Medical Academy named after S.I. Georgievsky
of V.I. Vernadsky CFU
Department of Neurology and Neurosurgery

Class 13

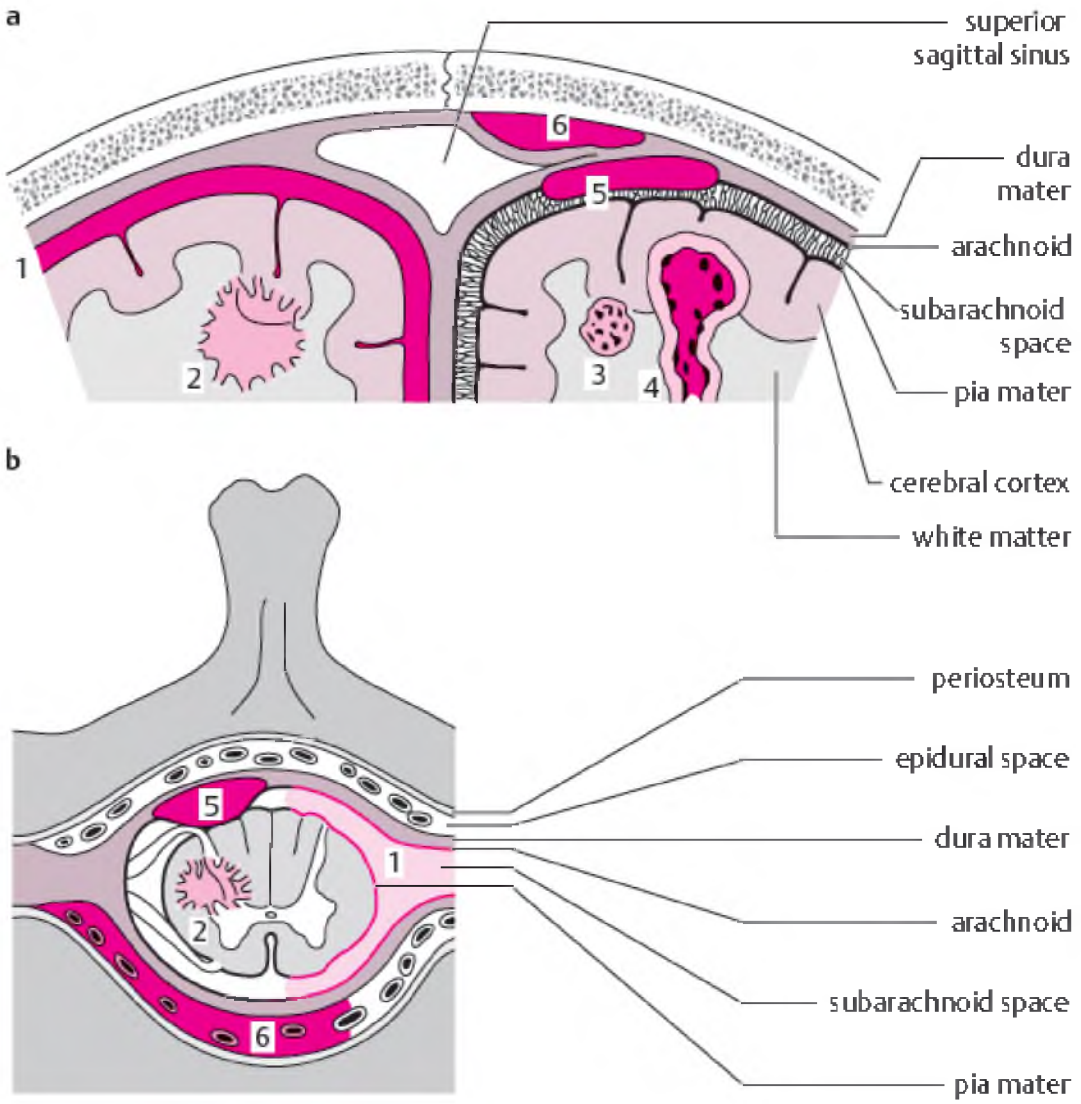
Infectious diseases of the Nervous system. Encephalitis, meningococcal and tubercular meningitis. NeuroAIDS.

1. Goals:
1.1. To study the Neurological fundamentals of the Brain Coverings.
1.2. To study the Neurological fundamentals of the Infectious Diseases of the Brain and Meninges.

2. Basic questions:
2.1. Meningitis:
2.1.1. Etiology. Pathogenesis. Classifications. Clinical Features. Diagnostic evaluation. Treatment. Prophylaxis. Prognosis.
2.2. Encephalitis:
2.2.1. Etiology. Pathogenesis. Clinical Features. Diagnostic evaluation. Treatment. Prophylaxis. Prognosis.
2.3. Brain Abscess:
2.3.1. Etiology. Pathogenesis. Clinical Features. Diagnostic evaluation. Treatment. Prophylaxis. Prognosis.

Literature:
Mark Mumenthaler, M.D., Heinrich Mattle, M.D. Fundamentals of Neurology. – 2006 – P.111-172.

The intracranial structures, like the rest of the body, can be infected by bacteria, viruses, parasites, and other microorganisms. Different organisms tend to infect either the meninges or the brain substance itself. Thus, there are two main forms of intracranial infection, **meningitis** and **encephalitis** (see the Fig. below).



	Brain:	Spinal cord:
	1=meningitis	1=arachnoiditis
	2=encephalitis	2=myelitis
brain abscess	3=cerebritis (=early stage)	
brain abscess	4=abscess (=late stage)	
	5=subdural empyema	5=spinal subdural abscess
	6=epidural abscess	6=spinal epidural abscess

Mixed forms also occur: a meningeal infection can spread to the brain (and/or spinal cord), or vice versa, causing **meningo-(myelo)encephalitis**. The latter term is only used if the patient unequivocally manifests clinical signs of *both* meningeal *and* cerebral involvement. Infectious diseases of the central nervous system can be classified, broadly speaking, into three basic clinical situations: a predominantly meningitic syndrome, which can be either acute or subacute to chronic, and a predominantly encephalitic syndrome.

Infections Mainly Involving the Meninges

General manifestations of a meningitic syndrome are:

_ headache;
_ fever (though elderly and immune-deficient patients are often afebrile);
_ nausea and vomiting due to intracranial hypertension;
_ meningism, which, in severe cases, may be evident as a spontaneous extended posture of the neck, or opisthotonus;
_ positive meningeal signs with neck extension, i. e., the Lasegue, Brudzinski, and Kernig signs.

Acute Meningitis

Acute Bacterial Meningitis

Acute bacterial meningitis is caused by bacteria that can reach the meninges by any of three routes: **hematogenous spread** (e. g., from a focus of infection in the nasopharynx), **continuous extension** (e. g., from the middle ear or paranasal sinuses), or **direct contamination** (through an open wound or CSF fistula).

The clinical onset of purulent meningitis is usually **acute or subacute** and patients very quickly become severely ill. The initiation of antibiotic therapy as rapidly as possible is essential for a good outcome.

Etiology. The organisms that most commonly cause acute, purulent meningitis are:
_ in **neonates**, *Escherichia coli*, group B streptococci, and *Listeria monocytogenes*;
_ in **children**, *Hemophilus influenzae*, pneumococci, and meningococci (*Neisseria meningitidis*);
_ in **adults**, pneumococci, meningococci, and, less commonly, staphylococci and gram-negative enterobacteria.

Clinical manifestations. The course of purulent meningitis is characterized by the meningitic signs and symptoms listed above, as well as by:
_ myalgia, back pain;
_ photophobia;
_ if the infection is mainly located over the cerebral convexity, with irritation of the underlying brain parenchyma, epileptic seizures (40 %);
_ cranial nerve deficits (10 to 20%, sometimes permanent deafness, particularly after pneumococcal infection);
_ variably severe impairment of consciousness;
_ in infection with *Neisseria meningitidis*, there may be petechial cutaneous hemorrhages and hemorrhagic necrosis of the adrenal cortex due to endotoxic shock (Waterhouse–Friderichsen syndrome).

Diagnostic evaluation. The most important and most urgent component of the diagnostic evaluation is *lumbar puncture*. Whenever acute meningitis is suspected, a lumbar puncture should be performed at once, as soon as papilledema (a sign of intracranial hypertension) has been ruled out by ophthalmoscopy. The CSF is typically turbid, with 1000 to several thousand cells/mm3 (mainly granulocytes), the protein concentration markedly elevated (positive Pandy test), and the glucose concentration diminished. CSF examination enables confirmation of the diagnosis of meningitis and, in two-thirds of patients, demonstration of bacteria by Gram stain and identification of the causative organism by CSF culture.

Treatment begins with *antibiotic therapy*, with a single drug, or multiple drugs, chosen for their effectiveness against the most likely causative organisms in the given clinical setting. Once the organism has been identified by CSF culture and its antibiotic sensitivity spectrum has
been determined, the antibiotic treatment can be tailored for maximum effectiveness against this organism.

! The antibiotic treatment of bacterial meningitis must be started immediately after the lumbar puncture, without waiting, e. g., for a CT or MRI to be performed (if these or other tests are planned). The elapsed time between the clinical presentation and the beginning of treatment is the most important prognostic factor!

Acute Viral Meningitis

A number of viruses can cause so-called **aseptic** or **lymphocytic meningitis**, which usually presents acutely (less commonly, subacutely) after a nonspecific prodromal stage with flulike or gastrointestinal symptoms.

The more common ***causative viruses*** are *enteroviruses* (polio- and Coxsackie viruses), *arboviruses*, and *HIV*; other, rarer ones include *lymphocytic choriomeningitis virus (LCV)*, *cytomegalovirus*, *type II herpesvirus*, and the *mumps*, *Epstein–Barr*, and *influenza viruses.*

The ***main clinical manifestations*** are headache, fever, meningism (often mild), and general symptoms such as fatigue and myalgia. The causative virus is identified by serologic testing. The natural course of aseptic meningitis is usually favorable, provided the brain is not involved (i. e., provided there is no encephalitic component).

Antiviral ***treatment*** is given if the causative virus is found to be one for which an effective treatment exists. Residual neurological deficits, such as deafness, are rare.

Chronic Meningitis

Chronic meningitis is caused by different organisms from the pus-forming bacteria that cause acute meningitis and therefore takes a **less acute and dramatic course**, at least initially: the **meningitic symptoms** arise gradually, often fluctuate, and, depending on the causative organism, may progressively worsen over a long period of time. **Fever** and other clinical and laboratory **signs of infection** (elevated ESR and CRP, blood count abnormalities, general symptoms such as fatigue andmyalgia) are common but may be absent. There may be variably severe neurological deficits. The spectrum of causative organisms is very wide.

Tuberculous Meningitis

Etiology. *Mycobacterium tuberculosis* bacilli reach the meninges by hematogenous spread, either directly from a primary complex (*early generalization*), or else from a focus of tuberculosis in an internal organ (*late generalization*). The site of origin may be clinically silent.

Clinical manifestations. *Meningitic symptoms* usually develop gradually. Febrile bouts and general symptoms (see above) are often but not always present. Because the infectious process typically centers on the base of the brain (so-called *basal meningitis*, in contrast to bacterial meningitis, which is typically located around the cerebral convexities), *cranial nerve palsies* are common, particularly of the nerves of eye movement and the facial n. Moreover, *arteritis of the cerebral vasculature* may result in focal brain infarction. The protein concentration in CSF is typically markedly elevated and gelatinous exudates in the subarachnoid space, including the basal cisterns, cause progressive hardening of the meninges and *malresorptive hydrocephalus.*

Diagnostic evaluation. The most important part of the evaluation is the *detection of the causative organism* in the CSF or other bodily fluids (sputum, tracheal secretions, gastric juice, urine). In the past, the detection of mycobacteria in the CSF often required weeks of culture; at present, it can be done relatively quickly with *PCR.* Occasionally, a Ziehl–Neelsen stain of the CSF will directly and immediately reveal acid-fast bacilli (mycobacteria).

Treatment generally begins with a **combination of four tuberculostatic drugs** (*isoniazid, rifampicin, pyrazinamide, and myambutol*), followed by a combination of three drugs, and then of two, for at least 12 months. Untreated tuberculous meningitis is lethal.

Other Causes of Chronic Meningitis.

A number of other organisms can rarely cause chronic meningitis, usually accompanied by variably severe encephalitis.

Fungal meningitis mainly affects immune-deficient patients, though not exclusively; the causative species include *Cryptococcus neoformans*, *Candida albicans*, and aspergilli. Further causative organisms include **protozoa** (*Toxoplasma gondii*) and **parasites** (cysticerci, echinococci). The noninfectious causes of the chronic meningitic syndrome include **sarcoidosis**, which, like tuberculous meningitis, is mainly found around the base of the brain, and **seeding of the meninges with metastatic carcinoma or sarcoma** (carcinomatous or sarcomatous meningitis).

Infections Mainly Involving the Brain

Infections with a predominantly encephalitic, rather than meningitic, syndrome typically cause **focal neurological and neuropsychological deficits** as well as a variably severe impairment of consciousness.

Encephalitis, like meningitis, can be of **viral**, **bacterial**, **fungal**, **protozoal**, or **parasitic origin**.

Prion diseases are a special category of encephalitis.

These infectious processes often involve other structures in the nervous system simultaneously with the brain (e. g., the peripheral nerves and plexuses, nerve roots, spinal cord, and meninges). In particular, the three important clinical varieties of spirochetal infection (syphilis, borreliosis, and leptospirosis) often present initially with meningitic or polyradiculitic and polyneuritic manifestations.

General signs and symptoms of an encephalitic syndrome are:

_ fever,
_ headache,
_ impairment of consciousness,
_ personality changes and neuropsychological abnormalities,
_ epileptic seizures,
_ focal neurological deficits.

Viral Encephalitis

Herpes Simplex Encephalitis

Herpes simplex encephalitis is a serious infectious condition caused by the herpes simplex virus, type I. Pathogenesis. This viral disease is characterized by hemorrhagic–necrotic inflammation of the basal portions of the frontal and temporal lobes, combined with severe cerebral edema. The inflammatory foci are found in both hemispheres, but one is usually more strongly affected than the other.

Clinical manifestations. After a *nonspecific prodromal phase* with fever, headache, and other general symptoms, the disease presents with *progressive impairment of consciousness*, *epileptic seizures* (usually of complex partial type, with or without secondary generalization, because of the temporal localization of the disease), and *focal neurological* and *neuropsychological deficits*, particularly impairment of memory and orientation. *Aphasia* and *hemiplegia* may ensue.

Diagnostic evaluation. *CSF examination* reveals up to 500 cells/mm3, mainly lymphocytes but also granulocytes; the CSF is sometimes bloody or xanthochromic. Viral DNA can be identified in the CSF by the polymerase chain reaction (PCR) in the first few days of illness and, two weeks later, IgG specific for herpes simplex virus can be identified in the CSF as well. The *EEG*, in addition to nonspecific changes, may reveal characteristic focal findings over one or both temporal lobes. The *CT scan* is usually normal at first but, within a few days, reveals temporal or frontal hypodense areas, which may contain foci of hemorrhage (see Fig. below). *MRI* may reveal corresponding signal changes even earlier.

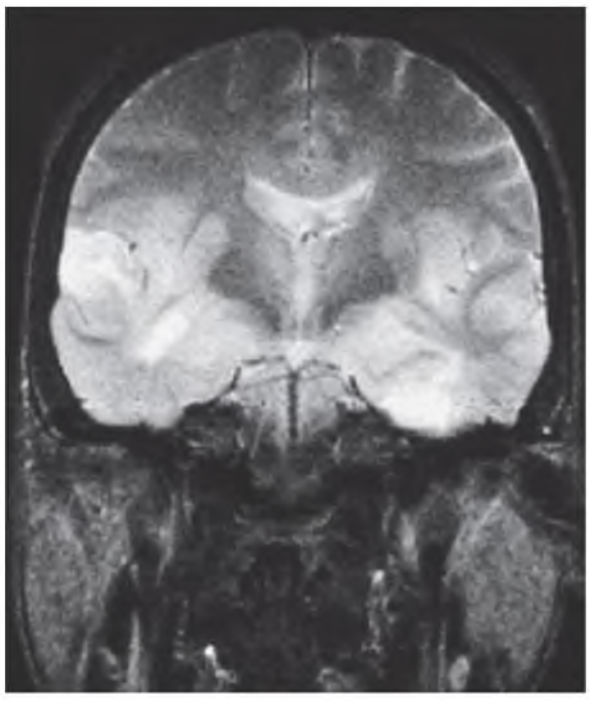


Figure: ***Herpes simplex encephalitis*** affecting both temporal lobes

Treatment. *Acyclovir* is given intravenously. *Corticosteroids* are given to combat cerebral edema and *antiepileptic drugs* to prevent seizures.

! If there is good reason to suspect herpes simplex encephalitis (progressive impairment of consciousness, aphasia, epileptic seizures [particularly of the complex partial type], an inflammatory CSF profile, focal EEG abnormalities), intravenous acyclovir therapy must be started immediately.

Early Summer Meningoencephalitis (ESME)

This disease ***is caused*** by an *arbovirus* and transmitted by tick bites. In endemic areas (e. g., Austria and southern Germany), it affects one in every 100 to 1000 tickbite victims.

After an incubation period of one to four weeks, in which there are nonspecific prodromal ***manifestations*** such as fever and flulike or gastrointestinal symptoms, about 20% of patients develop *headache*, *meningism*, and *focal neurological deficits* referable to the brain and spinal cord. *Peripheral nerve deficits* may also appear some time later.

When the patient has recovered from the acute illness, residual paresis and, less commonly, neuropsychological deficits may remain. The essential diagnostic test is the demonstration of virus-specific IgM antibodies. ESME can be effectively prevented by exposure prophylaxis (adequate clothing in endemic forest areas) and active immunization. Immune serum given within 48 hours of a tick bite is protective.

Other Types of Viral Encephalitis

Herpes zoster encephalitis is accompanied by a segmental vesicular rash in the territory of a peripheral nerve (cranial nerve). CSF examination reveals lymphocytic pleocytosis up to 200 cells/mm3. The disease may appear in particularly severe form after a generalized herpes zoster infection.

Encephalitis in Prion Diseases

Prions are infectious particles composed of protein that replicate within the body's cells even though they possess no genetic material (nucleic acids) of their own. They can arise in situ by mutation of the host's genetic material or reach the body from outside and incorporate themselves into its cells, where they replicate. Neurons in the brain that have been infected by prions may die after a latency period of years or even decades. The typical pathological findings in prion infection are vacuolization and the formation of amyloid plaques (*spongiform encephalopathy*, *SEP*). The main prion diseases are *Creutzfeldt–Jakob disease*, *kuru*, *Gerstmann–Strɔussler–Scheinker syndrome*, *familial progressive subcortical gliosis*, and *familial fatal insomnia.*

Creutzfeldt–Jakob disease, the most common prion disease in Europe and North America, is nevertheless rare, with an incidence of about one case per million individuals per year. It presents initially with *mental abnormalities*, *insomnia*, and *fatigability.* Soon, progressive *dementia* develops, along with *pyramidal tract signs*, *cerebellar signs*, *abnormalities of muscle tone*, *fasciculations*, and *myoclonus.* In about two-thirds of patients, the EEG reveals characteristic, periodic triphasic and tetraphasic theta and deltawaves. The disease progresses rapidly, leading to a decorticate state and death within months of onset. A *variant of Creutzfeldt–Jakob disease* has attracted considerable attention in the past decade, particularly in the United Kingdom, because it is contracted by eating beef derived from cows with *bovine spongiform encephalopathy* (BSE, "mad cow disease").

Intracranial Abscesses

Brain abscesses are produced by **focal infection of the brain parenchyma** leading to tissue destruction and pus formation. They can be solitary or multiple. A special form is **focal encephalitis**, in which systemic sepsis or the embolization of infectious material into the central nervous system gives rise to **multilocular**, **disseminated microabscesses**.

Brain Abscess

Etiology. Brain abscesses are caused by one or more pathogens, mainly streptococci and staphylococci and, less commonly, *Pseudomonas*, *Actinomyces*, and fungi. Like the organisms that cause bacterial meningitis, these pathogens can reach the brain through **local extension of infection** (especially mastoiditis, sinusitis, and otitis), **hematogenous dissemination** from a distant infectious focus (usually pulmonary infections or endocarditis), or **direct contamination** (open brain injury). Immunocompromised patients are at increased risk.

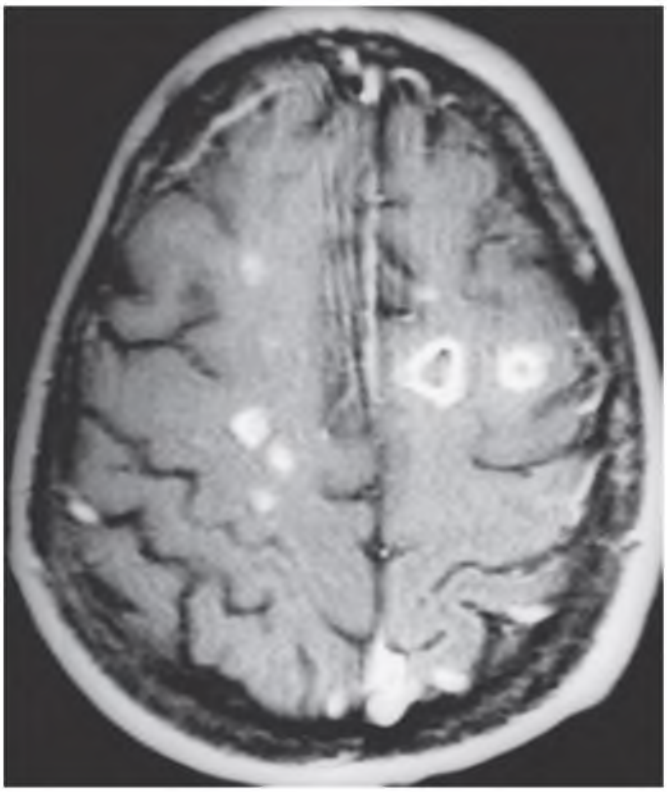


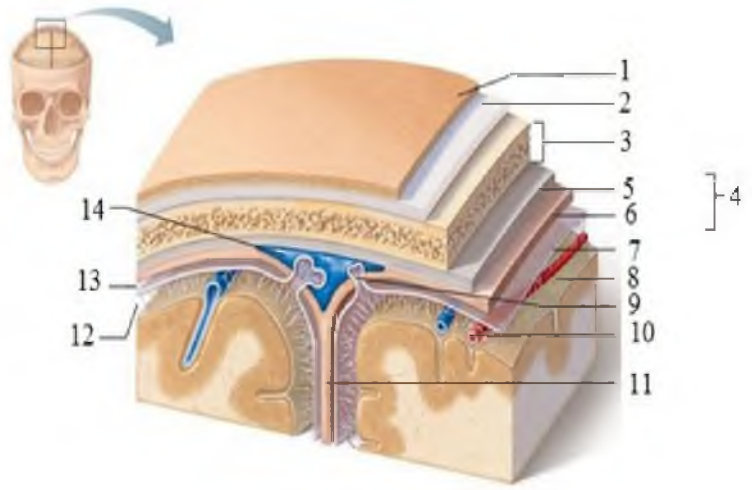
Figure: Focal encephalitis and brain abscesses

Clinical manifestations. A large **brain abscess** exerts mass effect and typically causes fever, leukocytosis, and rapidly progressive intracranial hypertension. Marked perifocal edema generally adds to the mass effect. Alternatively, there may be a **subdural empyema** between the dura mater and the arachnoid, or an **epidural abscess** between the dura mater and the inner table of the skull. These processes usually arise as a complication of sinusitis or otitis, less commonly after trauma. Fever, headache, and meningism, accompanied by neurological deficits, are their clinical hallmarks. The course of subdural empyema is often fulminant and lifethreatening, that of epidural abscess usually more protracted.

Diagnostic evaluation. The diagnosis is suspected on the basis of the *typical clinical findings* (intracranial hypertension with papilledema, impaired consciousness, sometimes hemiparesis or other focal neurological deficits), *accompanying signs of infection* (fever, elevated laboratory parameters of inflammation), and *relevant aspects of the past medical history* (such as traumatic brain injuries, known lung or heart disease, and immune suppression or diseases of the immune system). *CSF examination* may reveal inflammatory changes (predominantly granulocytic pleocytosis, elevation of total protein), and the *CT or MRI scan* shows a ring-shaped area of contrast enhancement (abscess wall) surrounding the hypodense interior of the abscess.

Treatment. *Operative removal of the abscess* is the preferred form of treatment in most patients, accompanied by *antibiotic therapy*, which is initiated before surgery and continued thereafter for at least six weeks.

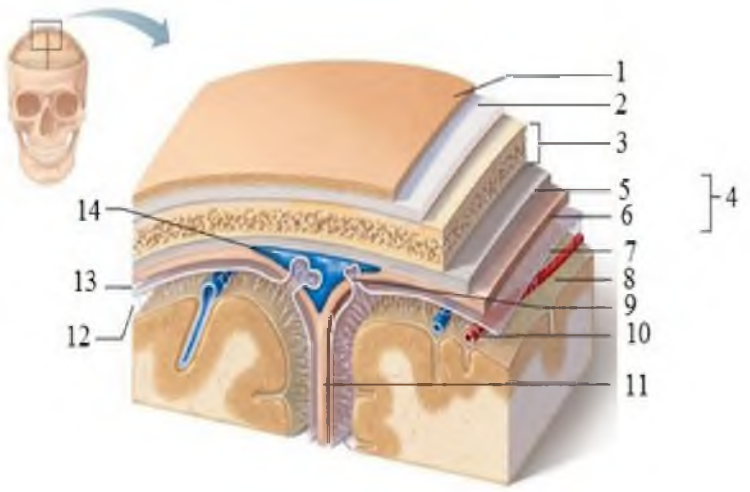
QUESTIONS FOR SELF-EDUCATION



1. Using the figure from the left indicate structure pointed with **4**:
    1. Arachnoid villus
    2. Bone of skull
    3. Pia mater
    4. Skin of scalp
    5. Periosteum
    6. Periosteal dura mater
    7. Blood vessel
    8. Dura mater
    9. Arachnoid mater
    10. Falx Cerebri
    11. Subdural space
    12. Superior sagittal sinus
    13. Subarachnoid space
    14. Meningeal dura mater

2. Using the figure from the left indicate structure pointed with **8**:
    1. Arachnoid villus
    2. Bone of skull
    3. Pia mater
    4. Skin of scalp
    5. Periosteum
    6. Periosteal dura mater
    7. Blood vessel
    8. Dura mater
    9. Arachnoid mater
    10. Falx Cerebri
    11. Subdural space
    12. Superior sagittal sinus
    13. Subarachnoid space
    14. Meningeal dura mater

3. Using the figure from the left indicate structure pointed with **12**:
    1. Arachnoid villus
    2. Bone of skull
    3. Pia mater
    4. Skin of scalp
    5. Periosteum
    6. Periosteal dura mater
    7. Blood vessel
    8. Dura mater
    9. Arachnoid mater
    10. Falx Cerebri
    11. Subdural space
    12. Superior sagittal sinus
    13. Subarachnoid space
    14. Meningeal dura mater



4. Using the figure from the left indicate structure pointed with **9**:
    1. Arachnoid villus
    2. Bone of skull
    3. Pia mater
    4. Skin of scalp
    5. Periosteum
    6. Periosteal dura mater
    7. Blood vessel
    8. Dura mater
    9. Arachnoid mater
    10. Falx Cerebri
    11. Subdural space
    12. Superior sagittal sinus
    13. Subarachnoid space
    14. Meningeal dura mater

5. Using the figure from the left indicate structure pointed with **7**:
    1. Arachnoid villus
    2. Bone of skull
    3. Pia mater
    4. Skin of scalp
    5. Periosteum
    6. Periosteal dura mater
    7. Blood vessel
    8. Dura mater
    9. Arachnoid mater
    10. Falx Cerebri
    11. Subdural space
    12. Superior sagittal sinus
    13. Subarachnoid space
    14. Meningeal dura mater

6. Indicate NORMAL CSF parameters (choose applicable):
    1. CSF glucose = ¾ % of serum glucose
    2. CSF protein = 1 g/L
    3. CSF cell count = 0-4 cells/mm^3
    4. CSF cells = neutrophils only
    5. CSF glucose = ½ % of serum glucose
    6. CSF color = transparent
    7. CSF Pandi reaction = four pluses (++++)
    8. CSF cells = neutrophils and single erythrocytes
    9. CSF protein = 0,2-0,3 g/L
    10. CSF cell count = 10-15 cells/mm^3
    11. CSF color = xanthochromic
    12. CSF glucose = 2,2-4,0 mmol/L
    13. CSF color = turbid
    14. CSF cells = lymphocytes only
    15. CSF pressure = 250 mm. of H_2O

7. Indicate CSF changes in BACTERIAL meningitis (choose applicable):
    1. CSF glucose = ¾ % of serum glucose
    2. CSF protein = 1 g/L
    3. CSF cell count = 0-4 cells/mm^3
    4. CSF glucose = ¼ % of serum glucose

5. CSF color = xanthochromic
6. CSF cell count = 8000 cells/mm^3
7. CSF color = transparent
8. CSF Pandi reaction = four pluses (++++)
9. CSF cells = neutrophils and lymphocytes
10. CSF protein = 0,2-0,3 g/L
11. CSF cell count = 10-15 cells/mm^3
12. CSF glucose = 2,2-4,0 mmol/L
13. CSF color = turbid
14. CSF cells = erythrocytes only
15. CSF pressure = 250 mm. of H_2O

8. Indicate CSF changes in VIRAL meningitis (choose applicable):
1. CSF color = xanthochromic
2. CSF protein = 0,5-0,8 g/L
3. CSF glucose = ¼ % of serum glucose
4. CSF color = transparent
5. CSF cell count = 600-800 cells/mm^3
6. CSF glucose = ¾ % of serum glucose
7. CSF cell count = 6000 cells/mm^3
8. CSF pressure = 190 mm. of H_2O
9. CSF cells = lymphocytes
10. CSF cells = neutrophils
11. CSF protein = 0,2-0,3 g/L
12. CSF cell count = 30-75 cells/mm^3
13. CSF glucose = 2,2-4,0 mmol/L
14. CSF color = turbid
15. CSF cells = erythrocytes only

9. Indicate CSF changes in TUBERCULOUS meningitis (choose applicable):
1. CSF glucose = ¾ % of serum glucose
2. CSF protein = 1 g/L
3. CSF cell count = 0-4 cells/mm^3
4. CSF pressure = 250 mm. of H_2O
5. CSF glucose = ¼ % of serum glucose
6. CSF color = xanthochromic
7. CSF cell count = 400 cells/mm^3
8. CSF cells = neutrophils
9. CSF protein = 0,2-0,3 g/L
10. CSF cells = lymphocytes
11. CSF cell count = 10-15 cells/mm^3
12. CSF glucose = 2,2-4,0 mmol/L
13. CSF color = turbid
14. CSF Pandi reaction = four pluses (++++)
15. CSF cells = erythrocytes

10. Meningitis is (choose applicable):
1. A disease caused by the inflammation of the brain and spinal cord
2. A disease caused by the inflammation of the cerebral pathways connecting the brain and spinal cord
3. A disease caused by the inflammation of the intracranial and extracranial vessels
4. A disease caused by the inflammation of the membranes covering the brain and spinal cord
5. A disease caused by the inflammation of the vascular plexuses in the lateral brain ventricles
6. A disease caused by the obstruction of the pacchionian granulations (arachnoid granulations)

11. Indicate causes of meningitis (choose applicable):
    1. Bacteria
    2. Migraine
    3. Viruses
    4. AIDS
    5. Fungi
    6. Stroke
    7. Cancer
    8. Drugs
    9. Parasites
    10. Epilepsy

12. Indicate causes of bacterial meningitis (choose applicable):
    1. Streptococcus pneumonia
    2. Herpes zoster virus
    3. Mycobacterium tuberculosis
    4. Neisseria meningitides
    5. Streptococcus aureus
    6. Hemophilus influenzae
    7. Herpes simplex virus
    8. Escherichia Coli
    9. Listeria monocytogenes
    10. Cryptococcus
    11. Streptococcus agalactiae
    12. Staphylococcus aureus

13. Indicate symptoms of meningitis (choose applicable):
    1. Ataxia
    2. Fever
    3. Hemianesthesia
    4. Tremor
    5. Headache
    6. Photophobia
    7. Peripheral paralysis
    8. Nausea
    9. Vomiting
    10. Irritability
    11. Central paralysis
    12. Poor eating
    13. Altered mental status
    14. Coordinatory disturbances
    15. Stiff neck

14. Patient is supine with hip and knees flexed at 90 degrees, examiner cannot extend knee. This is (choose applicable):
    1. Positive neck rigidity sign
    2. Positive Babinski sign
    3. Positive Brudzinski sign
    4. Positive neck stiffness sign
    5. Positive Kernig sign
    6. Positive Marinesco sign
    7. Positive Romberg sign
    8. Positive Lasegue sign
    9. Positive Lhermitte's sign
    10. Positive Wasserman sign

15. Patient is supine, when examiner flexes neck the patient involuntarily flexes hip and knees. This is (choose applicable):

1. Positive Romberg sign
2. Positive Brudzinski sign
3. Positive neck rigidity sign
4. Positive Babinski sign
5. Positive Kernig sign
6. Positive neck stiffness sign
7. Positive Marinesco sign
8. Positive Lasegue sign
9. Positive Lhermitte's sign
10. Positive Wasserman sign

16. The patient turns out to have bacterial meningitis. Which of the following is the best treatment? (choose applicable):

1. Intravenous acyclovir
2. Intravenous combination of ceftriaxone and ampicilline
3. Oral fluconazole
4. Oral combination of sulfadiazine and pyrimethamine
5. Combination of isoniazid, rifampicin, pyrazinamide and myambutol

17. Indicate complications of meningitis (choose applicable):

1. Hearing loss
2. Memory difficulty
3. Learning disabilities
4. Brain damage
5. Gait problems
6. Seizures
7. Kidney failure
8. Shock
9. Death

18. After lumbar puncture and spinal fluid examination in the patient with headache, fever, neck rigidity it has been found that cerebrospinal fluid was turbid, its pressure was increased, amount of cells was 8000 neutrophils per μl, protein – 3.3 g/L, glucose – 2.57 mmol/l, Pandy reaction +++. Based on the described data what neurological disease in the patient would be considered? (choose applicable):

1. Viral encephalitis
2. Brain abscess
3. Tuberculous meningitis
4. Cryptococcal meningitis
5. Bacterial meningitis
6. Viral meningitis
7. Acute stroke
8. Fungal meningitis
9. Aseptic meningitis
10. Chronic meningitis

19. After lumbar puncture and spinal fluid examination in the patient with headache, subfebrile temperature, neck rigidity it has been found that cerebrospinal fluid was slightly opalescent, its pressure was slightly increased, amount of cells was 600 lymphocytes per μl, protein – 2.0 g/L, glucose – 1.22 mmol/l, Pandy reaction +++. Based on the described data what neurological disease in the patient would be considered? (choose applicable):

1. Acute subarachnoid hemorrhage
2. Cryptococcal meningitis
3. Viral encephalitis
4. Brain abscess

5. Aseptic meningitis
6. Bacterial meningitis
7. Tuberculous meningitis
8. Viral meningitis
9. Secondary bacterial meningitis
10. Fungal meningitis

20. The 36-year-old man has suffered from migraine-like headaches for the previous several years. He was brought to the emergency department with severe headache, which had developed after physical workload. The patient reported about sudden onset of the headache, followed by vomiting and short-lasting loss of consciousness. Neurological examination: psychomotor agitation, neck rigidity, positive Kernig's sign, positive upper and lower Brudzinski sign, focal neurological symptoms are absent. Based on the described data what neurological disease in the patient would be considered? (choose applicable):
1. Viral encephalitis
2. Tuberculous meningitis
3. Fungal meningitis
4. Brain abscess
5. Viral meningitis
6. Subarachnoid hemorrhage
7. Cryptococcal meningitis
8. Aseptic meningitis
9. Bacterial meningitis
10. Secondary bacterial meningitis

21. The 56-year-old woman, who has been suffering from essential hypertension for the previous ten years, suddenly lost consciousness while washing floors in her apartment. The patient was brought to the emergency with the following symptoms: reddened face, tachypnea and irregular breathing, arterial blood pressure 220/120 mm/Hg, pulse rate 58 beats per minute (vibrating and rhythmic). Neurological examination: bilateral midriasis, diminished pupillary photoreactions, floating eyes symptom, significantly diminished corneal reflexes, weakness and asymmetry of the left side of the lower face, left hemiplegia, absence of deep tendon and superficial reflexes, positive Babinski sign, absence of reaction to panful stimuli, mild Kernig's sign and neck rigidity. Based on the described data what neurological disease in the patient would be considered? (choose applicable):
1. Brain abscess
2. Fungal meningitis
3. Aseptic meningitis
4. Viral encephalitis
5. Tuberculous meningitis
6. Cryptococcal meningitis
7. Viral meningitis
8. Hemorrhagic stroke
9. Bacterial meningitis
10. Secondary bacterial meningitis

22. General signs and symptoms of an encephalitic syndrome are (choose applicable):
1. Impairment of consciousness
2. Fever
3. Neck rigidity
4. Headache
5. Personality changes
6. Neuropsychological abnormalities
7. Kernig sign
8. Epileptic seizures
9. Focal neurological deficits

23. The patient turns out to have tuberculous meningitis. Which of the following is the best treatment? (choose applicable):
1. Intravenous acyclovir
2. Intravenous combination of ceftriaxone and ampicilline
3. Oral fluconazole
4. Oral combination of sulfadiazine and pyrimethamine
5. Combination of isoniazid, rifampicin, pyrazinamide and myambutol

24. Neuroimaging of the brain before attempting a lumbar puncture is advisable in cases of acute encephalitis because (choose applicable)
1. The diagnosis may be evident on the basis of magnetic resonance imaging (MRI) alone
2. Massive edema in the temporal lobe may make herniation imminent
3. The computed tomography (CT) picture may determine whether a brain biopsy should be obtained
4. Shunting of the ventricles is usually indicated, and the imaging studies are needed to direct the placement of the shunt
5. It may establish what pathology is responsible

25. Most of the organisms found in brain abscesses are (choose applicable):
1. Staphylococcal
2. Bacteroides spp.
3. Proteus spp.
4. Fungal
5. Herpes viruses
6. Cryptococcal
7. Pseudomonas spp.
8. Streptococcal
9. Mycobacterium tuberculosis
10. Neisseria meningitides

26. Encephalitis is (choose applicable):
1. A disease caused by the inflammation of the brain and spinal cord parenchyma
2. A disease caused by the inflammation of the intracranial and extracranial vessels
3. A disease caused by the inflammation of the membranes covering the brain and spinal cord
4. A disease caused by the inflammation of the vascular plexuses in the lateral brain ventricles
5. A disease caused by the obstruction of the pacchionian granulations (arachnoid granulations)

27. Indicate causes of encephalitis (choose applicable):
1. Adenoviruses
2. Streptococcus pneumonia
3. Herpes virus
4. Mycobacterium tuberculosis
5. Neisseria meningitides
6. Streptococcus aureus
7. Hemophilus influenzae
8. Mumps virus
9. Escherichia Coli
10. Listeria monocytogenes
11. Cryptococcus
12. Streptococcus agalactiae
13. Staphylococcus aureus
14. Enteroviruses
15. HIV

28. Indicate symptoms of encephalitis (choose applicable):
1. Confusion or disorientation
2. Hallucinations

3. Tremors
4. Seizures
5. Cranial nerve palsies
6. Exaggerated deep tendon reflexes and absent superficial reflexes
7. Paralysis of the extremities
8. Neck stiffness
9. Vomiting
10. Aphasia
11. Involuntary movements
12. Ataxia
13. Sensory defects
14. Amnesia
15. Dysphasia

29. Diagnostic evaluation of encephalitis includes the following (choose applicable):
1. Neurological examination
2. CT scan
3. MRI scan
4. Lumbar puncture
5. EEG
6. Serologic studies

30. Indicate complications of encephalitis (choose applicable):
1. Bronchial pneumonia
2. Urine retention
3. Urinary tract infection
4. Paralysis
5. Pressure ulcers
6. Coma
7. Epilepsy
8. Parkinsonism
9. Mental deterioration
10. Syndrome of inappropriate secretion of antidiuretic hormone (SIADH)