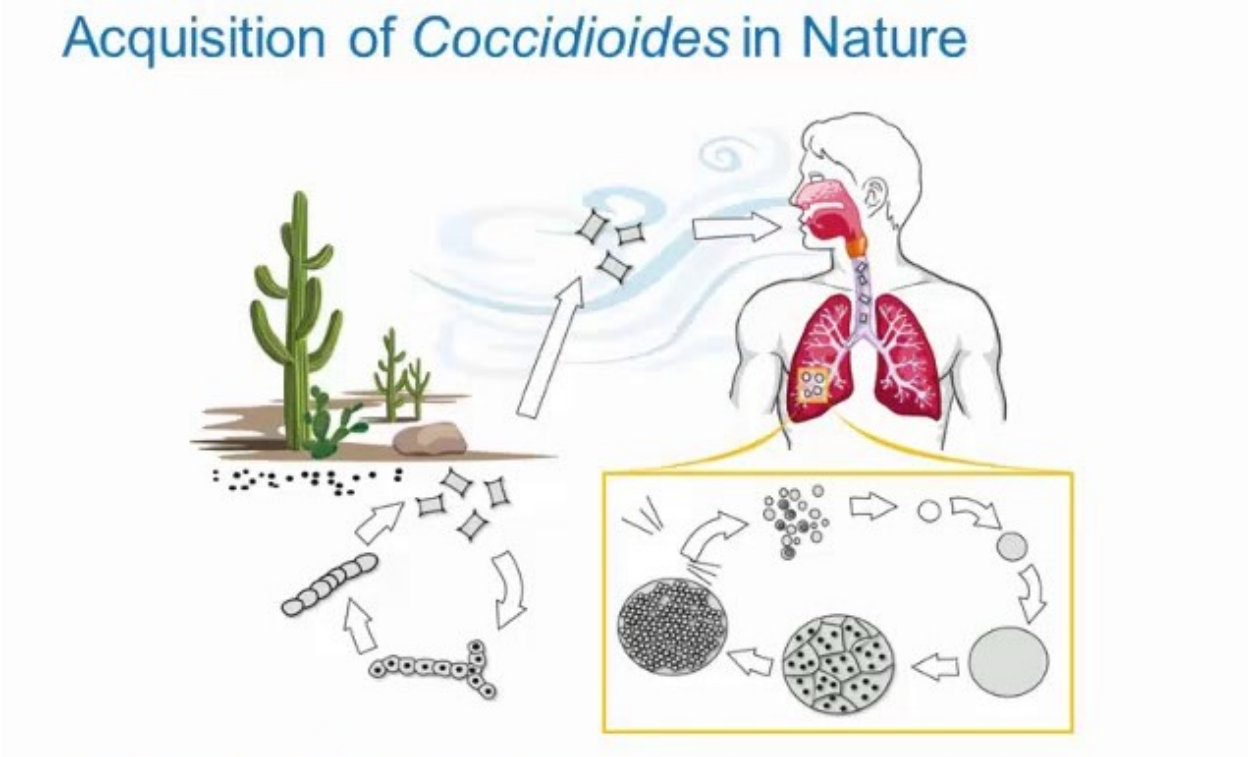
The differential diagnostics is carried out with the tuberculous meningitis, tumors, abscess, and subdural hematoma.

The treatment is similar to the treatment of the candidiasis of the nervous system. Additional endolumbar introduction of amphotericin B is recommended.

The prophylaxis consist in carrying out the well-timed diagnostics and therapy of the somatic cryptococcosis, keeping of rules of personal hygiene.

COCCIDIOIDOMYCOSIS

Etiology. The causative agent is Coccidioidesimmits, the fungus of family of Phycomycetes, existing in two forms: micellar, which lives in the environment with formation of the endemic foci of high contagiousness; and tissue (parasitic), living in the patient's organism who isn't contagious. The susceptibility of the person to infection with this fungus is about 100%. The virulent fungus spores get into the lungs with air.



Picture 48. Acquisition of coccidioides in nature *(https://cronkitenews.azpbs.org)*

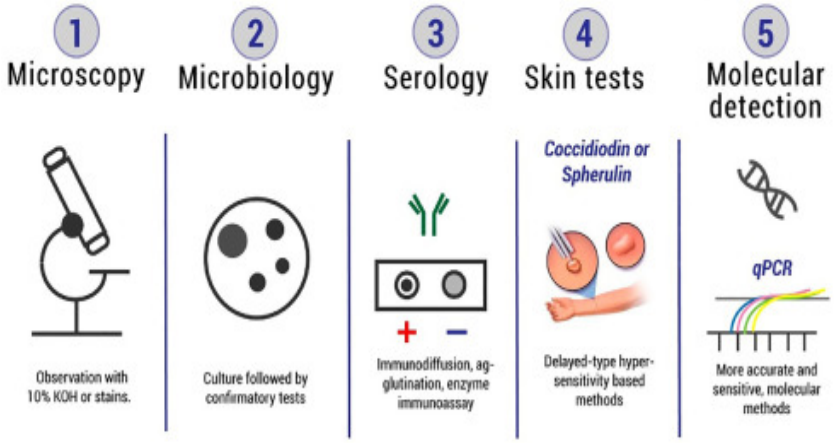
Clinical picture. The meningitis with meningeal and liquor-hypertension syndromes, signs of irritation of the cortex of cerebral hemispheres, disorders of consciousness and memory is about 25% of cases of the dissemination lesions in the coccidioidomycosis. They observe the lymphadenopathy, hectic fever, and specific pneumonia. In various organs the granulematous foci with disintegration and formation of fistulae are formed.

The diagnostics consist in microscopial identification of fungi in the cerebrospinal fluid with further growth on the nutrient medium. The serologic diagnostics (reaction of precipitation, complement binding reaction) and allergic samples are not critical in connection with common antigenic properties of coccidian and other fungi. Also the biological method is used. If the skin test is negative, and serological test will always be negative, too.

The differential diagnostics is carried out with other mycoses, nonspecific meningitis, and tuberculosis.

The treatment is similar to the treatment of the cryptococcosis on the background of a good high-albuminous nutrition, restorative therapy, use of vitamins of the group B, and blood preparations.

The preventive measures consist in the control of people who are coming back from the endemic centers by carrying out the X-ray examination and laboratory diagnostics.



Picture 49.The preventive measuresof the cryptococcosis*(https://www.sciencedirect.com)*

TEST

1. What are the signs of meningococcal meningitis?

   A. Fibrillary tremor In muscles, bulbar disturbances
   B. Paraplegia, fibrillar twitching
   C. Protein-cellular dissociation in CSF, bulbar disturbances
   D. Headache, hyperthermia, Kerning's sign
   E. Paresis of extremities, hyperkinesis

2. What are the features of tuberculous meningitis?

A. Respiratory and cardial malfunction, bulbar disturbances
B. Lymphocytic pleocytosis, fibrin membrane in CSF
C. Neutrophilic pleocytosis, paresis of the extremities
D. Positive Wassermann reaction, sensitive disorders
E. Protein cellular dissociation in CSF, paraplegia

3. What are the characteristics of epidemic encephalitis in acute form?

A. Pathological somnolence, oculomotor disturbances
B. Bulbar disturbances, paralyses of the extremities
C. High muscular tone, hypokinesis
D. Low muscle tone, hyperkinesis
E. Mannequin's posture, hypomimia

4. What are the characteristics of tick-borne encephalitis?

A. Pathological somnolence, oculomotor disturbances
B. Hypomimia, hypokinesis
C. Sensitive disorders, ataxia
D. Conductive hemihypeshesia, spastic hemiplegia
E. Flaccid pareses of upper extremities, bulbar disturbances

5. Indicate main clinical features of tabes dorsalis.

A. Bulbar disturbances. Bernard-Homer syndrom

B. Meningeal symptoms, papilledema
C. Pathological somnolence, oculomotor disturbances
D. Sensitive ataxia, the absence of the knee and Achilles reflexes
E. Paraplegia, fibrillar twitching

16. For encephalitis is characterized (4):
A. Central paresis of limbs
B. Fever
C. Headache
D. Rash on the face and upper limbs
E. Epileptic seizure

17. In cerebrospinal fluid with encephalitis revel:
A. Increase in the number of cells more than 1000 in ml
B. Lymphocytic pleocytosis
C. Glucose is increased
D. The formation of fibran films

18. Treatment of encephalitis of the not clear etiology
A. Piracetam
B. Acyclovir
C. Baclofen
D. Immuneglobulin
E. Prednisone

19. Etiology of the purulent meningitis (2):
A. Pale spirichete
B. Pneumococcus
C. Streptococcus

D. Mycobacterium TB

E. Viruses

20. Etiology of serous meningitis (3):

A. Enteroviruses

B. Meningococcus

C. AIDS virus

D. Armstrong virus (choriomeningitis)

E. Stophylococcus

21. For purulent meningitis is typical (3):

A. Neck stiffness

B. Kernig’s sign

C. Epileptic seizures

D. Headache

E. Aphasia

22. For serous meningitis is characteristic (2):

A. Headache

B. Hemiparesis

C. Changes of consiousness, coma

D. Neck stiffness

E. Hemorrhagic rach of face and body

23. Main additional method of diagnosis of meningitis:

A. MRI head

B. Lumbar puncture

C. CT scan

D. Electroencephalography

E. Duplex scanning of the carotid and vertebral arteries

24. Fortuberculosis meningitis specifically (3):

A. Chronic course
B. Sensory aphasia
C. Damage to the oculomotor nerves
D. Headache
E. Cerebellum ataxia

25. In cerebrospinal fluid with purulent meningitis detected (2):

A. An increase in the number of cells up to 1000 or more per mkl
B. Lymphocytic pleocytosis
C. Decreased of protein
D. Decreased of sugar
E. The formation of fibrin limbs

26. In cerebrospinal fluid with serous meningitis detected:

A. An increase in the number of cells up 100 or more per mkl
B. Neutrophil pleocytosis
C. Normal glucose of CSF
D. Decreased of protein
E. Colorless and treanparent liguar

27. Main treatment of purulent meningitis:

A. Antibiotics
B. Hormones
C. Plasmopheresis
D. Immuneglobulin
E. Repeated lumbar puncture

28. Poliomyelitis is (2):

A. Caused by the Coxacci
B. Occurs predominantly in children under the age of 10 years
C. Manifested by the development of motor aphasia
D. Manifested b development of cerebellum ataxia
E. Manifested by peripheral paresis of lower limbs

29. Clinical form of neurosyphilis (3):

A. Tabes dorsalis
B. Meningitis
C. Syndrome of lateral amyotrophic sclerosis
D. Progressive paralisis
E. Neuralgia of trigeminal nerve

30. Meningovascular syphilis is (3):

A. Manifested by cerebral stroke
B. Manifested by spinal stroke
C. Manifested by lateral amyotrophic sclerosis syndrome
D. The most common form of neurosyphilis is currently

31. Damage to the nervous system in acquired immunodeficiency syndrome (AIDS) (4):

A. Meningitis
B. Encephalitis
C. Myelopathy
D. Damage of facialis nerve
E. Myopathy

32. AIDS-dementia is (4):

A. Occurs in half of the patients (in the advanced stage of the disease)
B. Manifested memory disorders
C. Manifested of ataxia and central paresis of limbs
D. Manifested of epileptic attacks
E. Regresses with adequate treatment of the underlying disease

33. A patient had fever, chills malaise for 3 days then flaccid paresis of lower limb appeared which changed on the spastic tetraparesis with bulbar syndrome, disorders of breathing in a few days. Which diagnosis is the most appropriate?

A. Myelitis of upper cervical level of the spinal cord
B. Myelitis of cervical enlargement level of the spinal cord
C. Poliomyelitis, paralitic form
D. Spinal cord
E. Amyotrophic lateral sclerosis, bulbar form

34. A 65-year-old patient has been suffering fromchoking while eating slurred speech, incorrect articulation, fibrillar twitching and hypotrophy of the tongue muscles, and oral automatism signs for there month. What is the most likely diagnosis?

A. Poliomyelitis, bulbar form
B. Amyotrophic lateral form
C. Amyotrophic lateral sclerosis
D. Acute disseminated encephalomyelitis
E. Multiple sclerosis
F. Ischemic stroke of brain steam

TASKS

1. A 10-year-old boy complains of severe headache and nausea. His body temperature is 39.3 °C; examination reveals labial herpes, petechial leg hemorrhages, occipital muscle rigidity, and positive bilateral Kernig's sign.

    1. What disease has most likely affected the patient?
    2. Where must the patient be treated?
    3. What additional diagnostic procedures should be performed?

2. A patient complains of severe headache and fever. He has been feeling ill for two days, his body temperature is 39.3 °C. Examination reveals labial herpes, decreased muscle strength in the left extremities, increased tendon and periosteal reflexes on the left side, and the positive Babinski reflex on the left side as well.

    1. Are there any focal symptoms of the patient? If so, what are they?
    2. What disease has most likely affected the patient?
    3. What additional diagnostic procedures should be performed to confirm the diagnosis?

3. A 32-year-old woman complains of double vision, headache, and nausea. Before these symptoms appeared, she had been coughing for a month and her body temperature was 37.6 °C. Neurological examination reveals neck stiffness and positive bilateral Kernig's sign. CSF analysis demonstrates lymphocytic pleocytosis (650 per 1 mm^3), fibrinous membrane, protein — 1.3 g/l, glucose — 1.2 mmol/l.

    1. What syndromes have occurred in the patient?
    2. What disease has most likely affected the patient?
    3. Determine diagnostic options.

4. A 40-year-old woman complains of severe headache, nausea, vomiting, and elevated body temperature up to 38.6 °C. These complaints have occurred after acute pneumonia. Neurological examination reveals neck stiffness, bilateral

positive Kernig’s sign and positive Brudzinski’s sign. CSF analysis demonstrates neutrophilic pleocytosis, increased protein level, and pneumococci.

1. What syndromes have occurred in the patient?
2. What disease has most likely affected the patient?
3. Determine treatment options.

5. A patient's body temperature is 38.2 °C; examination reveals neck stiffness and bilateral positive Kernig's sign. This condition has developed secondary to pyogenic otitis. CSF analysis demonstrates neutrophilic pleocytosis.

1. What is the most likely diagnosis?
2. Determine the examination methods and treatment options.

6. A 50 year-old patient complains of acute thoracic pain and elevated body temperature up to 38.5 °C. These complaints have occurred after his exposure to cold. Neurological examination reveals lower central paraparesis, conductive hyperesthesia of all types of sensitivity from the middle thoracic segments, and central urination disorders.

1. What disease has most likely affected the patient?
2. What additional diagnostic procedures should be performed to confirm the diagnosis?

7. A 7-year-old girl complains of severe headache and vomiting. Her body temperature is 39 °C; examination reveals sore throat, petechial skin rash, neck stiffness and positive bilateral Kernig's sign. The peripheral blood test demonstrates leukocytosis — $13.0*10^9$, erythrocyte sedimentation rate of 35 mm/h; CSF analysis demonstrates neutrophilic pleocytosis (250 per 1 mm^3).

1. What syndromes have appeared in the patient?
2. What is the most likely disease?
3. Which of the patient's present syndromes is the most significant for making a diagnosis?

8. A patient complains of weakness, drowsiness and double vision. He has been feeling ill for three days, body temperature is 38.6 °C. Neurological examination reveals left eye divergent strabismus, and a syndrome opposite to Argyll-Robertson's syndrome.

1. What syndrome is characterized by both pathological drowsiness and eye movement disorders?
2. What disease has most likely affected the patient?

9. A 32-year-old patient suffers from gradual hand tremor, stiffness and slowness of movement. The year before he had been ill with cold accompanied by fever, general weakness, headache, increased drowsiness, short-term double vision. Examination revealed mask-like seborrhea face, quiet monotonous speech, slow movements, sialorrhea, flexed posture, and walking with small steps; high muscle tone in the legs and fine tremor of the fingers at rest; the normal pupillary response to light and lack of response to convergence.

1. What syndromes have occurred in the patient?
2. What parts of the nervous system are affected?
3. Make a clinical diagnosis.
4. What treatment will you prescribe to this patient?

10. A 10 year-old child suffering from rheumatism has developed fast compulsive movements of the face and leg muscles resembling grimacing and motor restlessness. The limb muscle paresis is not determined, the muscle tone in the legs is low.

1. What is the most likely syndrome?
2. What is the name of the described compulsive movements?
3. What is the most likely disease?

11. A 35-year-old woman that has been bitten by a tick complains of weakness, headache, muscle ache in the shoulder girdle, and muscle weakness in the upper

limbs. Neurological examination reveals dropped head, flaccid paralysis of the arms with decreased tendon and periosteal reflexes. Sensitivity disorders are not found.

1. What structures are impaired? At what level?
2. What is the most likely disease?
3. What additional diagnostic procedures should be performed to confirm the diagnosis?

12. A patient with labial herpes complained of elevated body temperature to 38.0 °C and periodical Jackson's seizures in the left arm. Oneday later, she developed ataxia in extremities, tetraparesis, stupor, and then coma. Brain MRI revealed inflammatory lesion of the white matter, round-shaped, partially fused, up to 2 cm in diameter in the frontal and temporal lobes of the cerebrum and cerebellum.

1. What is the preliminary diagnosis?
2. What additional diagnostic procedures should be performed to confirm the preliminary diagnosis?
3. What treatment must be prescribed?

13. A child developed elevated body temperature of 37.8 °C, sore throat, abdominal pain, vomiting, and diarrhea. Three days later, he began feeling weakness in both legs. Examination revealed a reduction in active movement volume and muscle strength in the legs, hypotonia of the leg muscles and areflexia of deep reflexes. All types of sensitivity are preserved.

1. What syndromes have occurred in the patient?
2. What structure impairment can explain all the pathological symptoms?
3. Make a preliminary diagnosis.
4. What is the etiology of this disease?
5. What is the route of contamination?

14. A 60-year-old patient gradually developed unsteadiness increasing in the dusk

and dark. Examination revealed a reduction in muscle and joint sensation in the legs, lack of direct and consensual pupillary response to light while the pupillary response to convergence and accommodation was preserved.

1. What is the name of this syndrome of pupillary response pathology?
2. Impairment of what structure can explain these disorders?
3. What are the names of coordination disorders?
4. What is the most likely disease?

15. A patient who was treated for syphilis 10 years ago developed back pain, paresthesia, and radicular pain of the extremities. Neurological examination revealed hypalgesia in the T3—T4 segments and on the lateral surface of both calves, decreased knee and Achilles reflexes, and positive Argyll Robertson's syndrome. An ophthalmologist diagnosed primary optic disk atrophy.

1. Make a preliminary diagnosis.
2. What additional diagnostic procedures should be performed confirm the preliminary diagnosis?
3. What treatment must be prescribed?

16. A patient complains of severe headache, fainting, nausea, and vomiting. Examination reveals neck stiffness and positive bilateral Kernig's sign, slight ptosis of the right upper eyelid and right-sided hearing impairment. CSF analysis demonstrates lymphocytic pleocytosis (200 per 1 mm^3), positive Treponema Pallidum Immobilization and Immune Adherence Tests.

1. Impairment of what structures can explain such symptoms?
2. Make a preliminary diagnosis.

17. A patient complains of headache, blurred vision, pain in the major joints of his hands, feet and along the peripheral nerves of the extremities. Examination revealed tenderness on the exit points of the right trigeminal nerve branches, incomplete right eye closure, smoothed right nasolabial fold, and inability to raise

the right eyebrow. The tendon reflexes of the limbs are reduced and Kernig's sign is slightly positive; there is tenderness on the exit points of the femoral and sciatic nerves. Then the patient mentioned that before he had been bitten by a tick and there was a reddish nodule (lymphocytoma) on his chest. CSF analysis demonstrates lymphocytic pleocytosis and high blood protein.

1. What is the most likely diagnosis?
2. What additional diagnostic procedures should be performed to confirm the preliminary diagnosis?
3. What treatment must be prescribed?

18. A 35-year-old HIV-positive man complains of gradual deterioration of intellectual abilities (memory, attention) and behavior. The patient's face looks amimic and there are the symptoms of oral automatism; movements are slow, there is hand tremor at rest and while moving. Hyperreflexia of deep reflexes is determined.

1. What is the most likely disease?
2. What additional diagnostic procedures should be performed and what results can be obtained?

19. An HIV-positive patient suddenly developed a series of generalized seizures. Examination revealed impaired judgment, disorientation, slight meningeal symptoms, motor aphasia, and right-side hemi plegia. Brain MRI demonstrated ring-like foci in the frontal and temporal lobes, which showed "mass effect" and accumulated the contrast agent in the periphery as a thin rim while contrasting.

1. What is the most likely disease?
2. What diagnostic procedures should be performed to identify the disease etiology?

20. A 55-year-old woman complains of deterioration of her memory, headache, insomnia, hand tremor, and gait instability. Examination reveals static and kinetic

ataxia, pyramidal and subcortical symptoms, light dementia, and dysarthria. EEG demonstrates high-amplitude triphasic sharp waves (1—2 Hz) against the background of generalized deceleration and flattening of electrical activity. Brain CT shows extension of the subarachnoid spaces in the frontal lobes. All the mentioned manifestations constantly progress. The woman is known to like consuming meat without proper thermal processing.

1. What is the most likely disease?
2. What diagnostic procedures should be performed to confirm the diagnosis?

21. A 25 year-old woman complains of severe headache and elevated body temperature of 37.5 °C, axillary lymphadenopathy, and maculopapular skin rash. Periodically she suffers from double vision, neck stiffness, and positive Kernig's sign. Three weeks ago the woman was playing with her cat — there are scratches on the skin.

1. What is the most likely disease?
2. What diagnostic procedures should be performed to confirm the diagnosis?
3. What are treatment options?

22. A woman complains of severe headache, vomiting, and fever of 40.0 °C secondary to general weakness. Examination reveals axillary lymphadenopathy, rosy spots-like rash on the body, and hepatolienal syndrome. Neurological examination shows right-sided central paresis of the mimic muscles, right-sided hemiparesis, and positive meningeal symptoms. The craniogram demonstrates calcification foci, the toxoplasmin skin test is positive.

1 What disease does the woman suffer from?

2. What structures are impaired?

23. A patient complains of severe headache, vomiting, fainting, blurred vision, and attacks of right hand numbness lasting 3-5 minutes and repeating several times a day. Psychotic disorders are noticed. CSF analysis demonstrates lymphocytic pleocytosis and eosinophilia; the cranial X-ray reveals calcified cysts. The disease

has a remitting character with acute exacerbations.

1. What is the most likely disease?
2. What diagnostic procedures should be performed to confirm the diagnosis?
3. What are treatment options?

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Кафедра неврології

О. А. Козьолкін, І. В. Візір, М. В. Сікорська

ІНФЕКЦІЙНІ ТА ПАРАЗИТАРНІ ЗАХВОРЮВАННЯ НЕРВОВОЇ СИСТЕМИ

Навчальний посібник для студентів – іноземних громадян

VI курсу медичних факультетів спеціальності «Медицина»

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Рецензенти:

Л. В. Лукашенко - завідувачка кафедри пропедевтики, внутрішніх хвороб, променевої діагностики та променевої терапії, доктор медичних наук, професор.

О. В. Рябоконь - завідувачка кафедри інфекційних хвороб, доктор медичних наук, професор.

Автори:
О. А. Козьолкін, завідувач кафедри неврології, професор
І. В. Візір, канд. мед. наук, доцент.
М. В. Сікорська, канд. мед. наук, доцент;

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