MINISTRY OF HEALTH OF UKRAINE
DANYLO HALYTSKYI LVIV NATIONAL MEDICAL UNIVERSITY

PEDIATRIC INFECTIOUS DISEASES DEPARTMENT

GUIDELINES

TO PRACTICAL TRAINING OF PROFILE COURSES OF CHOICE
"**OBSTETRICS AND GYNECOLOGY**" AND "**INTERNAL MEDICINE**"
FOR 6TH YEAR STUDENTS OF THE MEDICAL FACULTY
SPECIALTY
"GENERAL MEDICINE"
TOPIC:

"Differential diagnosis of meningococcal infection in children. Aseptic meningitis in children. Differential diagnosis of aseptic and purulent meningitis. Emergency states (conditions) in neuroinfections: toxic shock syndrome in meningococcal infection, brain edema, cerebral coma. Emergency treatment"

LVIV-2021

Guidelines are made according to the Study program on Pediatric infectious diseases for students of the second (Master Dergree) level of higher education in the field of knowledge 22 " Health Care " specialty 222 "General Medicine"

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Guidelines on the course of Pediatric Infectious Diseases

for students of the 6th year of General Medicine Faculty

Approved at meeting of the

Pediatric infectious diseases department

Protocol № 229, 22.06.2021.

“Approved”

Methodical Commission of Pediatric disciplines

Protocol № 3, 07.09.2021.

Guidelines to lesson for students of the 6th year

(practical classes – 6 hours)

« Differential diagnosis of meningococcal infection in children. Etiological, epidemiological, pathogenetic features, leading clinical symptoms and variants of meningococcal infection. Differential diagnosis of meningococcemia with diseases accompanied by hemorrhagic rash (hemorrhagic vasculitis, thrombocytopenic purpura, etc.).
Aseptic meningitis in children. Differential diagnosis of aseptic and purulent meningitis (primary, secondary, viral, bacterial) among themselves and with other conditions. Meningeal syndrome in the course of infectious diseases. Clinical and laboratory diagnosis of neuroinfections. Diagnosis of cerebrospinal fluid.
Emergency states (conditions) in neuroinfections: toxic shock syndrome (TSS) in meningococcal infection, brain edema, cerebral coma.»
RELEVANCE (actuality) OF THE TOPIC. Meningococcal infection is an acute infectious disease with airborne transmission of the infection caused by meningococcus. It is characterized by a variety of clinical manifestations: from carriers and nasopharyngitis to generalized forms of purulent meningitis (meningoencephalitis), meningococcemia, in severe cases with manifestations of toxic shock syndrom (TSS).
Children under 14 get sick 16 times more often than adults. Meningococcemia in children of the first two years of life is often complicated by TSS, the mortality of which reaches 80%.
Insufficient awareness of the early symptoms of TTS, clinical manifestations of meningitis in children of the first year of life, leads to untimely diagnosis, which in combination with late hospitalization and inadequate treatment can lead to adverse termination of the disease.
Enterovirus infection is a group of acute diseases caused by numerous viruses of the genus Enteroviruses from the picornavirus family and characterized by a significant variety of clinical forms, one of which is aseptic meningitis, rarely encephalitis. Aseptic meningitis is caused by viruses, some bacteria, protozoa, and some medications.

I.

Aim: to know diagnostic criterions of Meningococcal infection, meningitis in children; to perform differential diagnosis of them. to teach students on the basis of patient complaints, medical history, epidemiology, objective examination and paraclinical tests to recognize various forms of Meningococcal infection, to assess the dynamics of major clinical manifestations and laboratory tests, to help learn the

principles of treatment of patients with Meningococcal infection and hospital to study measures for the prevention of this disease.

Professional motivation: Meningitis – is a group of diseases, caused by different agents. Mortality in case of meningitis still remains high, predominantly in infants, peculiarly in purulent meningitis, caused by Pneumococci, Pseudomonas aeruginosa, Gram-negative strains, Candida. That's why it is very important to differentiate them and perform right diagnosis for adequate etiological and pathogenetical treatment.

Basic level

1. To know how to ask complaints, history of the disease and life in children with meningitis [propedeutic pediatrics, pediatric infectious diseases].
2. To perform clinical examination of the child with meningitis [propedeutic pediatrics, pediatric infectious diseases].
3. To diagnose meningitis after clinical, laboratory and instrumental examination of the child [infectious diseases, propedeutic pediatrics, microbiology, and pathophysiology].

Etiological, pathogenetical and symptomatical treatment of meningitis
[pharmacology, children infectious diseases].

II. Primary aims of the study

A student should know:

- **etiology of the disease** (morphology, antigenic structure, classification of meningococci, their pathogenic, virulent, invasive and toxigenic properties)
- **epidemiology of the disease** (source of infection, mechanism and ways of infection, susceptibility, seasonality of the disease among young children and older)
- **pathogenesis of the disease** (entrance gate of infection, causes and pathogenesis of meningococcemia, endotoxic syndrome, infectious-toxic shock, meningitis and edema-swelling of the brain)
- **classification of meningococcal infection** (bacteriocarrier, localized, generalized, rare forms; severity, course of the disease)
- **clinical picture of the disease** in typical forms, especially in children depending on age

- **complications of the disease** and their clinical picture (toxic shock syndrome, DIC syndrome, brain edema, cerebral hyper- and hypotension syndrome, ependymatitis)

- **laboratory diagnostics methods** (bacteriological, serological)

- **meningococcal infection should be differentiated** from scarlet fever, yersiniosis, hemorrhagic vasculitis, thrombocytopenic purpura, toxic influenza; serous meningitis enterovirus, mumps, tuberculosis, leptospirosis, etc. etiology; of purulent meningitis of pneumococcal, hemophilic, streptococcal, blue purulent**,** Klebsiella and other etiology

- **Patients management** with meningococcal infection (indications for hospitalization, rules and means of etiotropic, pathogenetic detoxification and dehydration, symptomatic therapy)

- **prevention measures** (increase of sanitary and hygienic culture of the population, anti-epidemic measures in the center: isolation of the source of infection, examination of contacts)

A student should be able:

- follow the basic rules of work at the patient's bedside

- collect a history of the disease

- evaluate epidemiological data

- examine the patient, identify the main symptoms of the disease (in particular, signs of damage to the central nervous system)

- to reproduce the obtained data in the medical history and substantiate the previous diagnosis

- appoint a survey plan and evaluate the results of laboratory tests

- substantiate the clinical diagnosis according to the classification of the disease

- to carry out differential diagnosis of the disease

- prescribe treatment to the patient, taking into account the form and severity of the disease, its complications, premorbid background:

a) in a hospital

b) at the prehospital stage

- explain how to organize anti-epidemic measures in the center of infection

A student should be able:

1. To follow the basic rules of work with a bed patient with Meningococcal

infection; Bacterial and viral meningitis; Encephalitis.

2. To take anamnesis with the estimation of epidemiology information (taking into account seasonality, origin of febricities, polymorphism of clinical signs of illness).

3. To examine a patient and reveal the basic clinical signs of illness.

4. To represent information of anamnesis and objective inspection in a hospital chart and formulate the preliminary diagnosis.

5. To write a plan of examination.

6. To write a clinical diagnosis (form of disease, type, severity, course of disease).

7. To prescribe the treatment taking into account age, severity of illness.

8. To write out a prescription.

9. To organize disease measures in the focus of infection (to find out the source of infection, fill an urgent report in SES, to set the quarantine, to define the circle of contact persons).

10. To write epicrisis with the estimation of illness development, results of inspection, efficiency of treatment, prognosis, recommendations for a subsequent supervision or treatment depending on the form of Meningococcal infection;Encephalitis.

III. Educational aims of the study

to form the student's responsibility to the patient: during the conversation with the patient and his relatives from the first words to try to inspire confidence; do not rush to ask intimate questions; provide information about the patient's health without causing stress reactions

ORGANIZATIONAL STRUCTURE OF THE CLASS:

Preparatory stage - 20% of working time: organization of classes, goal setting, control of the initial level of knowledge.

The main stage - 60% of working time: the formation of professional skills and abilities. At this time, the student supervises patients under the

supervision of a teacher, masters the skills of objective examination of the patient, interprets laboratory data, substantiates previous and clinical diagnoses, prescribes examination and treatment data, writes prescriptions, masters the method of taking material for laboratory research.

The final stage - 20% of working time: control and correction of the level of professional skills, summarizing, homework

MATERIALS OF METHODICAL SUPPORT OF STUDENTS 'SELF-TRAINING

(study of meningococcal infection)

№	Learning tasks	Instructions for the task
	Study	
1.	Etiology of meningococcal infection	name, morphology, antigenic structure, classification of meningococci, their pathogenic, virulent, invasive and toxigenic properties
2.	Epidemiology	source of infection, mechanism and ways of infection, susceptibility, seasonality of the disease among young and older children
3.	Pathogenesis	entrance gate of infection, causes and pathogenesis of endotoxic syndrome, infectious-toxic shock, bacteremia, development of meningococcemia, meningitis, meningoencephalitis
4.	Classification of the disease	typical, atypical forms, bacteriocarriers; severity, course
5.	Clinical symptoms of typical forms	syndromes of endotoxicosis, toxic shock syndrome, bacteremia, development of meningococcemia, meningitis, meningoencephalitis, etc.
6.	Diagnostics laboratory methods	bacteriological, serological
7.	Differential diagnosis	заповнити таблицю диференційної діагностики менінгококового менінгіту з менінгітами іншої етіології
8.	Treatment	показання до госпіталізації, правила та засоби етіотропної, патогенетичної детоксикаційної та дегідратаційної, симпто-матичної терапії
9.	Prevention	prevention of the spread of meningococcal infection; increase of sanitary and hygienic culture of the population, anti-epidemic

		measures in the center: isolation of a source of an infection, inspection of contact

IV. Interdisciplinary integration

Table 1

Subjects	To Know	To Know How
Human Anatomy	The main anatomic characteristics of CNS and respiratory tract	
Physiology	Respiratory, nervous and immune system function	To explain a variety of clinical signs and laboratory abnormalities
Pathological Physiology	Pathogenesis of disease. Pathogenesis of toxic shock syndrome. Pathogenesis of brain edema.	To explain the main symptoms and signs appearance, causes of relapses, failure of inadequate therapy
Pathological Anatomy	Pathology	To explain the causes of complications and death
Microbiology	Etiology (classification, morphologic characteristic of the pathogen, methods of revealing and identification)	To culture the organism
Pharmacology	The main antibacterial agents. Regimens of treatment. Treatment of complicated meningococcal infection. Supportive care	To administer treatment of specific infection including ancillary therapy. To write the scheme of treatment of severe meningococcal infection
Histology	Histological changes in different clinical forms of meningococcal infection	Explanation of clinical signs appearance

Propedeutics of Internal Diseases	History of disease. Patient's examination.	To gather information about patient's history and chief complaints, to distinguish those, most important for diagnosis of different clinical forms of meningococcal infection. To examine the patient, to reveal the main symptoms and signs of disease. To distinguish the set of diagnostic features of meningococcal infection. To argue the diagnosis.
Surgery	Hemorrhages, hypotonia, tachicardia, renal failure, abdominal pain, fever, soft gangrene	Differential diagnosis with surgical disorders, diagnosis of complications
Internal Diseases	Haemorrhagic syndrome, fever	To differentiate with other disorders of cardiovascular system
Neurology	Severe headache, vomiting, meningeal signs, delirium, altered consciousness	Differential diagnosis with encephalitis, stroke
Clinical immunology and allergology	Immunologic changes as a part of pathogenesis and host defenses	To explain confirmative serologic tests
Epidemiology	The routs of transmission, main	Epidemiological history

	sources of infection	
Themes integration		
Encephalitis, viral and tuberculous meningitis, common cold, haemorrhagic fevers, leptospirosis, staphylococcal sepsis	To know peculiarities of manifestations, laboratory diagnosis, treatment	To differentiate meningococcal infection with other infectious diseases with similar symptoms

V. The contents of the theme

Meningitis is a potentially fatal inflammation of the meninges membranes which encase the brain and spinal cord.

Etiology.

Meningitis is most commonly caused by an infection of bacteria, viruses, or fungi, although there can be other causes, including bleeding into the meninges, cancer, or diseases of the immune system. The origin of an infection leading to meningitis varies according to an individual's age, habits, living environment, and health status. In newborns, the most common agents of meningitis are those contracted from the mother, including Group B streptococci, *Escherichia coli*, and *Listeria monocytogenes*. Older children are more frequently infected by *Haemophilus influenzae*, *Neisseria meningitidis*, and *Streptococcus pneumoniae*, while adults are infected by *S. pneumoniae* and *N. meningitidis. N. meningitidis* is the only organism that can cause epidemics of meningitis. Epidemics of meningitis can occur in crowded conditions, such as when a child in a day care falls ill with *N. meningitidis* meningitis and exposes other children and workers to the infection.

Viral causes of meningitis include herpes simplex, mumps and measles, chicken pox, rabies, and a number of viruses which are acquired through the bite of infected mosquitoes. Non-bacterial, or inflammatory, meningitis is most often

caused by a virus, but can also be triggered by some medications. Inflammatory meningitis can also be caused by the presence of certain atypical cells in the cerebrospinal fluid (CSF). Other causes of meningitis include fungal infections, malignancies, and syphilis. Patients with AIDS (Acquired Immunodeficiency Syndrome) are more susceptible to certain infectious causes of meningitis, including certain fungal agents, as well as by the agent that causes tuberculosis.

Clinical forms of meningococcal infection:

1. Localized:

a) nasopharyngitis;

b) meningococcal carrier;

2. Generalized:

a) meningococcemia (typical, fulminant, chronic);

b) meningitis;

c) meningoencephalitis;

d) combined forms (meningitis + meningococcemia).

3. Rare forms:

a) endocarditis;

b) pneumonia;

c) arthritis;

d) iridocyclitis.

Meningococcal meningitis. The disease begins acutely with fever till 38^0-40^0C, chills, severe headache. The patients become restless, headache is increased at sound and light stimuli, at head turning; signs of hyperparesthesias are prominent. Repeated vomiting develops, which is not connected to meals and do not bring relief. Meningeal signs are positive (neck stiffness, Kernig and Brudzinski signs, in first year old children also symptom of Lessage, “suspension”), large fontanel bulging and pulsation is seen. Patient’s face is pale, sclera vessels are widened.

Cerebral spinal fluid changes are typical: by the end of the first day it becomes cloudy, milk-white, is leaking under increased pressure. Neutrophilic cytosis and increased protein contain are typical. At disease onset CSF can have signs of serous inflammation.

Combined form of meningococcal meningitis and meningococcemia is possible.

For the **fulminant form** of meningococcemia is characterized by a sudden onset with rapid increasing symptoms of general intoxication:

1. From the first hours of the disease, the temperature rises to high numbers (40-41oC), with a tendency to decrease (to subnormal).

2. Cold with severe hyperhidrosis, tremor of the extremities.

3. General hyperesthesia from the first hours of illness, agitation, convulsions, loss of consciousness, repeated vomiting.

4. In the first hours of the disease bright redness of the skin, then replaced by a sharp pallor, total cyanosis.

5. The rash occurs in the first 4-6 hours, pours quickly, abundant, hemorrhagic, large in size with deep necrosis. Multiple hemorrhages on the mucous membranes of the oropharynx, conjunctiva, genitals. Possible necrosis and dry gangrene of the phalanges of the fingers, hands, nose, ears.

6. Severe tachycardia, deafness of heart sounds, a sharp drop in blood pressure.

7. Shortness of breath, respiratory rhythm disorders.

8. Oliguria, later anuria.

9. Growing DIC syndrome.

The above symptoms are characteristic of TSS.

The most perfect and widespread in the world is the prognostic scale for meningococcemia Glasgow (1991), which should be used to identify patients in need of aggressive support due to unfavorable prognosis.

Glasgow Meningococcal Septicemia Prognostic Scale

Signs	Units
1. Systolic blood pressure (<75 mm Hg in children under 4 years), Systolic blood pressure (< 85mm Hg in children older than 4 years)	3
2. Skin-rectal temperature gradient > 3ºC	3
3. Glasgow scale score - less than 8 points or worse by 3 points or more per hour	3
4. Deterioration in the last hour	2
5. Lack of meningism	2
6.Common purpura, large ecchymoses	1
7. Deficiency of bases in arterial or capillary blood > 8.0	1
Maximum score	15

Rare forms of MI: arthritis, endocarditis, pneumonia, iridocyclitis. These forms of the disease do not have specific clinical symptoms and are mostly diagnosed during outbreaks.

Diagnosis. Clinical criteria of meningococcemia:

- sudden acute beginning with fever till 38^0-40^0C;

- prominent intoxication: general weakness, headache, muscle pain, skin paleness;

- majority of patients develop macular papule rash without specific localization several hours after disease onset. Several hours later hemorrhagic rash elements of 1-2mm till several centimeters in diameter appear on the skin of buttocks, legs, thighs and lower part of the trunk. Later necrosis appears in the center of the largest elements;

- sclera, oral mucosa, nasal and gastric bleeding can develop;

- at fulminate forms symptoms of infectious toxic shock rapidly increase, bluish hypostatic spots appear on the body.

Final diagnosis is confirmed by laboratory investigations. The latter include bacteriological, bacterioscopic, serological methods and express diagnosis (latex agglutination, immunochromatography). Materials for bacteriologic investigation are nasopharyngeal mucus, blood and cerebro-spinal fluid.

As a rule, meningococci can be cultivated on media in 50% of cases. However, if patients receive antibacterial therapy before hospitalization, the efficacy of bacteriological method is about 5%.

Microscopy of "thick blood film" with Gram's stain in patients with meningococcemia allows detect gram-negative diplococci inside neutrophils. At CSF microscopy intracellular and extracellular diplococci are found. To detect polysaccharide antigen, reaction of co-agglutination and reaction of cross-counter immunoelectrophoresis are used.

Complete blood count shows prominent leukocytosis, young neutrophilic forms till myelocytes, aneosinophilia and accelerated ESR.

At suspicion of meningitis, lumbar puncture (LP) is performed. If patient presents with hemorrhagic rash, urgent lumbar puncture is not required for diagnosis as it can delay beginning of antibiotic therapy. Lumbar puncture should also be delayed if child shows signs of shock or prominent intracranial hypertension (fontanel bulging, edema of ophthalmic nerve disk, decreased consciousness and focal neurological signs).

Changes of CSF at MI are those typical for purulent meningitis. Meningococci can be seen in neutrophils and can be cultivated in 80-90% of cases.

Meningococcus can be detected in CSF by PCR.

TREATMENT of meningococcal infection

Generalized forms before transporting:

- Ceftriaxoni (50-100 mg/kg, d/d), chloramphenicol single dose 25 mg/kg I/m (TSS);
- prednisolon 3-5 mg/kg;
- anticonvulsants: seduxen 0,3mg/kg;
- antipyretics: analgin 50% 0,1 ml/year of life or lytic suspension: novocaine 0,25% 4ml, aminazini 2,5% 1ml, pipolpheni 2,5% 1 ml (0,1 ml/kg of prepared fluid).

In the hospital: ethiothrope therapy (when toxic shock syndrome isn't suspected) penicillin G for children 0-3 months of old 400-500,000 lU/kg/day every 2 hours; 3-6 months - 300-400,000 lU/kg/day every 3 hours; >6 months - 200-300 lU/kg/day every 4 hours. Duration of therapy is 10 days. You can abolish penicillin when: temperature is normal, meningeal signs are absent, CSF is sanated (number of cells <100 in mm^3, number of polymorph nuclear cells is less then 30%). When CSF isn't sanated penicillin should be changed for cefotaxim (200 mg/kg/day in 4 doses every 6 hours), or ceftriaxon (100 mg/kg/day in 2 doses every 12 hours), or chloramphenicol (80-100 mg/kg/day in 4 doses, every 6 hours), or sulphonamides may be used.

Pathogenetical treatment includes:

1. Detoxycation with saline or water solutions 2:1 for children before 6 months (150-200 ml/kg/day); 6-12 months (120-150 ml/kg/day); 1-5 years (100-120 ml/kg/day); 5 years (75 ml/kg/day); 10 years (50 ml/kg/day). In case of meningitis amount of liquid for infants must not exceed 50 ml/kg/day, and not more then 20-30 ml/kg in one infusion. Saline solutions include: Cryoplasm, albumin, rheopolyglucin, physiological NaCl. Water solution is isotonic glucose.
2. Correction of temperature of the body;
3. Anticonvulsants;
4. Corticosteroids (prednisone) for 3-5 days: in case of meningitis, meningoencephalitis - 1-5mg/kg/day in 2 doses; in case of meningococcemia typical 5-10 mg/kg/day in 2-4 equal doses; in case of fulminant

meningococcemia - prednisone - 10-20 mg/kg/day, hydrocortisone -20-30 mg/kg/day in 4 equal doses;

5. Anticoagulants for hemorrhagic syndrome (according coagulogram).
6. Symptomatically treatment of arthritis, iridocyclitis, pneumonia, myocarditis and complications.

Discharge of the patient: clinically healthy, with normal CSF analyses; with two documented negative nasopharyngeal cultures which perform in 3 days after antibiotic therapy with 2 days interval between each other.

Dispensarization for 2 years by paediatricians, in case of meningitis, meningoencephalitis – also neurologist.

Prophylaxis: sanation of carriers by erythromycin, chloramphenicol, sulfonamides or rifampin for 3-5 days; quarantine for 10 days, looking after contacts with one bacteriological test of nasopharyngeal culture.

PEDIATRIC ASEPTIC MENINGITIS is an inflammation of the meninges caused mainly by nonbacterial organisms, specific agents, or other disease processes. Aseptic meningitis (including viral meningitis) is the most common infection of the central nervous system (CNS) in the pediatric population, occurring most frequently in children younger than 1 year. Despite advances in antimicrobial and general supportive therapies, CNS infections remain a significant cause of morbidity and mortality in children.

Etiology

Although many agents and conditions are known to be associated with pediatric aseptic meningitis, often a specific cause is not identified, because a complete diagnostic investigation is not always completed. Viruses are the most common cause, and enteroviruses (EVs) are the most frequently detected viruses. The use of molecular diagnostic techniques (eg, polymerase chain reaction [PCR] assay) has significantly increased diagnostic accuracy.

Viruses

EV is a frequent cause of febrile illnesses in children. Other viral pathogens include paramyxovirus, herpesvirus, influenza virus, rubella virus, and adenovirus. Meningitis may occur in as many as 50% of children younger than 3 months with EV infection. EV infection can occur at any time during the year but is associated with epidemics in the summer and fall.

Viruses associated with aseptic meningitis include the following:

- EV 71, EV 70, EV 75
- Polioviruses types 1, 2, and 3
- Coxsackievirus type A (23 serotypes) and type B (6 serotypes)
- Echoviruses (31 serotypes; see the image below)
- Human parechoviruses (HPeV) (6 serotypes; HPeV types 1 and 2 were previously classified as echovirus types 22 and 23 within the genus Enterovirus)
- Arbovirus (eastern, western, and Venezuelan equine encephalitis viruses; Powassan virus; California group viruses [primarily LaCrosse virus]; St. Louis encephalitis virus; West Nile virus; and Colorado tick fever)
- Mumps virus
- Herpes simplex virus (HSV) types 1 and 2
- Cytomegalovirus (CMV)
- Epstein-Barr virus (EBV)
- Human herpesvirus type 6 (HHV6) and type 7 (HHV7)
- Varicella-zoster virus (VZV)
- Adenovirus types 3 and 7
- Human immunodeficiency virus (HIV)
- Lymphocytic choriomeningitis (associated with contact with guinea pigs, hamsters, and pet mice)
- Rhinovirus
- Measles virus

- Rubella virus
- Influenza A and B viruses, including H1N1
- Parainfluenza virus
- Parvovirus B19
- Rotavirus
- Coronavirus
- Variola virus
- Flavavirus

Physical Examination

Physical examination findings vary widely, depending on the patient's age and the organism or condition responsible for the meningitis. The younger the child, the less specific the signs: In a young infant, findings that definitely point to meningitis are rare, but as the child grows older, the physical examination becomes more reliable. Because clinical signs are unreliable, particularly in the younger patient, they should not be the only factors considered when deciding on investigations and lumbar puncture.

The infant may be febrile or hypothermic. Lymphadenopathy may be present. Bulging of the fontanel, diastasis of the sutures, and nuchal rigidity point to meningitis but are usually late findings. Examination should specifically exclude a nonblanching petechial rash, other signs of bacterial meningitis, and features suggestive of a noninfectious etiology.

Neurologic examination includes evaluating for signs of meningism (eg, headache, photophobia, neck stiffness, and positive Kernig or Brudzinski sign) and focal or generalized neurologic signs. Focal neurologic signs may be present in as many as 15% of patients and are associated with a worse prognosis.

A definitive diagnosis of meningitis requires examination of CSF via lumbar puncture. Lumbar puncture should not be carried out in the presence of any contraindications (listed below). The presence or absence of classic meningeal signs and symptoms should not be used as the sole criterion for referring patients for further diagnostic testing.

Contraindications to lumbar puncture, per the Meningitis Research Foundation, are as follows:

- Clinical or radiological signs of raised intracranial pressure
- Shock
- After convulsions until stabilized
- Coagulation abnormalities
- Clotting study results (if obtained) outside the normal range
- Platelet count below 100 x 10^9/L
- On anticoagulant therapy
- Local superficial infection at lumbar puncture site
- Respiratory insufficiency
- Focal neurological signs (will need brain imaging before considering the safety of lumbar puncture)

Perform delayed lumbar puncture in children with suspected meningitis when contraindications are no longer present.

Laboratory Studies

The following studies are indicated in patients with suspected aseptic meningitis:

- White blood cell (WBC) count
- C-reactive protein (CRP)
- Procalcitonin (PCT) – PCT has been suggested as a potentially useful predictor for distinguishing between bacterial and aseptic meningitis but is not yet widely available
- Blood glucose (to compare with CSF glucose)
- Blood culture to exclude bacterial meningitis
- Viral culture of throat swab, nasopharyngeal aspirate, and stool sample
- Serology – Save serum for paired convalescent sample comparison of serology at 2-3 weeks following acute illness

Other Tests

CT and MRI

When the clinical presentation of aseptic meningitis is typical, imaging studies (ie, early computed tomography [CT] or magnetic resonance imaging [MRI]) are rarely required for initial management, unless (1) other pathology must be ruled out before lumbar puncture or (2) focal neurologic signs are present. Imaging may be useful to check for abscesses, subdural effusions, empyema, or hydrocephalus. Normal CT findings do not rule out increased intracranial pressure (ICP).

EEG

Electroencephalography (EEG) may be considered if atypical febrile seizures have occurred. A neuroimaging study is required for complicated cases, including children with meningoencephalitis.

ENTEROVIRUSES, a group of single-stranded sense RNA viruses, are commonly encountered infections, especially in infants and children. They are responsible for a myriad of clinical syndromes, including hand-foot-and-mouth (HFM) disease, herpangina, myocarditis, aseptic meningitis, and pleurodynia. Patients with enterovirus infections may present with symptoms as benign as an uncomplicated summer cold or as threatening as encephalitis, myocarditis, or neonatal sepsis. Enteroviral infections annually result in a large number of physician and emergency department visits.

NONPOLIO ENTEROVIRUSES cause an astronomical number of infections per year. More than 90% of enteroviral infections are either asymptomatic or cause a nonspecific febrile illness. A wide range of symptoms is observed, but most cases include fever, a viral prodrome, and gastrointestinal symptoms.

- Patients with nonspecific febrile illness, the most common form of enteroviral infection, present with a sudden onset of fever, temperature ranging from 38.5-40°C. Accompanying symptoms include general upper respiratory and GI complaints. Clinical indicators include a flulike syndrome consisting of malaise, myalgias, sore throat, headache, conjunctivitis, nausea, emesis, and

diarrhea. Genitourinary manifestations such as orchitis and epididymitis are possible. Symptoms generally last 3-7 days and are caused by all enteroviral subtypes.

- Herpangina occurs in children aged 3-10 years. These patients report painful vesicles on the posterior pharynx and tonsils. These lesions are associated with fever, sore throat, odynophagia, and other viral symptoms. Mothers may notice a decreased oral intake by the child due to the painful ulcers. The causative agent is most commonly coxsackievirus group A and, sometimes, coxsackievirus group B. Herpangina is self-limited, and symptoms last 3-7 days.
- Hand-foot-and-mouth disease is a vesicular eruption in the oropharynx, palms, soles, and interdigits of toddlers and school-aged children. The oral vesicles are not usually painful. Patients often present after 1-2 days of fever and have a characteristic viral exanthem. Lesions are more common on the dorsal surfaces of the hands and feet than in other locations. The most common causative agent is coxsackievirus group A, serotype 16, but strains of enterovirus 71 circulating in East Asia are currently causing outbreaks of hand-foot-and-mouth disease (HFMD) that are associated with a serious rhombencephalitis, with significant mortality.
- Atypical HFM disease was recently reported around the globe and is caused by coxsackievirus A6. It is characterized by a relatively paucity of oral lesions, but a striking bullous eruption on the extremities. Children with eczema may be more affected and "eczema coxsackium" was coined as far back as 1968 for this condition. Postinfectious loss of the nails is reported frequently.
- Viral exanthems, a frequent cause of emergency department visits, manifest as rubelliform or roseolalike rashes that occur in the summer months. These exanthems occur in children younger than 5 years and have a benign 3-day to 5-day course. The responsible agents are usually echoviruses.

- Patients with aseptic meningitis have symptoms that mimic the initial symptoms of nonspecific febrile illnesses, but, as aseptic meningitis progresses, patients report a headache, stiff neck, and photophobia. A nonspecific rash can accompany these symptoms, raising the question of meningococcemia. The clinical course of aseptic meningitis is self-limited and resolves in 1-2 weeks.
    - The coxsackievirus group B and echoviruses are responsible for 80-90% cases in which a causative organism of aseptic meningitis is identified.
    - Neurotropic strains, such as enterovirus 71, can be responsible for more aggressive cases of CNS infections. Ninety percent of some cohorts with enterovirus 71 infection also had rhombencephalitis. This can lead to neurogenic pulmonary edema and has an overall fatality rate of 14%. Early signs of severe infection include myoclonus and sleep disturbance. Fever that lasts longer than 3 days duration, high fevers (>38.5C), and lethargy are predictors of CNS involvement.
- Patients with myocarditis or pericarditis report chest pain, fatigue, and dyspnea on exertion. These symptoms can progress to dysrhythmia and heart failure. The most common cause of cardiac involvement is coxsackievirus group B5 infection, but echoviruses are also etiologies of infection.
- Pleurodynia (Bornholm disease, devil's grippe) is an uncommon epidemic that causes severe muscular pains in the chest and abdomen. These sharp pains worsen with breathing or coughing and are associated with profuse sweating. Spasmodic muscular pains last 15-30 minutes in older children and adolescents. The condition can mimic serious surgical conditions and can cause periodic episodes of respiratory difficulty. These symptoms are accompanied by fever, headache, anorexia, nausea, and emesis. Symptoms last for 2 days. Coxsackieviruses B3 and B5 infect the intercostals muscles, causing these frightening but rare outbreaks.
- Neonates with nonpolio enterovirus infections are at a high risk of developing a sepsislike condition, including meningoencephalitis, myocarditis, and

hepatitis. Presenting symptoms include poor feeding, lethargy, fever, irritability, hypoperfusion, and jaundice. Differentiating these infections on clinical grounds from bacterial sepsis is impossible. Infants younger than 10 days are unable to mount a significant immune response and are at a higher risk of a serious infection from echoviruses and coxsackie group B viruses. A history of a mother who had a febrile illness with GI symptoms around the time of birth is often reported; this acute presentation results in exposure to viral shedding without significant transplacental transfer of maternal antibodies.

- Poliovirus infections are divided into 4 groups of clinical syndromes: asymptomatic, abortive, nonparalytic, and paralytic.
    - Most infections (90-95%) are asymptomatic.
    - Abortive poliomyelitis involves a nonspecific febrile illness that spares the CNS and spontaneously resolves after a few days. Temperature is not higher than 103°F. Patients report a minor febrile upper respiratory infection, such as cough and sore throat, and gastrointestinal infection with nausea and diarrhea.
    - Patients with nonparalytic poliomyelitis (aseptic meningitis) present in the same manner as patients with abortive poliovirus, but nonparalytic poliomyelitis progresses to aseptic meningitis. During the initial flulike illness, patients report stiffness in the posterior neck muscles, limbs, and trunk. This minor viremia is followed by nuchal and spinal rigidity, the hallmark of nonparalytic polio.
    - Paralytic poliomyelitis starts with a nonspecific febrile illness and muscle weakness that resolves after 2-3 days but is followed by a sudden onset of asymmetric flaccid paralysis. Pain, nuchal rigidity, and hypertonia are indicators of brainstem, spinal ganglia, and posterior column involvement. Bulbar poliomyelitis involves the speech and central cardiorespiratory centers of the brain stem and can cause death because of cessation of cardiac and respiratory activity.

Physical Examination

Nonspecific febrile illness can include normal findings on physical examination or can include an erythematous pharynx, mild conjunctivitis, and cervical lymphadenopathy.

- Patients with herpangina present with punctate macules that progress to vesicles that eventually ulcerate. Usually, 3-6 erythematous vesicles about 1-2 mm in size are found on the posterior pharynx, anterior tonsils, and soft palate. The oropharynx may be erythematous, but no exudates are present.
- Patients with HFM disease present with less painful or painless vesicles that may ulcerate on the buccal mucosa and tongue; the less significant pain differentiates the vesicles of HFM disease from the posterior pharyngeal vesicles of herpangina. See the image below.

  Erosions on the base of the tongue.

- In addition to the oral findings, an exanthem of vesicles appears on the palms, soles, and intertriginous digits of the hands and feet. These vesicles heal by resorption of fluid and do not crust over.
- See the list below:
  - Occasionally, nonvesicular eruptions are present on the buttocks, proximal extremities, and genitalia. The truncal area is not usually involved, differentiating HFM disease from varicella infections.
  - The absence of gingival erythema, high fevers, and lack of significant cervical lymphadenopathy aid in distinguishing HFM disease from herpetic gingivostomatitis.

    Calf blisters from coxsackievirus A6 as seen in atypical hand-foot-mouth disease. Courtesy of Elsevier (Feder HM Jr, Bennett N, Modlin JF. Atypical hand, foot, and mouth disease: a vesiculobullous eruption caused by Coxsackie virus A6. Lancet Infect Dis. Jan 2014;14(1):83-6).
- Viral exanthems appear as a pink, maculopapular, blanching rash that can mimic rubella and roseola. This rash is less commonly vesicular, urticarial,

and petechial. Unlike rubella, no significant adenopathy is present. Similar to roseola, it may appear following the cessation of fever.

- Atypical HFM disease tends to present with bullous lesions on the extremities and may be more severe over areas of preexisting eczema. The lesions are not limited to the hands and feet and may be seen on the arms and legs.
- Aseptic meningitis reveals physical findings consistent with meningeal irritation, including nuchal rigidity, a bulging fontanel, and, possibly, positive Kernig and Brudzinski signs in children older than 1 year. The accompanying rash is often nonspecific but can occasionally be petechial.
- Myocarditis and pericarditis symptoms depend on the severity of the disease. The physician should look for signs of congestive heart failure. Patients with pericarditis may have an auscultatory friction rub, Hamman crunch, and poor perfusion.
- Patients with pleurodynia (Bornholm disease) may present with respiratory distress or in a shocklike state. Patients may localize pain in the abdomen and may have tender abdominal muscular walls. A pleural friction rub may be auscultated during the muscular spasm.
- Patients with acute hemorrhagic conjunctivitis present with subconjunctival hemorrhage, erythema, lacrimation, chemosis, preauricular lymphadenopathy, and serous eye discharge. Some of these cases may progress to a bacterial conjunctivitis with purulent ocular discharge.
- Neonatal infections cause the infant to be irritable, lethargic, and inconsolable by the mother. The infection can progress to signs and symptoms that reflect hypoperfusion, such as cool mottled skin, delayed capillary refill, and ashen gray color.
- Polioviruses should be differentiated into their subtypes.
    - Abortive (nonspecific febrile) illness appears as a general viral upper respiratory and GI infection. Cough, coryza, and pharyngeal exudates are common.

- Patients with nonparalytic (aseptic meningitis) illness have a nonspecific viral picture, but the physician should recognize symptoms of meningeal irritation. Increase or decrease of the superficial and deep tendon reflex usually occurs prior to onset of weakness. If these reflexes are decreased, the physician should be wary of impending weakness and paralysis. As with all types of polio, sensory examination findings remain intact.
- The paralytic form is similar to the nonparalytic with the additional classic finding of asymmetric flaccid paralysis. Proximal muscle groups are usually more affected than the distal musculature. Deep tendon reflexes are decreased or absent, and sensory findings are unchanged. Associated symptoms include hypertonia, respiratory and cardiac arrhythmias, and blood pressure and vasomotor changes. Observe for symptoms of respiratory distress, including difficulty speaking, nasal flaring, tachypnea, and immobility of accessory muscles of respiration. Impending respiratory failure may rapidly occur.

Laboratory Studies

See the list below:

- The diagnosis of enteroviral infection is most often based on the clinician's assessment of the patient in conjunction with seasonal outbreaks, known exposure risks, geographic locations, and age groups. Ancillary laboratory test results aid the physician in supportive care of the patient and eliminate other potentially harmful and treatable bacterial illnesses. Diagnostic testing plays a role in enteroviral infections. As newer methods have demonstrated increased sensitivities, determining viral etiologies of aseptic meningitis and neonatal sepsis has resulted in improved patient care.
- Cell culture, serology, and polymerase chain reaction (PCR) laboratory testing can diagnostically isolate enteroviral infections. Enteroviruses are found in stool, the pharynx, blood, and cerebral spinal fluid (CSF). Blood cultures and serology are of questionable use because the viral levels may be undetectable by the time symptoms have appeared. Pharyngeal viral levels

remain present from 2 days to 2 weeks after the infection. Stool isolation of enteroviruses is not specific to acute infections because viral stool shedding persists for as long as 3 months after the infection.

- Historically, the criterion standard of isolation has been cell cultures; however, clinical evidence is proving PCR tests to be both more sensitive and more efficient. Tissue cultures take approximately 3-8 days to grow the enterovirus, and the identification of the subtype requires even more time. Overall, low cell culture sensitivity rates of 65-75% have been repeatedly demonstrated in enteroviral meningitis.
- Another method, serologic testing, uses multiple titers to identify a pattern of rising antibody levels over a 2-week to 4-week period. A single level of enteroviral antibodies can be present in a healthy patient; therefore, monitoring the serology to identify a 4-fold increase in levels is needed. Identifying the specific subtype and monitoring the antibody levels is labor intensive. Furthermore, waiting for periods of 2-4 weeks for tissue results is not useful in improving patient care.
- In contrast, the reverse transcriptase PCR testing is designed to detect a common genetic area in the enteroviral subtypes. The results are available in 24 hours, making detection more sensitive (95%), more specific (97%), and more time efficient. Both Chonmaitree et al in 1982 and Singer et al in 1980 demonstrated the positive outcomes of viral detection in aseptic meningitis, yielding shortened hospital stay and antibiotic course.
    - Recent studies have demonstrated the efficacy and increased sensitivity of using the PCR technique to isolate CSF enterovirus.
    - PCR testing may also play a pivotal role in identifying epidemiological outbreaks of infections.
    - In 1997, Ahmed et al demonstrated 100% sensitivity and 90% specificity using PCR CSF assays in conjunction with viral cultures to detect enteroviral meningitis in infants younger than 3 months.

- Poliomyelitis can be isolated from stool, nasopharyngeal mucosa, and CSF. Stool specimens have the greatest yield for polio. Antibody serology titers demonstrate a 4-fold rise and must be acquired at early onset of illness. If positive, samples must be sent to the CDC.
- Ancillary laboratory tests may also be helpful in treating patients. CBC count results vary, demonstrating a WBC count within the reference range or demonstrating a mild elevation of WBCs with neutrophilia or leukocytosis.
- A basic chemistry panel is only useful in patients with extreme lethargy or dehydration and is used to eliminate possible diagnosis of electrolyte imbalances.
- Erythrocyte sedimentation rate is a nonspecific test, and the results should be elevated in any inflammatory process, including enteroviral infections.
- Urinalysis is a part of the sepsis workup in neonates and young children to eliminate bacterial infections. Also, blood and urine cultures should be obtained.
- Measure cardiac enzymes.

TREATMENT Unfortunately, no specific antiviral medication or treatment is available for an enteroviral infection. The best care is provided through supportive measures. Fluid hydration and antipyretics are the mainstays of care for a viral syndrome.

- In patients with severe illness, if a bacterial infection is suspected, antibiotics are administered at the physician's discretion. Test results, such as polymerase chain reaction (PCR) test results from cerebrospinal fluid (CSF) samples, require 24 hours to return, and a positive result does not necessarily eliminate a bacterial infection. Thus, the use of cultures is important.
- Corticosteroids have been proposed to have a beneficial effect on myocarditis, but no significant improvement has been demonstrated. Furthermore, because of deleterious side effects, steroids are not recommended for treatment.

- Intravenous immune globulin (IVIG) has been suggested to be beneficial in the outcome of myocarditis because of immunoglobulin G (IgG) and T-cell modulation. Other indications include possible efficacy in infections in newborns and patients with agammaglobulinemia.
- The best medical care involves continued efforts for worldwide poliovirus vaccination.

POLIOMYELITIS is an acute infectious disease caused by poliovirus from subgroup of enteroviruses and is characterized by damage of spinal gray matter with development of flaccid paralyses.

Etiology. Poliovirus belongs to picornavirus group, enterovirus genus; there are 3 independent types: I (Brungild), II (Lansing) *и* III (Leon). Type I is seen most often. There are strains which circulate in population (so called wild strains) and virulent strains of vaccine origin, which sometimes appear after vaccination with oral poliovaccines. It is stable in environment (it can survive till 100 days in water and till 6 months in feces), resistant to freezing and drying. It is not destroyed by digestive enzymes or antibiotics. The virus is inactivated by boiling, by ultraviolet radiation and by disinfecting solutions.

Epidemiology. Human is the only source of infection. The virus is excreted into outer environment with saliva and feces. Virus excretion begins on the 4^{th} day after contact and continues for 2-3 days from oropharynx and 4-7 weeks from feces.

Main mechanism of virus transmission is fecal-oral, which is due to length of viral excretion with feces and its high concentration there. Massive excretion of poliovirus with feces into environment creates possibility for its spread through water, food, hands and by flies. In waste waters the virus can survive during the whole year. There are cases of poliovirus isolation from tap water at inadequate sanitary conditions.

Human infection by poliovirus leads to development of typical disease in only one case per 100-1000, that's why the main role in infection spread is played by healthy viral carriers. Children under one year of age are predominantly infected

with poliovirus and are spreading it. In moderate climate countries poliomyelitis cases are registered mostly in summer and autumn months, in tropical countries morbidity is annually distributed.

Clinical manifestations. Incubational period of poliomyelitis is on average 7-14 days but can be prolonged till 35 days. Poliovirus infections caused by wild strains of polioviruses can have several disease forms. Those include asymptomatic course, which occurs in 90-95% of poliovirus infections, abortive poliomyelitis, non-paralytic poliomyelitis (meningeal form) and paralytic poliomyelitis. Clinical presentations of paralytic poliomyelitis, caused by wild or vaccinated strains, are similar; cases of abortive or not-paralytic forms after vaccine-associated poliomyelitis are not described.

There are such clinical forms of poliomyelitis:

Abortive form of poliomyelitis.

Non-paralytic forms of poliomyelitis.

Paralytic form.

Bulbar poliomyelitis.

Polioencephalitis

Diagnosis. ***Clinical criteria:***

Spinal form:

- Incubational period is 4 to 30 days;
- Acute beginning with general infectious syndrome: fever, headache, pain in back, neck and extremities, running nose, dyspepsia (vomiting, diarrhea). Meningeal symptoms and stretch symptoms can be seen.
- Fever can be two-waved.
- Acute flaccid paralysis develops from several hours till 1-5 days from the disease onset:

- more often proximal parts of extremities are involved;
- pareses and paralyses have asymmetrical “mosaic” spread;
- muscle tone is decreased;
- legs, hands and trunk muscles are damaged;

- tendon reflexes are decreased;
- sensitivity is preserved;
- pelvic disturbances and pyramid signs are absent;
- rapid development of muscle atrophy: 2-3 weeks with subsequent progression;
- short period of paralyses appearance: 2-4 weeks.

Due to WHO definition, poliomyelitis is the case of acute disease with acute flaccid paralyses (AFP) and poliovirus isolation.

Materials for virusological investigation are blood, CSF, washing from nasopharynx, feces. According to WHO recommendations, for laboratory confirmation of the disease it is necessary to perform isolation and identification of poliovirus from child's feces and to perform verification between wild and post-vaccinate strains. Feces are taken for analysis in two portions during 24-48 hours after suspicion of poliomyelitis.

According to results of serological investigations, the diagnostic is 4-fold antibody titer increase in neutralization and hemagglutination reactions.

TREATMENT of poliomielitis. *Regimen* is strict bed rest. The sick child should lie on wooden board in physiological body position for contractures prophylaxis, changing body positions every 2 hours. Parentheral manipulations should be minimal, providing calmness and quietness in the room, comfortable temperature regimen. Permanent control of breathing and swallowing should be done.

Pathogenic and symptomatic therapy:

1. Pain relievers, antipyretic drugs (paracetamol, ibuprofen);
2. Diuretic drugs (furosemide, acetazolamide);
3. In severe cases – glucocorticosteroids 1 mg/kg/day (on prednisone) during 3-4 weeks (during period of paralysis increase);
4. Patients with severe spinal and bulbar forms should be treated in intensive care units.

At paralytic forms, when formation of paralysis is completed (4-6 weeks of the disease), complex rehabilitation treatment is performed, later – periodic therapeutic spa treatment. Rehabilitation physical activity, massage, physiotherapy are important therapies in recovery period. Surgical and orthopedic help is required for patients with consequences of paralytic poliomyelitis.

Prophylaxis. Specific planned prophylaxis of poliomyelitis can be done by two types of vaccines, inactivated (IPV) and oral poliomyelitis vaccine (OPV). IPV can be used as monovaccine or be included into combined vaccines.

After usage of live poliomyelitis vaccine development of vaccine-associated poliomyelitis is possible.

Patients with suspicion on poliomyelitis should be immediately isolated. Patient isolation continues not less than 4-6 weeks from disease onset. In infection nidus current and final disinfection are done with usage of disinfecting solutions. Contact people are followed under quarantine till 21 days after sick person isolation. They are followed by pediatrician (every day) and neurologist (once). All the children under 5 years of age, who were not vaccinated against poliomyelitis or were vaccinated in incomplete schedule, are urgently vaccinated by live poliomyelitis vaccine.

VI. Planning of the lesson

Table 2

	The main stages of a lesson, their contents	**The methods of control**	**Methodical equipment**	**Time in % from total time of a lesson**
10-20 %				
1	Organization stage			
2	Purposes of a		Relevance of the	1-3min

	lesson		Theme. Tutorial goals of a lesson	
3	Basic knowledge and skills control	Control questions	The list of control questions	10-15 min
	1.Etiology, epidemiology, classification of disease	Test-control (first grade)	Tests of the first level	
	2. Manifestations in connection with pathogenesis	Methods of the second grade: Individual questioning in oral and writing form. Standard task's solution. Second grade test-control	Questions Clinical cases (tests of the second grade) Theory tasks for writing answers. Second grade tests	
	3. Treatment	Methods of the third grade: 1. Solution of complicated tasks. 2.Third grade test-control	Third grade questions and tasks Third grade tests	
	4. Prevention			
70-80 %				
1	Professional skills formation	Patients with studied disease and		

		similar diseases, patient's histories, medical cases.	
	To master the skills of: a) Diagnosis b)Laboratory confirmation c) Treatment	Laboratory data of the patients, antibacterial drugs and drugs for supportive care	
	Independent work with patients	Patients, patient's histories, medical cases.	
	Differential diagnosis	Drawing schemes of pathogenesis and clinical course of disease; making up a differential diagnosis table and list of prescriptions for intensive care.	
10 %			
	Teacher's control, recommendations, the task for the next lesson		10-15 min

Students' self- study program.

1. Objectives for students' self-studies.

You should prepare for the practical class using the existing textbook and lectures. Special attention should be paid to the following:

Differential diagnosis of meningitis

Table 3

Signs	**Meningismus**	**Viral meningitis**	**Tuberculosis meningitis**	**Purulent bacterial meningitis**	**Subarachnoid hemorrhage**
Color, transparence	Colorless, transparent	Colorless, transparent or opalescent	Colorless, xanthochromic or opalescent	White-yellow or green, muddy	bloody, after settling – xanthochromic
Pressure (mm. H_2O), Flow out speed (drops per 1 minute)	below 180-200 50-80	200-300 60-90	250-500 60-90	250-500 jet, sometimes rare drops	250-400 > 70 or jet
Cytosis (in 1 mkl.)	2-12	20-800	200-700	500-1000 and more	It is hard to count in the first days, from 5-7 day 15-120
Cytogram Lymphocytes, %	80-85 15-20	80-100 0-20	40-60 20-50	0-30 30-100	from 5-7 day lymphocyt

Neutrophils, %					es prevail
Protein, g/l	0.16-0.33	0.33-1.0	1.0-3.3	0.66-16.0	0.66-16.0
Sedimentation tests (Pandy)	–	+(++)	+++(++++)	+++(++++)	+++
Dissociations	Absent	cellular-protein on the low level (from 8-10 day – protein-cellular)	protein-cellular	cellular-protein on the high level	–
Fibrin pellicle	–	- in 3-5 %	Often rough in 30-40 %	Often as a sediment	Rare
Glucose, mmol/l	2.2-3.3	2.2-3.3	For 2-3 weeks 1.0-2.0	Normal or slightly less then normal	normal

Self-study

To table 3

Signs	**Meningismus**	**Viral meningitis**	**Tuberculosis meningitis**	**Purulent bacterial meningitis**	**Subarachnoid hemorrhage**
Color, transparence					
Pressure (mm. H_2O), Flow out speed (drops per 1					

minute)					
Cytosis (in 1 mkl.)					
Cytogram Lymphocytes, % Neutrophils, %					
Protein, g/l					
Sedimentation tests (Pandy)					
Dissociations					
Fibrin pellicle					
Glucose, mmol/l					

Tasks and assignments for self-assessment

Choose the correct answer / statement:

1. To the boy, 4 years old, was put the diagnosis of meningococcal infection (generalized form). The child is treated in infectious department. What generalized form is the most often one?

A. Meningitis.

B. Meningococcemia.

C. Combined form (Meningococcemia + meningitis).

D. Meningoencephalitis.

E. Encephalitis.

2. In entrance department of the infectious diseases hospital to the child, 5 years old, physician put diagnosis of meningococcal infection, meningococcemia. Indicate the start dose of the penicillin for meningococcemia treatment.

A. 100 thous. IU/kg/day.

B. 200-300 thous. IU/kg/day.

C. 500 thous. IU/kg/day.

D. 800 thous. IU/kg/day.

E. 1 mln. IU/kg/day.

3. In a child, 6 years old, who has Chickenpox, on the 6 day of the disease has increased body temperature, appeared the headache, vomiting. During examination stiff neck was fined. Meningitis was diagnosed. What change in spinal fluid is the most probable?

A. Lymphocyte pleocytosis.

B. Neutrophyl pleocytosis.

C. Protein more than 1 g/l.

D. Normocytosis.

E. Glucose level is increased.

4. In a child, 9 years old, who has Chickenpox, on 7th day of the disease again has increased body temperature to 39.2º C, has appeared headache, vomiting, existed the shaky gait, declaimed speech. During examination was noted remaining elements of the rash (crusts), nuchal rigidity; the child is falling in Romberg pose. What complication is possible?

A. Polyneuropathy.

B. Meningitis.

C. Meningoencephalitis.

D. Encephalomyelitis.

E.Ventriculitis.

5. In a boy, 7 years old, who has mumps, has increased body temperature to 39º C, appeared: headache, sleepiness, vomiting, nuchal rigidity. The child was hospitalized in infectious department. What changes in spinal fluid are probable?

A. Decreased liquor pressure.

B. Expressed neutrophyl pleocytosis.

C. Moderate neutrophyl pleocytosis.

D. Moderate lymphocyte pleocytosis.

E. Expressed lymphocyte pleocytosis.

Answers for the self-control:

Tests: 1-C. 2-B. 3-A. 4-B.5-D.

Tasks

Case 1

The 3 year old man presented with 5-day fever 39 C, headache, vomiting, painful chewing, painful swelling of the neck, dryness of mouth. On the physical examination: submaxillary swelling beneath the mandible just anterior to the angle of the jaw, spreading to cheeks, is painful while palpation, skin is not hyperemic; ear lobe was pushed to upwards, dry lips and tongue, positive meningeal signs. CSF: cells 600 mm3, lymphocytes 86%, protein 1,2 g/L, glucose 2,3 mmol/L.

What is the diagnosis? What are the hallmark of the disease? Which the laboratory confirmative tests should be performed?

Case 2

A 3-year-old male admitted to the emergency department with a 1-day history of fever, headache, and myalgia. On examination, he is found to be lethargic and hypotensive. He has signs of meningeal irritation. A petechial and

purpural rash is spreading rapidly on his trunk, face, and extremities, hyperemic pharynx. He has received all recommended immunizations.

What is the initial diagnosis? Which laboratory tests are informative and the most likely finding on Gram stain of the CSF can be revealed? What is possible outcome of disease?

Case 3

A 15 year female with severe headache for 30 hr followed by fever, nausea, vomiting, muscle and joint pain, disorientation, hemorrhagic rash. On physical examination: BP 90/55 mm Hg, PR 125, T 39,4^{0}C, lethargy, hemorrhagic petechiae and purpura on the hands and legs; positive Kernig signs, stiff neck; lung auscultation showed no evidence of pathology. Chest film with no consolidation. Laboratory tests revealed platelets 140 x10^6. Lumbar puncture: CSF neutral in colour with 22,000 WBC (75% PMN's), protein 2.4 g/L, and sugar 0.72 μmol/L. Gram stain showed few gram-negative diplococci.

What is the clinical diagnosis in this patient? What complication occurred? Administer the appropriate treatment according to this diagnosis.

Case 4

A 14 year girl was admitted with fever, severe headache, vomiting for one day duration. She was initially placed on oral antibiotic for throat infection. Epidemiologic history: no known contact. Physical findings on admission revealed all meningeal symptoms, red coloration of pharynx, pale skin without rash, BP 120/80, P 125, T 38,8^{0}C. Lumbar puncture revealed white colored fluid with 2.560 WBC (84% PMN's), protein 1.2 g/hr, sugar 1,5 μmol/L. Culture – growth negative Gram diplococci.

What is the diagnosis? Which more laboratory test is beneficial for diagnosis and its expected results? Administer the treatment.

Student's practical activities:

I. Curation of patients with meningites in children infectious department.

1. Ask complaints, anamnesis and life history.
2. Examine the patients, find clinical features of meningitis.
3. Prescribe laboratory investigations to prove the diagnose.

II. To perform the diagnosis:

1. Make previous diagnose due to complaints, disease history, epidemiological anamnesis, clinical objective features.
2. Make complete diagnose due to previous diagnose, laboratory dates, differential diagnosis.

III. Provide the treatment (diet, medicine) depending on patient's age, severity of the disease.

IV. Prescribe measures in the focus of infection, specific prevention of the disease.

V. Clinical analyzing of the case.

Students must know:

1. Diagnostic features of different meningitis in children.
2. Differential diagnosis of meningitis in children.
3. Main treatment (etiological, pathogenetical, symptomatical) of meningitis, neurotoxicosis, hyperthermia, seizures in children.
4. Prevention of meningitis in children.

Student should be able to

1. Find diagnostic clinical criterions of meningitis during examination of patients.
2. To perform urgent prehospital and hospital treatment in case of meningitis, neurotoxicosis, hyperthermia, seizures.
3. To prescribe measures in the focus of infection.

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