

Ministry of Health of the Republic of Belarus
Educational Institution
“Gomel State Medical University”

Department of Pathological Anatomy

Author:

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METHODICAL GUIDELINES
for conducting a practical class
in the academic discipline “Pathological Anatomy”
for students
of the 3rd year, Faculty of International Students (instruction in English),
enrolled in the specialty
7-07-0911-01 “General Medicine”

Topic: “Infectious pathology: pediatric infections”

Duration: 3 hours

Approved at the meeting of the Department of Pathological Anatomy
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EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, classification, etiology, pathogenesis, morphology, clinical presentation, complications, and outcomes of common pediatric infections. To analyze exanthematous (rash-associated) viral infections: measles (rubeola) – paramyxovirus, Koplik spots (oral mucosa), maculopapular rash, giant cell pneumonia (Warthin-Finkeldey giant cells), complications (otitis media, pneumonia, encephalitis, subacute sclerosing panencephalitis – SSPE); rubella (German measles) – togavirus, maculopapular rash, postauricular and suboccipital lymphadenopathy, congenital rubella syndrome (heart defects, cataracts, deafness, patent ductus arteriosus, pulmonary artery stenosis); varicella (chickenpox) – VZV, vesicular rash in different stages ("dew drops on a rose petal"), pneumonia, encephalitis, Reye syndrome (with aspirin); erythema infectiosum (fifth disease) – parvovirus B19, "slapped cheek" rash, aplastic crisis (in hemolytic anemias), hydrops fetalis; roseola infantum (sixth disease/exanthem subitum) – human herpesvirus 6 (HHV-6) and 7, high fever followed by rash. To analyze bacterial infections in children: pertussis (whooping cough) – Bordetella pertussis, catarrhal, paroxysmal, and convalescent stages, lymphocytosis, pneumonia, bronchiolitis, intraepithelial lymphocytes; diphtheria – Corynebacterium diphtheriae, pseudomembranous pharyngitis (fibrinous membrane), myocarditis, neuropathy; scarlet fever – Streptococcus pyogenes (group A strep), "strawberry tongue," "sandpaper" rash, pharyngitis, rheumatic fever, poststreptococcal glomerulonephritis; meningococcemia – Neisseria meningitidis, Waterhouse-Friderichsen syndrome (bilateral adrenal hemorrhage), purpura fulminans, meningitis. To analyze congenital (perinatal) infections: TORCH complex (Toxoplasma, Other – syphilis, varicella, parvovirus B19, HIV, Rubella, Cytomegalovirus, Herpes simplex). To describe morphological features: cytomegalic inclusion disease (CMV – "owl's eye" inclusions, microcephaly, periventricular calcifications), congenital rubella (patent ductus arteriosus, pulmonary artery stenosis, cataracts, deafness, microcephaly), congenital toxoplasmosis (intracranial calcifications, hydrocephalus, chorioretinitis), congenital syphilis (osteochondritis, Hutchinson teeth, saddle nose, saber shins). To discuss complications: dehydration, sepsis, respiratory failure, encephalitis, myocarditis, sudden infant death syndrome (SIDS) associations.

Instructional Goal:

- Within the framework of the educational process for this academic discipline, the student must acquire not only theoretical knowledge, practical skills and abilities in the specialty but also develop their personal potential. They should cultivate qualities of responsibility and patriotism, be prepared to actively participate in the economic, socio-cultural, and public life of the country, recognize the social significance of their future professional activity,

understand the norms of medical ethics and deontology, and learn to observe academic and labor discipline. In the course of studying the educational material, students should realize the importance of maintaining a healthy lifestyle and, as an example in the future, when performing professional duties, set an example for those around them and their patients.

Objectives: Upon completion of the practical session, the student must:

Know:

- The etiology, pathogenesis, and morphology of measles, rubella, varicella, and exanthematous viral infections;
- The etiology, pathogenesis, and morphology of pertussis, diphtheria, scarlet fever, and meningococemia;
- The TORCH complex of congenital infections and their morphological manifestations;
- The complications and outcomes of pediatric infections;
- The role of vaccination in prevention.

Be able to:

- Identify Warthin-Finkeldey giant cells in measles;
- Recognize pseudomembranous pharyngitis in diphtheria;
- Diagnose congenital infections based on morphological findings (intracranial calcifications, chorioretinitis, etc.);
- Diagnose pathological processes and diseases based on the description of macroscopic and microscopic changes in organs and tissues of the body.

Master:

- Basic techniques for working with a microscope;
- Skills in clinical-anatomical analysis;
- The fundamentals of synthetic generalization of morphological diagnostic signs of diseases and their correct interpretation in cause-and-effect relationships.

Motivation for Mastering the Topic:

Pediatric infections remain a leading cause of morbidity and mortality in children worldwide, particularly in developing countries. Many of these infections are vaccine-preventable, yet outbreaks continue to occur due to inadequate vaccination coverage. Understanding the morphological features of these diseases is essential for pediatricians, infectious disease specialists, pathologists, and public health professionals for accurate diagnosis, treatment, and implementation of preventive measures. Congenital infections can cause devastating lifelong disabilities, making early recognition critical.

QUESTIONS

1. Meningococcal infection. Definition. Etiology. Classification. Morphology. Complications and outcomes. Causes of death.

2. Diphtheria. Definition. Etiology. Morphology. Differential diagnosis of true and false croup. Complications and outcomes.

3. Scarlet fever. Definition. Etiology. Morphology. Complications of the first and second periods of the course of the disease.

4. Measles. Definition. Etiology. Morphology. Differences between chickenpox and measles. Complications and outcomes.

COURSE OF THE SESSION

Diphtheria is an acute infectious disease primarily affecting the throat and respiratory tract. It is caused by the bacterium **Corynebacterium diphtheriae**, also known as the Loeffler bacillus.

Corynebacterium diphtheriae, a gram-positive, non-spore-forming, non-motile, slightly curved bacterium, is responsible for diphtheria. It is transmitted through airborne transmission or direct contact.

Following transmission, the bacteria **enter the body through mucous membranes**, including the throat, larynx, genitalia, conjunctiva, stomach, or occasionally through wounds or intact skin. **Exotoxins** produced by *C. diphtheriae* **result in superficial coagulative necrosis of the epithelium**, widening of blood vessels, and exudate production containing large amounts of coarse dispersed proteins, forming a characteristic **fibrinous membrane**. This membrane is the hallmark feature of diphtheria. In severe cases, the toxin can cause damage to various systems including the cardiovascular, adrenal, peripheral nervous, and excretory systems.

Diphtheria typically has an **incubation period of 3-10 days**, with symptoms appearing over 2-3 weeks. The disease can manifest in various forms:

1. **Localized Form:** Involving only the tonsils without extending beyond their boundaries.

2. **Widespread Form:** Involving the mucous membrane of the palate and tongue.

3. **Toxic Form:** Characterized by edema of the mucous membranes of the throat, pharynx, and neck tissue, with cellular infiltrates and focal necrosis of muscles. The hypertoxic form is associated with severe intoxication and rapid progression.

4. **Hemorrhagic Form:** Characterized by hemorrhagic infiltration of the membrane and multiple hemorrhages in the skin, nose, throat, gums, and gastrointestinal tract. This is a rare but severe complication.

Morphology of diphtheria

Inflammation can spread from the throat to the nasal mucosa, larynx, trachea, and bronchi.

The regional lymph nodes increase in size due to hyperplasia of the lymphoid tissue, congestion, and edema, with focal necrosis at the center of follicles.

In the heart, there can be dystrophic and necrotic changes leading to toxic myocarditis and acute heart failure.

Peripheral nervous system damage can lead to multiple toxic neuritis and degenerative changes in the myelin sheath, with late cardiac paralysis possible.

Adrenal glands may show hyperemia, hemorrhages, and necrosis of some cells, while kidneys may exhibit tubular necrosis.

In the respiratory tract, complications such as croupous inflammation, descending croup, fibrinous pneumonia, asphyxia, pressure sores, and secondary viral-bacterial pneumonia may occur.

Meningococcal infection is an acute infectious disease caused by the gram-negative diplococcus **Neisseria meningitidis**, also known as meningococcus.

It primarily affects the central nervous system and can manifest in various forms, including localized and generalized presentations.

Neisseria meningitidis is the causative agent of meningococcal infection. There are four serogroups: A, B, C, and D, with serogroup A having the greatest epidemic importance.

Meningococci are primarily located **inside leukocytes and produce endotoxins**.

The source of infection is a sick person or carrier, and transmission occurs via airborne droplets.

Children under five years old are most susceptible, while newborns are less likely to be affected. Intrauterine infection is also possible.

Entry portals for meningococcal infection are the upper respiratory tract. Meningococcal carriage and acute nasopharyngitis are **localized forms**, while fulminant meningococcemia, typical meningococcemia, meningitis, and meningoencephalitis are **generalized forms**.

In localized forms, **meningococcal carriage** is observed in 2-5% of healthy children, and meningococcal nasopharyngitis develops in 10-30% of infected individuals. Hematogenous generalization occurs in 0.1-1% of cases. Morphological changes in fulminant meningococcemia include endotoxin shock with a pronounced thrombohemorrhagic syndrome, leading to plasma leakage, hyperemia, stasis, fibrin thrombi, fibrinoid necrosis of vessel walls, multiple hemorrhages, and necroses in various organs. Skin hemorrhages, such as petechial rash, are characteristic. Other effects may include adrenal medulla hemorrhages and necrosis (Waterhouse-Friderichsen syndrome), myocardial hemorrhages and necrosis, and cerebral edema.

In **meningitis** and **meningoencephalitis**, purulent exudate appears and can spread to the spinal cord. Ependymitis and, rarely, purulent otitis media may develop. Neglected cases can lead to subdural empyema and hydrocephalus.

Meningococcal infection

Clinical manifestations vary based on the form of the disease but may include fever, headache, neck stiffness, altered mental status, petechial rash, and signs of shock. Fulminant meningococcemia can progress rapidly with symptoms of shock and multi-organ failure.

Morphological changes in fulminant meningococcemia include thrombohemorrhagic syndrome with plasma leakage, hyperemia, stasis, fibrin thrombi, and necrosis in various organs.

Skin hemorrhages, adrenal medulla necrosis, myocardial necrosis, and cerebral edema may occur.

In meningitis and meningoencephalitis, purulent exudate can spread to the spinal cord, with potential complications including subdural empyema and hydrocephalus.

Additionally, necrotic changes and inflammatory infiltrates may be observed in affected tissues.

Scarlet fever is an acute infectious disease primarily affecting children aged 3 to 10 years, caused by a **beta-hemolytic toxigenic group A streptococcus**.

It is characterized by fever, sore throat, and a **distinctive rash** known as scarletina.

The disease is caused by group A streptococcus, specifically strains that produce **erythrogenic exotoxins**.

Infection occurs through airborne and contact transmission, with an **incubation period of 2-7 days**.

Children aged **3 to 10 years** are more susceptible.

The **mucous membrane of the pharynx and throat** serves as the entry gate for scarlet fever. Local changes include the formation of the **primary scarlatinous complex**, consisting of primary scarlatinous affection (angina) and regional lymphadenitis. The **tonsils become enlarged, swollen, and hyperemic**, with **necrotic foci appearing in the crypts**. Streptococcal colonies deepen into the crypts, leading to necrosis of the crypt epithelium. Regional lymph nodes may exhibit hyperemia, necrosis, and myeloid metaplasia.

In moderate and severe cases, scarlet fever progresses through **toxic, toxico-septic, and septic** phases. **Toxicemia** manifests as a distinctive rash, dystrophic changes, interstitial inflammation in parenchymal organs, and acute brain swelling with circulatory disturbances. The **scarlatinous rash** is small, bright red, and quickly spreads to the face, neck, torso, and limbs. Peeling, especially on the palms and soles, follows the rash's disappearance. The septic phase may develop at the end of the first week, characterized by streptococcal spread through lymphatic and hematogenous pathways, leading to complications such as lymphangitis, lymphadenitis, and thrombophlebitis.

Clinical manifestations include fever, sore throat, enlarged tonsils, and a distinctive rash. The rash appears on the 1st-2nd day, spreading to the face, neck, torso, and limbs, sparing the nasolabial triangle. Peeling, especially on the palms and soles, follows rash resolution. Complications of the first period include peritonsillar abscess, cervical cellulitis, otitis, sinusitis, suppurative osteomyelitis of the temporal bone, brain abscess, and purulent meningitis. Allergic complications, such as vasculitis, verrucous endocarditis, synovitis, myocarditis, and glomerulonephritis, may develop in the second period.

The **scarlatinous rash** microscopically has hyperemic foci, perivascular infiltrates, and coagulation necrosis of the epidermis.

Microscopically, scarlet fever is characterized by **necrotic foci in the tonsillar crypts**, perivascular infiltrates in the skin, and coagulation necrosis of the epidermis. Complications may involve abscess formation, cellulitis, osteomyelitis, and allergic reactions affecting various organs.

Glomerulonephritis in the second period may lead to **nephrosclerosis**.

Measles is an acute highly contagious infectious disease caused by the **RNA virus** of the paramyxovirus family, specifically the measles virus.

It is characterized by fever, respiratory symptoms, and a distinctive rash.

The disease is caused by the measles virus, which is **transmitted via airborne droplets**.

Measles is more common in **children under 5 years of age**.

The average **incubation period is 10 days**, and clinical symptoms typically last for 2-3 weeks.

The measles virus enters the blood **through the mucous membranes** of the nasopharynx and conjunctiva, showing a particular affinity for the central nervous system (CNS), respiratory tract, and digestive tract. The disease begins with catarrhal **conjunctivitis, stomatitis, pharyngitis**, and laryngotracheobronchitis. Superficial necrosis of mucous membranes, especially in the larynx, may occur. **Common respiratory complications** include endo-, meso-, and peribronchitis, bronchiolitis, and giant cell interstitial pneumonia.

From the 5th-6th day of the disease, there is epithelial metaplasia into a multilayered squamous with keratinization, spreading from the larynx to the terminal bronchioles. Histological examination reveals hyperplasia of reticular cells in lymph nodes and the formation of giant multinuclear cells in tonsils and the appendix. **Belsky-Filatov-Koplik's spots**, whitish foci on the mucous membrane of the cheeks, are characteristic. A maculopapular rash appears on the skin, starting behind the ears, then spreading to the nose, neck, upper chest, trunk, upper limbs, and finally lower limbs over successive days.

Clinical manifestations include fever, respiratory symptoms, conjunctivitis, pharyngitis, and the characteristic rash.

Complications can include catarrhal colitis, otitis media, aseptic meningitis, encephalitis, myelitis, and subacute sclerosing panencephalitis.

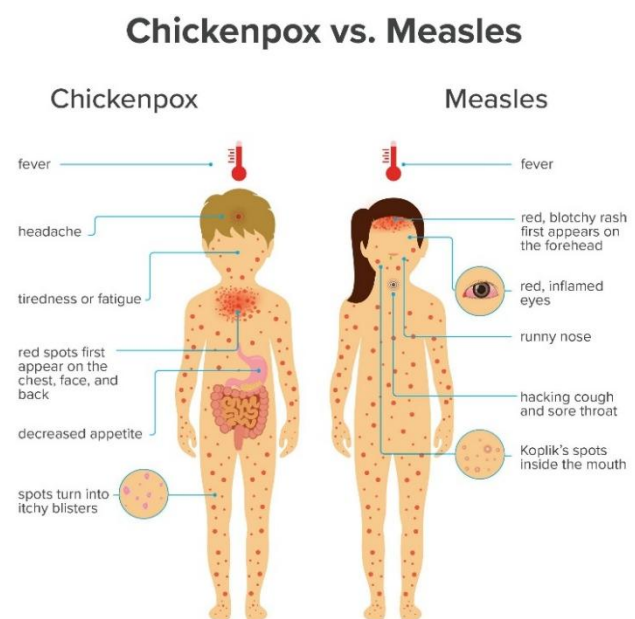
Fatalities can occur due to secondary viral and bacterial infections, especially bacterial pneumonia.

Histologically, measles presents with focal dystrophic changes in the epithelium, including focal necrosis, edema, and moderate lymphohistiocytic infiltration around blood vessels, sebaceous glands, and hair follicles.

Parakeratosis and sloughing of affected epithelium lead to flaky peeling.

In the lungs, there is congestion, edema, and focal necrosis of the epithelium, with subsequent metaplasia.

The rash on the skin exhibits characteristic stages of eruption, reflecting the progression of the disease.



INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

1. Pathomorphology : textbook / ed. by I.V. Sorokina, V.D. Markovskiy, D.I. Halata. - 2nd ed. - Kyiv : AUS Medicine Publishing, 2020. - 327 p., [10] col. inserts : tab.
2. Klatt, E. C. Robbins and Cotran Atlas of Pathology : [with Student Consult Online Access] / Edward C. Klatt. - third edition. - Philadelphia : Elsevier, 2015. - 587 p. : il.
3. Robbins Basic pathology / [ed. by] Vinay Kumar, Abul K. Abbas, Jon C. Aster. - 9th ed. - [Philadelphia] : Elsevier ; Saunders. - 910 p. : ill., scheme, tab.

ADDITIONAL LITERATURE

1. Sobotta. Atlas of human anatomy. [V. 1]. General anatomy and musculoskeletal system / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 400 p. : ill., col. ill.
2. Sobotta. Atlas of human anatomy. [V. 2]. Internal organs / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 259 p. : ill., col. ill.
3. Sobotta. Atlas of human anatomy. [V. 3]. Head, neck, and neuroanatomy / ed. by F. Paulsen, J. Waschke. - 15th ed. - München : Elsevier. Urban & Fischer, 2011. - 370 p. : ill., col. ill.
4. Weir & Abrahams' imaging atlas of human anatomy / ed.: J. D. Spratt [and oth.] ; consultant eds.: J. Weir, P. H. Abrahams. – 5th ed. - [S.l.] : Elsevier, 2017. - xvi, 263 p. : ill.

NORMATIVE LEGAL ACTS AND DOCUMENTS

1. Order of the Ministry of Health of the Republic of Belarus No. 111 of 06/01/1993 "On further improvement of the pathological service of the Republic of Belarus"

Access mode: <http://patan.by/menyu/administrativnyie-proczeduryi.html> - The date fccess: 20.03.2022.

ELECTRONIC DATABASES

1. Physician's consultant. Electronic Medical Library = Consultant of the doctor. Electronic medical library [Electronic resource] / Publishing group "GEOTAR-Media", LLC "IPUZ". – Access mode: <http://www.rosmedlib.ru/> . – Access date: 03/15/2022.
2. Springer Link [Electronic resource] / Springer International Publishing AG. – Access mode: <https://link.springer.com>. – Date of access: 03/15/2022.