Materials
for students for practical training in pathological anatomy
at the Department of Pathological Anatomy

II year Faculty of Dentistry

Topic: “Inflammation. Part 1. Inflammation, general characteristics. Acute inflammation. Exudative inflammation ".

1. The purpose of the lesson. To study the etiology and pathogenesis of acute inflammation, morphological characteristics, complications and outcomes of types of exudative inflammation.

2. Requirements for the level of the student for mastering the discipline - pathological anatomy. The student should know:

1. Definition of inflammation, etiology, mechanisms of development, phases of inflammation.

2. Classic clinical manifestations of inflammation, molecular mechanisms of development of each of them.

3. Classification of inflammation.

4. Cell mediators of inflammation: vasoactive amines, cytokines, nitric oxide, mediators of lysosomal granules.

5. Plasma mediators of inflammation: the system of blood coagulation, complement, kinins.

6. Characteristics of the stages of the inflammatory response.

7. Definition of exudative inflammation, its types.

8. Macro- and microscopic characteristics of various types of exudative inflammation.

9. Significance and outcomes of various types of exudative inflammation in various organs.

Theoretical aspects.

1. Inflammation.

Inflammation is a complex local reaction of the body in response to damage to its tissues by various pathogenic stimuli - aggressive stimuli and develops as a result of the body's interaction with numerous pathogenic factors of the external and internal environment of the body.

Exogenous (external) factors that can cause inflammation include: microorganisms (fungi, bacteria, viruses), animal organisms (protozoa, helminths, insects), toxic substances of a

chemical or any other nature, mechanical irritants (cold, heat), medicinal substances, ionizing radiation.

Endogenous (autogenous) factors causing inflammation include: products of nitrogen metabolism and decay of tumors, effector immune cells, as well as immune complexes precipitating in tissue.

In addition, the main causes of inflammation can be divided into the following groups:

1) necrobiotic changes in tissues and cells under the influence of exogenous physicochemical factors;

2) invasion of foreign microorganisms or antigens, immunogens into the internal environment;

3) malignancy of the body's own cells;

4) loss of immunological tolerance in relation to antigens of its own body.

Inflammation is a defensive reaction that achieves its biological goal mainly through:

1) activation of the complement system,

2) degranulation of mast cells,

3) increased permeability of microvessels and adhesive capacity of the endothelium,

4) migration of blood plasma into the intercellular spaces,

5) adhesion to endothelial cells of neutrophils, monocytes and lymphocytes of circulating blood and their exit into the interstitium,

6) phagocytosis, bactericidal and cytolotic action of phagocytes,

7) expansion, spasm and thrombosis of microvessels,

8) replacement of tissue defect through angiogenesis and proliferation of fibroblasts.

Inflammation, like any protective reaction of the body, is excessive in relation to the stimuli that caused it and therefore the typical pathological process is often transformed. The activation of the immune response during inflammation is provided by two cellular systems of nonspecific defense: the system of monocytic phagocytes, and also the plasma system - the complement system.

The kinetics of the inflammatory response to achieve the ultimate goal - the elimination of the damaging agent and tissue repair - is characterized by a change in the relationship of cellular defense systems with each other and with the connective tissue system, which is determined by mediator regulation. As a chain, largely self-regulating, the inflammatory response fits into a universal scheme: damage - mediation - reception - cellular cooperation - cellular transformations - repair.

Inflammation consists of interrelated and consistently developing phases: The first phase - alteration (damage) of tissues and cells (initial processes) with the release of mediators - morphobiochemical. The second phase - exudation - the reaction of the micro-circulatory bed with a violation of the rheological properties of blood. manifestations of increased vascular permeability in the form of plasma exudation and cell emigration, phagocytosis, and exudate formation. The third phase is cell proliferation with tissue repair or scar formation.

Classification of inflammation.

1. By etiology - with established or not established etiology. Chronic inflammation of bacterial etiology is subdivided into commonplace and specific.

2. Downstream - acute and chronic inflammation.

3. By morphology - exudative and productive inflammation.

2. Acute inflammation.

Acute inflammation is a form of inflammatory reaction that develops immediately after exposure to a damaging agent and is characterized by a predominance of exudative tissue reaction, as well as rapid completion with the elimination of the damaging agent and tissue repair.

Acute inflammation is classified depending on its location and the type of exudate formed:

1) serous inflammation,

2) fibrinous inflammation,

3) purulent inflammation,

4) hemorrhagic inflammation,

5) mixed inflammation,

6) catarrhal inflammation.

Serous inflammation is characterized by the formation of exudate containing 1-8% protein, while the transudate contains up to 1-2% protein. In addition, the exudate contains single polymorphonuclear leukocytes and desquamated epithelial cells. It develops most often in serous cavities, mucous membranes, pia mater, skin, less often in internal organs.

Causes: infectious agents, physical (thermal) factors, autointoxication.

Serous inflammation in the skin with the formation of vesicles is a characteristic sign of inflammation caused by viruses of the Herpes viridae family (herpes simplex, chickenpox). Thermal, less often chemical burns are characterized by the formation of blisters in the skin filled with serous exudate. Examples of serous inflammation are: erysipelas of the skin, urticaria, pemphigus, radiation damage.

The outcome of serous inflammation is usually favorable - complete resorption of the exudate.

Fibrinous inflammation is characterized by the formation of exudate with a high content of fibrin in the effusion, in addition to which stab leukocytes and elements of necrotic tissue are found in the exudate.

Localization: mucous and serous membranes.

There are the following types of fibrinous inflammation:

1) croupous inflammation and

2) diphtheria inflammation.

Croupous inflammation usually develops on the mucous membranes lined with multi-ciliated epithelium (trachea, bronchi) or on the serous membranes; characterized by the presence of fibrin films freely located on the surface of the mucous membrane; alterative phenomena are limited only to some desquamation of epithelial cells or mesothelium.

Diphtheritic inflammation usually develops on mucous membranes lined with multilayer flat and transitional epithelium (mouth, pharynx, esophagus, vagina, urinary bladder, vocal cords); characterized by the presence of a fibrin film tightly adhered to the surface of the inflamed tissue and soaking it to various depths; alternative phenomena are expressed significantly.

The course and outcome of fibrinous inflammation are usually favorable; adhesions may form in the serous cavities.

Purulent inflammation is characterized by the presence in the liquid exudate of a large number of polymorphonuclear leukocytes, both live and dead, which give pus greenish tints. In pus, microorganisms, tissue detritus, large amounts of protein, cholesterol, lecithin, fats, soaps, an admixture of deoxyribonucleic acid, which impart viscosity to pus, are almost always found.

The color of pus can be yellow-green, bright green, bluish, dirty gray, etc. depending on the impurities of certain pigments, microbes, as well as on the age of its formation.

The consistency of pus is sometimes liquid, sometimes more or less thick, sometimes stringy. Fresh pus is always liquid, thickened pus is a sign of the relative prescription of the process.

Types of purulent inflammation:

1) empyema,

2) phlegmon,

3) abscess.

Empyema is a purulent inflammation that develops in the body cavities, joints, and closed channels.

Phlegmon is a diffuse purulent inflammation of cellulose with the fusion of the latter.

Soft phlegmon is characterized by the absence of visible foci of necrosis in the tissues.

Solid phlegmon is characterized by the presence of foci of coagulation necrosis, which do not undergo melting, but are gradually rejected.

An abscess is a limited purulent inflammation characterized by tissue fusion with the formation of a cavity filled with pus and limited by a pyogenic membrane, which is a layer of granulation tissue.

The course and outcomes of purulent inflammation:

1) the formation of fistulas,

2) the formation of seals,

3) spontaneous emptying of pus with subsequent scarring of the site of inflammation,

4) sequestration.

Hemorrhagic inflammation is characterized by an admixture of blood (erythrocytes) to any exudate; observed with flu, plague, anthrax.

Catarrhal inflammation develops in the mucous membranes and is accompanied by hypersecretion of mucus. The nature of the exudate may be different, but mucus is an essential component of it. The outcome is usually favorable.

The outcome is unfavorable.

Mixed inflammation is observed in cases when another type of exudate is attached to one type of exudate, resulting in serous-purulent, serous-fibrinous, purulent-fibrinous and other types of inflammation.

3. Lesson plan

Macropreparations.

1. To study croupous inflammation according to the macroscopic picture. Describe the macro-preparation "**Croupous pneumonia**" - pay attention to the size and color of the lung, the prevalence of the process, changes in the pleura; determine the stage of croupous pneumonia.

2. To study croupous inflammation according to the macroscopic picture. Describe the macropreparation "**Fibrinous pericarditis**". - Pay attention to the thickness, transparency, color of the pericardium and the features of the fibrinous film on its surface - color, appearance and tightness of connection with underlying tissues.

3. To study diffuse purulent inflammation according to the macroscopic picture. Describe the macropreparation "**Phlegmonous appendicitis**" - pay attention to the size of the appendix, the color and condition of the serous membrane; on the cut, pay attention to the wall thickness, the severity of the layers, the contents in the lumen of the appendix.

4. To study diffuse purulent inflammation according to the macroscopic picture. Describe the macropreparation "**Purulent leptomeningitis**". - Pay attention to the appearance, color, thickness, state of the vessels of the pia mater, the contents of the subarachnoid space, the state of the brain tissue, as well as the type of convolutions and furrows.

5. To study hemorrhagic inflammation in the macroscopic picture. Describe the macropreparation "**Measles bronchopneumonia**" - pay attention to the size and color of the lung, the prevalence of the process.

6. To study focal purulent inflammation according to the macroscopic picture. Describe the macro-preparation **Embolic purulent nephritis** - Pay attention to the size and consistency of the kidney, the number, color, shape, size and localization of lesions.

7. To study diphtheria inflammation in the macroscopic picture. Describe the macropreparation "**Diphtheritic colitis**". - Pay attention to the color, surface, thickness and nature of the attachment of the film that replaces the mucous membrane of the colon.

8. To study catarrhal inflammation in the macroscopic picture. Describe the "**Catarrhal gastritis**" macro-preparation. - Pay attention to the thickness, color and appearance of the gastric mucosa, localization, amount, color and transparency of the exudate.

Micropreparations.

1. To study croupous inflammation on a microscopic picture. Describe the micropreparation "**Croupous pneumonia**" (staining with hematoxylin and eosin, for fibrin - according to Shuenino-vu). - Pay attention to the prevalence of the lesion, the localization and composition of the exudate, the state of the interalveolar septa and capillaries. When staining according to Shueninov, pay attention to the localization and color of fibrin filaments in the exudate.

2. To study diphtheria inflammation by a microscopic picture. Describe the micro-preparation "**Diphtheria throat inflammation in diphtheria**" (staining with hematoxylin and eosin). - Pay attention to the condition of the mucous membrane in the area of the tonsil crypt, the thickness, composition and localization of the fibrinous film, changes in the underlying tissues.

3. To study serous-hemorrhagic inflammation in a microscopic picture. To describe the microdrug "**Serous-hemorrhagic pneumonia**" - to pay attention to the nature of the exudate in the lumens of the alveoli, its cellular composition, the prevalence of the process, the state of the interalveolar septa, bronchi and vessels of the lung.

4. To study diffuse purulent inflammation in a microscopic picture. Describe the micropreparation "***Phlegmonous-ulcerative appendicitis***" - pay attention to the nature of the exudate, the state of the vessels, the integrity of the wall of the appendix.

5. To study diffuse purulent inflammation in a microscopic picture. Describe the micropreparation "***Purulent leptomeningitis***" (staining with hematoxylin and eosin). - Pay attention to the thickness of the pia mater, the location, prevalence and composition of the infiltrate, the state of the vessels, as well as the tissue of the membranes in the affected area and adjacent brain tissue.

6. To study diphtheria inflammation on the microscopic picture. Describe the micro-preparation "***Diphtheria colitis***" (staining with hematoxylin and eosin). - Pay attention to the condition of the intestinal mucosa, thickness, composition and localization of the fibrinous film, changes in the underlying tissues.

7. To study focal purulent inflammation on a microscopic picture. Describe the micro-preparation ***Embolic purulent nephritis*** (staining with hematoxylin and eosin). - Pay attention to the cellular composition of the infiltrate and the state of the kidney tissue in the lesion focus, as well as in the zone of demarcation inflammation, the localization of microbial colonies and their relationship with the vessel

Electron diffraction patterns.

1. To study the mechanism of exudate formation using electron microscopy. Describe the electron diffraction pattern "**Pinocytosis in the endothelium of the vessel during inflammation**." - Pay attention to the state of the cytoplasm of endothelial cells, the number of pinocytic vesicles and intercellular spaces.

2. To study the mechanism of exudate formation using electron microscopy. Describe the electron diffraction pattern "**Emigration of neutrophil through the vessel wall during inflammation**." - Pay attention to the part of the vascular bed where the neutrophilic leukocyte overcomes the endothelial barrier, the features of its relationship with the endothelial cell and the location of its pseudopodia.

Situation cases.

Situation case 1.

Patient M., 58 years old, had the flu for 10 days, on the 11th day there was an increase in body temperature to 39.5 ° C, dyspnea and cyanosis increased, with symptoms of progressive pulmonary heart failure and intoxication, the patient died.

Autopsy: the lungs are enlarged, hardened, dark red; microscopically: arterioles are dilated and filled with blood, in the lumens of the alveoli - serous fluid with an admixture of a significant amount of red blood cells.

Questions to the situation case 1

1) What pathological process has developed in the lungs?

2) What kind of the described pathological process is this?

Situation case 2.

Patient K., 29 years old, was admitted to the hospital with complaints of nausea, vomiting, pain in the right iliac region. During the operation, an enlarged crimson appendix was found in the abdominal cavity, the peritoneum of the small pelvis was hyperemic, dull, covered with loose, grayish-yellowish films; the appendicular process is removed. Microscopic examination of the removed appendix revealed: pronounced edema of the wall of the appendix, plethora of blood vessels and focal hemorrhages, diffuse infiltration of the wall by neutrophils.

Questions to the situation case 2

1) What pathological process took place in the appendix?

2) What kind of the described pathological process in the appendix is this?

3) What pathological process took place in the pelvic area (peritoneum)?

4) What kind of the described pathological process in the pelvic area (peritoneum) is this?

Situation case 3.

Patient S., 38 years old, had ARVI for 4 days, The mucous membrane of the upper respiratory tract is edematous, hyperemic, covered with mucus, from the 5th day the discharge from the nose became greenish-gray in color with an unpleasant odor.

Questions for situation case 3

1) What pathological process are we talking about?

2) What kind of the described pathological process is this?

3) Describe the microscopic picture of this pathological process.

Situation case 4.

In a 89-year-old patient, after a minor injury, the abrasion on the thigh turned red, after 3 days the soft tissues of the thigh became swollen, thickened, a viscous, greenish content began to stand out from the wound; the patient's temperature rose to 38 ° C, severe pains in the hip region, decreased appetite, and severe weakness were noted. During the operation for excision of the thigh wound, it was found that the subcutaneous fatty tissue of the thigh was saturated with greenish masses, spreading in the form of tongues on the soft tissues of the lower leg.

Questions for situation case 4

1) What pathological process are we talking about?

2) What kind of the described pathological process is this?

3) What are the names of the described "tongues" on the thigh?

4) Describe the microscopic picture of this pathological process.

Situation case 5

Patient D., 38 years old, who suffered from chronic glomerulonephritis, developed chronic renal failure. Auscultation revealed a rubbing noise of the pericardium and pleura. The patient died. On autopsy, the sheets of the heart shirt are thickened, dull, rough, with many filamentous overlays of a whitish-grayish color; overlays are easily removable. Pleural leaves of both lungs are full-blooded, with petechiae, dull due to easily removable grayish films. At dissection, the folds of the stomach are thickened, covered with a large amount of viscous mucus.

Questions for situation case 5

1. What kind of exudative inflammation developed on the sheets of the heart

shirts and pleura?

2. What kind of this inflammation developed on the pericardium and pleura?

3. What is the composition of the exudate?

4. What type of exudative inflammation developed in the stomach?

5. What kind of this inflammation takes place?

Situation case 6

Patient K., 70 years old, underwent intramuscular injections for the treatment of ischemic cerebral infarction. Signs of inflammation appeared at the injection site in the upper-outer quadrant of the right gluteal region. Fever 38.5 OS. Prescribed antibiotic therapy and compresses locally. After the course of treatment, the body temperature returned to normal, but the compaction in the gluteal region retained the axis. Suddenly the patient felt worsening of her condition: chills, sharp pain in the lower abdomen, fever up to 39 OS. Objectively: pain on palpation in the lower abdomen, blood leukocytes - 20 x 109 / l; ESR - 30 mm / h. When opening the focus of compaction in the gluteal region, the contents of a creamy consistency were released, a cavity was formed.

Questions to situation case 6

1. What local and general signs of inflammation did the patient have during the development of the disease?

2. What type of exudative inflammation developed in the gluteal region?

3. What kind of this inflammation?

4. What is the composition of the exudate?

5. Why was a cavity formed after evacuation of the exudate?

6. Name the causes and mechanisms of development of soft tissue inflammation

gluteal region, its complications.

7. What is a pyogenic membrane?

Situation case 7

Patient L., 34 years old, was admitted to the therapeutic department with complaints of chills, shortness of breath, pain in the right side during deep breathing. On a direct survey X-ray, an intense darkening in the projection of the lower lobe of the right lung was revealed. The content of leukocytes in the blood is 16 x 1012 / l, ESR is 26 mm / h. Biochemical blood test: total protein 72 g / l; albumin 57%; α-globulins 1.6% (norm 3-6%); α2-globulins 23.5% (norm 9-15%); γ-globulins 27% (norm 15-25%). The patient was diagnosed with right-sided croupous pneumonia. Treatment of the disease turned out to be ineffective, and the patient died on the 6th day of the disease. The diagnosis was confirmed by autopsy.

Questions to situation case 7

1. What type of exudative inflammation developed in the lung?

2. What component of the exudate should be detected during additional staining of micropreparations?

3. What changes in the blood test indicate the presence of an inflammatory process?

4. What is the mechanism of development of hematological changes?

Literature to the Topic 3

Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. “Pathology. Quick Review and MCQs” Harsh Mohan, 2004.

2. “Textbook of Pathology ” Harsh Mohan, 2002.

3. “General and Systemic Pathology” Joseph Hunter, 2002.

4. “General and Systematic Pathology ” Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2th).

5. “Histology for Pathologist” Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2th).

6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.

7. “Muir’s Textbook of Pathology” Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14th). 8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3th).

9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4th).

10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6th).

11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3th).

12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10th).

13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7th). 14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.

15. “Pathology” Virginia A. LiVolsi, Maria J. Merino, John S. J. Brooks, Scott H. Saul, John E. Tomaszewski, 1994.

16. “Short lectures on pathology” Zagoroulko A., 2002

17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.

18. “General pathology” Dr. Fatma Hafez, 1979.

19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10th).

https://www.volgmed.ru/ru/depts/list/69/

https://volgmu-pat-anat.3dn.ru/

https://webpath.med.utah.edu/