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

Department of Pathological Anatomy

Author:
Grigorii V. Tishchenko, Associate Professor of the Department of Pathological Anatomy, Candidate of Medical Sciences (Ph.D. in Medicine)

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: “Immunopathological processes. Hypersensitivity reactions”

Duration: 3 hours

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EDUCATIONAL AND INSTRUCTIONAL GOALS, OBJECTIVES, AND MOTIVATION FOR MASTERING THE TOPIC

Educational Goal:

- To study the definition, causes of development, and morphological changes in the central and peripheral organs of immunogenesis during antigenic stimulation and immune deficiency. To focus on the pathology of the thymus gland, emphasizing its significance for the organism. To analyze the mechanisms of hypersensitivity reactions, particularly highlighting the morphological manifestations of acute immune inflammation. To examine the processes of autoimmunization, identifying initiating, contributing, and predisposing factors. To become familiar with the concept of "immunological tolerance" and identify the group of autoimmune diseases. To review the classification, causes of development, and clinical-morphological manifestations of immunodeficiency syndromes. To study the etiopathogenesis of AIDS, emphasizing the stages of disease development and focusing on opportunistic infections.

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 definition of immunopathological processes and list them;
- The characteristics of changes in the immunocompetent system during antigenic stimulation and immune deficiency;
- The characteristics of hypersensitivity reactions;
- The definition of autoimmunization and autoimmune diseases, name them, and provide their morphological characteristics;
- The definition of immunodeficiency syndromes, name them, and provide their morphological characteristics.

Be able to:

- Identify major general pathological processes and diseases using histological specimens under light microscopy;
- 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:

The immune system, which normally participates in maintaining homeostasis, can also be a source of pathological conditions resulting from an excessive reaction or insufficient response to aggression. Immunopathology is a branch of medicine that studies the processes and diseases arising from immune conflict and disturbances of immunological homeostasis. The study of changes in peripheral and central organs of immunogenesis, mechanisms of hypersensitivity reactions and autoimmunization, as well as the analysis of the causes and clinical-morphological manifestations of primary and secondary immunodeficiency syndromes, will be necessary for the comprehensive assessment of these pathologies during clinical disciplines and in the practical activity of a physician for clinical-anatomical analysis.

QUESTIONS

1. Type 1 hypersensitivity reaction. Pathophysiology. Etiology. Morphology. Clinical examples. Outcomes.
2. Type 2 hypersensitivity reaction. Pathophysiology. Etiology. Morphology. Clinical examples. Outcomes.
3. Type 3 hypersensitivity reaction. Pathophysiology. Etiology. Morphology. Clinical examples. Outcomes.
4. Type 4 hypersensitivity reaction. Pathophysiology. Etiology. Morphology. Clinical examples. Outcomes.
5. Autoimmune diseases. Definition. Classification. Etiology. Morphology. Clinical examples. Outcomes.
6. Immunodeficiency disorders. Definition. Classification. Etiology. Clinical examples.
7. Acquired immunodeficiency syndrome (AIDS). Definition. Pathophysiology. Etiology. Morphology. Outcomes. Opportunistic infections associated with HIV.

COURSE OF THE SESSION

Lymph nodes are essential components of the lymphatic system, playing a crucial role in immune surveillance and response.

A typical lymph node exhibits a well-defined and continuous **fibrous capsule** that surrounds its structure. The capsule consists of **collagenous and elastic fibers**, serving as a protective barrier to external threats. Lymphatic vessels enter and exit the node at its hilum, where the capsule is less dense, facilitating lymphatic flow.

Within the lymph node, the **cortex** represents the outer region, featuring **lymphoid follicles**. These follicles are comprised of **B-cell-rich germinal centers**, where the generation of high-affinity antibodies occurs during the humoral immune response. The **paracortex**, nestled between the cortex and the medulla, is home to T-cells, forming the **T-zone**. The T-zone is an area critical for cell-mediated immunity and contains high endothelial venules (HEVs) that facilitate lymphocyte trafficking.

Lymph nodes

Moving inward, the **medulla**, or the innermost part of the lymph node, is composed of medullary cords and sinuses. Medullary cords contain **plasma cells**, **macrophages**, and **reticular cells**, contributing to the production of antibodies and the clearance of pathogens.

Sinuses within the medulla allow lymph to circulate through the node, facilitating the **removal of antigens and the interaction of immune cells**.

A fundamental aspect of lymph node histology is the **distinction between** the B-cell-rich **cortex** and the T-cell-dominant **paracortex**. The B-zones in the cortex are marked by a high concentration of follicular dendritic cells, which support B-cell activation and antibody production. Meanwhile, the T-zones in the paracortex contain a network of dendritic cells and antigen-presenting cells, promoting T-cell maturation and their interaction with antigen.

Thymus

The thymus is a primary **lymphoid organ** situated in the **anterior mediastinum** of the thoracic cavity, and its normal histology plays a pivotal role in the **development and maturation of T-lymphocytes**. Structurally, the thymus is bilobed, encapsulated by a thin fibrous capsule that sends trabeculae into its parenchyma, dividing it into lobules. These lobules consist of an **outer cortex** and an **inner medulla**, each with distinct cellular compositions and functions.

Within the thymic cortex, a characteristic feature is the presence of numerous **thymic (Hassall's) corpuscles**. These structures consist of **epithelial cells** and **keratinized debris** and are primarily found in the medulla. The cortex is primarily composed of developing T-lymphocytes, or thymocytes, which undergo a rigorous process of maturation and selection. The cortex houses cortical thymic epithelial cells (**cTECs**), which play a critical role in presenting self-antigens to developing thymocytes to ensure self-tolerance. The cortex is also rich in thymic dendritic cells, macrophages, and supporting stromal cells.

The **thymic medulla**, in contrast, contains fewer thymocytes but is characterized by its role in central tolerance. It is the site where self-reactive

thymocytes are deleted to prevent autoimmune reactions. Medullary thymic epithelial cells (**mTECs**) express a wider range of self-antigens, contributing to the negative selection of self-reactive thymocytes. In addition, regulatory T-cells, or **Tregs**, are generated in the medulla and play a critical role in maintaining peripheral immune tolerance.

The histological characteristics of the thymus are essential to its function as a primary lymphoid organ, facilitating the maturation and selection of T-lymphocytes necessary for the adaptive immune response. Understanding the normal histology of the thymus is integral to recognizing deviations indicative of thymic disorders and autoimmune diseases, which can have significant clinical implications.

Thymus involution

Thymus involution is a physiological process characterized by a **progressive reduction in the size and functional activity of the thymus gland** over time. This phenomenon is a normal part of aging and occurs predominantly during the post-pubertal and adult stages of life. Thymus involution is a consequence of the gradual replacement of functional thymic tissue with **adipose (fatty) tissue** and a decline in thymopoiesis, the production of new T-lymphocytes, or T-cells.

Thymus involution is a complex process influenced by multiple factors, including hormonal changes and immune system interactions.

Thymus involution, when viewed as a **pathological process**, represents an abnormal or accelerated decline in the structural and functional integrity of the thymus gland. This condition can occur **prematurely or at an atypical rate**, leading to significant **disruptions in immune system function**. The morphological changes associated with pathological thymus involution are distinct from the gradual age-related involution observed in healthy individuals.

Pathologically, thymus involution is characterized by a more **rapid** and **pronounced reduction** in thymic size and architecture. The thymus gland may undergo **atrophy**, becoming significantly smaller and losing its typical lobulated structure. This is often accompanied by **fibrosis**, where connective tissue replaces functional thymic tissue, impairing its ability to support thymocyte development.

In cases of pathological thymus involution, the reduction in thymic epithelial cells and stromal components can be more severe than in age-related involution. These changes result in a marked decrease in thymopoiesis, affecting the production and maturation of T-lymphocytes, which play a central role in adaptive immunity. The composition of thymic tissue may also become disorganized, with a loss of distinct cortical and medullary regions.

Moreover, pathological thymus involution may be associated with immune system dysfunction, **increased susceptibility to infections**, and **impaired immune responses**, making it a clinically significant condition.

Thymus hyperplasia

Thymus hyperplasia is a pathological condition characterized by an **abnormal enlargement and proliferation of thymic tissue**.

The **etiology** of thymus hyperplasia can be diverse and is often categorized into several subtypes:

1. **Reactive Thymus Hyperplasia**: This is a response to various stimuli, such as infections, autoimmune diseases, and immunological disturbances. In reactive thymus hyperplasia, the thymus enlarges in response to increased demand for T-cell production.
2. **Follicular Hyperplasia**: This subtype is characterized by the proliferation of lymphoid follicles within the thymus. It is often seen in the context of autoimmune diseases, such as myasthenia gravis or systemic lupus erythematosus.
3. **Epithelial Hyperplasia**: Epithelial hyperplasia involves an overgrowth of thymic epithelial cells and can be associated with thymomas (thymic tumors) or paraneoplastic syndromes.
4. **Medullary Hyperplasia**: This type of hyperplasia primarily affects the medullary regions of the thymus and may occur in autoimmune disorders like Addison's disease.

Morphology of thymus hyperplasia

1. **Increased Thymic Size**: Thymus hyperplasia leads to the enlargement of the thymus, which may affect the entire organ or specific regions within it.
2. **Distorted Architecture**: The excessive proliferation of thymic tissue can disrupt the typical lobulated structure of the thymus. Architectural disorganization is often observed.
3. **Cellular Changes**: Thymus hyperplasia is marked by an increased number of thymic epithelial cells, lymphocytes (thymocytes), and other thymic cell types. This includes both immature and mature T-cells, which may accumulate within the thymus.
4. **Histological Patterns**: Thymus hyperplasia may present with various histological patterns, which may assist in identifying the underlying cause. For instance, follicular hyperplasia displays prominent lymphoid follicles, while epithelial hyperplasia exhibits an overabundance of thymic epithelial cells.

Depending on the underlying etiology and the extent of thymus hyperplasia, clinical manifestations may vary. Patients can experience symptoms due to the **mass effect** on adjacent structures in the mediastinum, as well as **immunological abnormalities** caused by perturbations in T-cell maturation.

Hypersensitivity reactions

Hypersensitivity reactions, also known as allergic reactions, refer to **exaggerated and abnormal responses of the immune system** to substances that are typically harmless or well-tolerated by most individuals.

These reactions result from an overactive immune response, characterized by the production of **antibodies**, **immune cells**, or **inflammatory mediators**, and can lead to a wide range of clinical symptoms and pathological changes.

Hypersensitivity reactions are categorized into four main types (Type I, Type II, Type III, and Type IV), each involving distinct immunological mechanisms, and they can manifest as **allergic rhinitis**, **asthma**, **skin rashes**, or even severe and life-threatening conditions like **anaphylaxis**.

The severity and clinical presentation of hypersensitivity reactions vary depending on the specific immune mechanisms involved and the nature of the triggering antigens.

Type I Hypersensitivity

Type I Hypersensitivity (**Immediate Hypersensitivity**):

Pathological Mechanism:

Type I hypersensitivity is mediated by IgE antibodies and involves the degranulation of mast cells and basophils.

Upon re-exposure to an allergen, IgE-bound mast cells release vasoactive mediators like histamine, leading to immediate hypersensitivity reactions.

Pathological Features:

These reactions can manifest as urticaria (hives), angioedema, allergic rhinitis, bronchial asthma, and anaphylaxis.

Histologically, one may observe edema, vasodilation, and inflammatory cell infiltration, primarily eosinophils.

Type II Hypersensitivity

Type II Hypersensitivity (**Cytotoxic Hypersensitivity**):

Pathological Mechanism:

Type II hypersensitivity involves the formation of antibodies (IgG or IgM) against antigens on cell surfaces or tissues, leading to complement activation and cell destruction. It plays a role in autoimmune diseases.

Pathological Features:

This hypersensitivity can result in hemolysis, as seen in hemolytic disease of the newborn, or tissue damage, as in autoimmune disorders like myasthenia gravis and pemphigus vulgaris.

Histologically, affected tissues show signs of cell damage (degeneration and necrosis) and inflammation.

Type III Hypersensitivity

Type III Hypersensitivity (**Immune Complex-Mediated Hypersensitivity**):

Pathological Mechanism:

Type III hypersensitivity involves the formation of immune complexes (antigen-antibody complexes), which deposit in tissues, activating complement and attracting inflammatory cells.

Pathological Features:

This reaction underlies conditions such as systemic lupus erythematosus (SLE) and post-streptococcal glomerulonephritis.

Histologically, tissues may show vasculitis, glomerulonephritis, and tissue damage due to immune complex deposition.

Type IV Hypersensitivity

Type IV Hypersensitivity (**Delayed-Type Hypersensitivity**):

Pathological Mechanism:

Type IV hypersensitivity is T-cell mediated, involving sensitized CD4+ T-cells that release cytokines upon exposure to antigens.

This results in recruitment of macrophages and cytotoxic T-cells.

Pathological Features:

Delayed-type hypersensitivity is associated with conditions like contact dermatitis, tuberculosis, and autoimmune diseases like multiple sclerosis.

Histologically, it is characterized by mononuclear cell infiltrates, granuloma formation, and tissue damage.

Autoimmune diseases

Autoimmune disorders are often classified according to whether they are **organ-specific**, affecting only one organ or are **systemic** that is, affecting multiple organ systems. In addition, destruction of cells and tissues can be brought about by **autoantibodies** and/or **cell-mediated immunity**.

For example, in **multiple sclerosis** (MS) patients produce antibodies against myelin, the fatty material surrounding the axons of nerves. In addition, MS patients have TH and TC lymphocytes in their blood and cerebrospinal fluid, which are specific for myelin protein. Thus, humoral and cell-mediated autoimmunity may contribute to the demyelination of nerves in MS patients. In some instances, autoantibodies can block or stimulate a cell receptor. Myasthenia gravis is an example of the former, while Graves disease is an example of the latter.

Like most classification schemes, that of **autoimmune disorders is not perfect**. For example, Goodpasture's syndrome directly affects both kidneys and lungs while MS exerts systemic effects by attacking one type of tissue only.

Many, but not all, autoimmune disorders, affect a preponderance of **female patients**, with three times as many females as males presenting with autoimmune diseases. The reasons for this gender bias are unclear but may be related to sex hormone levels.

Many autoimmune disorders show a link with the type of MHC (major histocompatibility complex) antigens that are present on cells. In humans, the MHC is known as the **HLA system**. The links between HLA type and different diseases is described. So, for example, patients with Goodpasture's syndrome have a higher incidence of HLA-DR2 than the healthy population.

Autoimmune diseases

1) **Rheumatoid Arthritis (RA)**:

Target Organ: Joints, primarily synovial membranes.

Main Mechanism: Autoantibodies (e.g., rheumatoid factor and anti-citrullinated protein antibodies) and activated immune cells cause chronic inflammation, synovial hyperplasia, and joint destruction.

Morphological Features: Synovial lining inflammation, pannus formation (invasive synovial tissue overgrowth), and cartilage and bone erosion.

2) **Systemic Lupus Erythematosus (SLE)**:

Target Organs: Multiple, including skin, joints, kidneys, and blood vessels.

Main Mechanism: Formation of immune complexes containing autoantibodies (e.g., anti-dsDNA, anti-Smith) leads to tissue and organ damage due to complement activation and inflammation.

Morphological Features: Skin rashes (e.g., butterfly rash), glomerulonephritis, and various other manifestations, such as arthritis and serositis.

3) **Hashimoto's Thyroiditis**:

Target Organ: Thyroid gland.

Main Mechanism: Autoantibodies (anti-thyroid peroxidase and anti-thyroglobulin) cause inflammation and progressive destruction of thyroid tissue, leading to hypothyroidism.

Morphological Features: Lymphocytic infiltration of the thyroid gland, follicular cell damage, and formation of thyroid autoantibodies.

4) **Type 1 Diabetes Mellitus (T1DM)**:

Target Organ: Pancreatic islet cells (beta cells).

Main Mechanism: Autoimmune attack against insulin-producing beta cells, resulting in insulin deficiency.

Morphological Features: Islet cell inflammation (insulitis) and beta cell loss, eventually leading to hyperglycemia and diabetes.

5) Multiple Sclerosis (MS):

Target Organ: Central nervous system (CNS), specifically the myelin sheath of nerve cells.

Main Mechanism: Immune-mediated demyelination in the CNS, primarily driven by autoreactive T-cells.

Morphological Features: Multiple demyelinated plaques in the CNS, leading to neurological dysfunction, such as muscle weakness and sensory disturbances.

6) Celiac Disease:

Target Organ: Small intestine.

Main Mechanism: Autoantibodies (anti-tissue transglutaminase and anti-endomysial antibodies) cause inflammation and damage to the small intestine in response to gluten ingestion.

Morphological Features: Villous atrophy, crypt hyperplasia, and inflammatory cell infiltrates in the intestinal mucosa.

Immunodeficiency syndromes

Immunodeficiency syndromes, often referred to as primary immunodeficiency disorders, are a group of rare genetic or acquired medical conditions characterized by the impaired or dysfunctional immune system.

These syndromes result in a reduced ability to respond effectively to infections, leaving affected individuals susceptible to recurrent, severe, and sometimes unusual infections.

Immunodeficiency syndromes can affect various components of the immune system, including T-cells, B-cells, phagocytes, or complement proteins, leading to deficiencies in immune responses.

Proper diagnosis and management are crucial to minimize the risks associated with immunodeficiency syndromes, which may include severe infections, autoimmune diseases, and other complications.

Immunodeficiency syndromes

Primary immunodeficiencies:

- Common variable immunodeficiency (CVID)

- Congenital agammaglobulinemia (Bruton's disease)
- Severe combined immunodeficiency (TCI or SCID)
- Wiskott-Aldrich Syndrome (WAS)
- Thymus hypoplasia (DiGeorge syndrome)
- Isolated IgA deficiency
- Bloom's syndrome

Secondary immunodeficiencies:

- Drug therapy
- Malignant tumors
- Autoimmune diseases
- Prematurity
- Old age
- Trauma
- Burns
- Radiation therapy
- HIV infection

Immunodeficiency syndromes

1) **Common Variable Immunodeficiency (CVID)**:

Target Organ: Various, including the respiratory and gastrointestinal tracts.

Mechanism: Defective antibody production (B-cell dysfunction) results in recurrent bacterial infections and increased susceptibility to autoimmune diseases.

Clinical Features: Frequent respiratory and gastrointestinal infections, autoimmune conditions, and lymphoproliferative disorders.

Morphological Features: Histologically, one may observe lymphoid nodular hyperplasia and absence or reduction in plasma cells in affected tissues, particularly the gut-associated lymphoid tissue.

2) **X-Linked Agammaglobulinemia (XLA, Bruton's Disease)**:

Target Organ: Respiratory and gastrointestinal tracts.

Mechanism: Mutation in the BTK gene leads to a lack of mature B-cells and, consequently, very low or absent antibody production.

Clinical Features: Severe recurrent bacterial infections, especially with encapsulated bacteria.

Morphological Features: The absence of mature B-cells and plasma cells in lymphoid tissues.

3) **Severe Combined Immunodeficiency (SCID)**:

Target Organ: Multiple, affecting various systems due to profound immune dysfunction.

Mechanism: Genetic mutations result in impaired T-cell and B-cell development, leading to a severe lack of immune function.

Clinical Features: Profound immunodeficiency with life-threatening infections, failure to thrive, and opportunistic infections.

Morphological Features: The thymus is often hypoplastic or absent, and there's a marked deficiency of T-cells in lymphoid tissues.

4) **Wiskott-Aldrich Syndrome (WAS)**:

Target Organ: Multiple, with a significant impact on the blood, immune system, and skin.

Mechanism: Mutations in the WASP gene result in defective T-cell and B-cell function, leading to a triad of symptoms including eczema, thrombocytopenia, and immunodeficiency.

Clinical Features: Eczema, increased susceptibility to infections, and thrombocytopenia with a propensity for bleeding.

Morphological Features: Abnormal immune cell morphology, including small and fragmented platelets, and impaired lymphocyte function.

5) **DiGeorge Syndrome (22q11.2 Deletion Syndrome)**:

Target Organ: Thymus, parathyroid glands, heart, and facial structures.

Mechanism: Deletion in the 22q11.2 chromosomal region results in thymic hypoplasia and T-cell deficiency.

Clinical Features: Thymic hypoplasia, recurrent infections, hypoparathyroidism, and congenital heart defects.

Morphological Features: Thymic hypoplasia or aplasia, leading to reduced or absent thymic tissue.

HIV-infection

HIV (**Human Immunodeficiency Virus**) infection is a complex and chronic viral illness that primarily affects the human immune system. It is a significant global health concern, with more than 38 million people living with HIV worldwide. HIV belongs to the retrovirus family, with two main strains: HIV-1 and HIV-2. HIV-1 is the most widespread and pathogenic strain.

Etiology:

HIV is transmitted through contact with certain body fluids, including **blood**, **semen**, **vaginal fluids**, **rectal fluids**, and **breast milk**, from an infected individual.

The virus primarily spreads through **sexual contact**, sharing of needles for drug use, **perinatal transmission** from mother to child, and occupational exposure to infected blood.

Pathogenesis:

HIV primarily targets **CD4+ T-lymphocytes** (helper T-cells), which play a crucial role in orchestrating the immune response. The virus attaches to CD4 receptors and a co-receptor (typically CCR5 or CXCR4) on the surface of these cells, allowing viral entry and replication within the host cell. This process ultimately leads to the destruction of CD4+ T-cells and a decline in immune function.

Clinical Manifestations (stages of HIV-infection):

1) **Acute HIV Infection**: Initial exposure leads to flu-like symptoms, followed by a period of asymptomatic infection.

2) **Chronic HIV Infection**: This stage, which can last for several years, is often asymptomatic, but CD4+ T-cell counts gradually decline.

3) **Symptomatic HIV Infection**: As CD4+ T-cell counts drop below a certain threshold, patients may experience various symptoms and opportunistic infections.

4) **AIDS** (Acquired Immunodeficiency Syndrome): AIDS is the advanced stage of HIV infection, characterized by a severely compromised immune system and the occurrence of opportunistic infections and certain malignancies.

Laboratory Diagnosis:

Diagnosis typically involves the detection of **HIV-specific antibodies**, antigens (p24 antigen), or viral genetic material (**HIV RNA**) in blood or other body fluids. Confirmation often requires a combination of multiple tests.

Treatment:

Antiretroviral therapy (**ART**) is the cornerstone of HIV management. ART consists of a combination of medications that target different stages of the HIV life cycle, effectively **suppressing viral replication** and slowing disease progression. Early and consistent treatment can help maintain CD4+ T-cell counts and reduce the risk of opportunistic infections.

Prevention:

Preventive measures include practicing **safe sex**, using **sterile needles** for drug injection, pre-exposure prophylaxis (PrEP) for high-risk individuals, and early diagnosis and treatment to prevent transmission.

Opportunistic infections

Opportunistic infections are infections caused by pathogens, including bacteria, viruses, fungi, and parasites, that **typically do not cause illness in individuals with a healthy and functional immune system**.

These infections take advantage of a weakened or compromised immune system, such as in individuals with **HIV/AIDS**, cancer, organ transplantation, or immunosuppressive therapy, to establish disease.

Opportunistic infections often exhibit increased severity and a broader range of potential causative agents in individuals with **compromised immunity**.

Opportunistic infections

Pneumocystis jirovecii Pneumonia (PCP): PCP is one of the most common and potentially severe opportunistic infections in people with advanced HIV infection. It presents with fever, cough, and difficulty breathing. PCP can be life-threatening if not treated promptly.

Candidiasis: Oral candidiasis, or thrush, and esophageal candidiasis are common fungal infections seen in HIV-positive individuals. They can cause white patches in the mouth and throat, along with pain and difficulty swallowing.

Tuberculosis (TB): TB is a bacterial infection that can affect the lungs, as well as other organs, and is more common in people with HIV. It can lead to severe respiratory symptoms, night sweats, and weight loss.

Opportunistic infections

Toxoplasmosis: Toxoplasmosis is caused by the parasite Toxoplasma gondii and can affect the brain. Symptoms may include headaches, confusion, and seizures.

Cryptococcal Meningitis: This fungal infection can lead to severe inflammation of the brain and meninges, resulting in symptoms like headache, fever, and altered mental status.

Cryptosporidiosis: Cryptosporidium is a parasite that can cause severe diarrhea, abdominal cramps, and dehydration.

Opportunistic infections

Cytomegalovirus (CMV) Infection: CMV is a member of the herpesvirus family and can cause retinitis, leading to vision problems. It can also affect the gastrointestinal tract and other organs.

Herpes Simplex Virus (HSV) Infections: HIV-positive individuals are more susceptible to recurrent and severe HSV infections, which can cause oral and genital ulcers.

Kaposi's Sarcoma: Although technically a cancer, Kaposi's sarcoma is considered an opportunistic disease in the context of HIV. It presents as skin lesions and may also affect internal organs.

INFORMATIONAL AND METHODOLOGICAL PART

MAIN LITERATURE

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ADDITIONAL LITERATURE

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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.