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Acute inflammation. The morphology of exudative inflammation.

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1. Purpose: To study the pathological processes in acute inflammation. Etiology of inflammation, mediators, phases: alteration, exudation.
2. Requirements for the level of the student for mastering the discipline - general pathology.

The student should know:
1. The concept of inflammation. The biological meaning of inflammation. The area of development of the inflammatory process.
2. The etiology of inflammation. Inflammatory mediators.
3. Classification of inflammation. Morphogenesis of inflammation.
4. Phases of inflammation: Alteration, exudation.
5. Characteristics, outcomes of acute inflammation.

Theoretical aspects:
Inflammation is a complex, local and general pathological process that occurs in response to damage or the action of a pathogenic stimulus and manifests itself in reactions aimed at eliminating the products of damage, and, if possible, agents (irritants), as well as leading to the maximum recovery for these conditions in the damaged area.
The etiology of inflammation is diverse. It can be caused as exogenous (toxic, chemical agents - drugs, toxins and poisons; physical factors - exposure to high and low temperatures, electricity, radiation energy, dust, aerosols, trauma; microbes and their metabolites viruses, mycoplasmas, bacteria, fungi, animal parasites). and endogenous factors of various origins. (ischemia - inflammation on the border with the zone of ischemic necrosis; toxic agents formed in some diseases - uric acid salts in gout, urea and its metabolites in uremia; microbes - saprophytes and their metabolites in conditions of immunosuppression - pneumonia caused by autogenous flora; immunopathological reactions - hypersensitivity reactions, autoimmune reactions), accompanied by the so-called immune inflammation).
The inflammatory response goes through three successive phases:
1. Inflammation begins with alteration (damage) of tissue and cells, including cells - carriers of inflammatory mediators - chemicals that regulate inflammation (mast cells, macrophages, platelets). Alteration provides the initial release and accumulation of inflammatory mediators in tissues.
2. Exudation - the exit of the liquid part, blood; and shaped elements outside the vascular bed.
Exudation stages.
1. Reaction of the microvasculature with impaired blood rheological properties: short-term vasoconstriction; vasodilation (arterioles, capillaries and postcapillaries) with the development of inflammatory hyperemia; slowing down of blood flow, increased hydrostatic pressure, plasmorrhage, increased blood viscosity, stasis.
2. The second component of the exudative reaction - the formation of a liquid part of the inflammatory exudate due to an increase in tissue-vascular permeability as a result of structural rearrangements in the microvasculature - is provided according to Starling's law by an increase in hydrostatic pressure in the microcirculation vessels with a simultaneous decrease in osmotic pressure in them as a result of the release of plasma proteins in combination with an increase in the permeability of the vascular walls.
3. Emigration of cellular elements (release of cells from blood vessels) and phagocytosis.
The accumulation of leukocytes in the focus of inflammation goes through several stages:
• marginal standing of leukocytes in the bloodstream, rolling of leukocytes on the surface of endothelial cells and adhesion of leukocytes to the surface of the latter;
• migration of leukocytes through the vascular wall, also called leukodiapedesis;
• migration of leukocytes into the interstitial tissue in the inflammation focus in the direction of the difference in concentration gradients of certain chemicals (chemotaxis).
Phagocytosis is the absorption and digestion of various particles by cells (phagocytes) - microbes, foreign bodies, apoptotic bodies, detritus, etc.

The following stages of phagocytosis are distinguished:
• recognition and attachment of the object of phagocytosis to the surface of the leukocyte; in the case of microbial phagocytosis, these processes occur due to the interaction of receptors located on the surface of the leukocyte with opsonins (Fc fragments of immunoglobulin G and C3b components) that cover the surface of microbes;
• capture of the object of phagocytosis and the formation of a phagolysosome;
• destruction and / or destruction of absorbed material. In the case of bacterial phagocytosis, their destruction is carried out, as a rule, through the generation of reactive oxygen species and HOCl-generated during the respiratory explosion and during the interaction of leukocyte myeloperoxidase with hydrogen peroxide and chlorine ions. In addition to the described mechanism, there is a way to destroy bacteria using bactericidal proteins contained in secondary granules of leukocytes (bactericidal cationic protein, lysozyme, lactoferrin), and eosinophils (main basic protein).
Classification of inflammation.
1. Depending on the nature of the course, inflammation can be acute, subacute and chronic.
2. By the prevalence of the phase of inflammation, exudative inflammation (mainly acute) and productive (mainly chronic) are distinguished.
The morphological feature of acute inflammation is the predominance of exudative tissue reaction. Acute inflammation is classified according to its location and the type of exudate that forms. With the localization of the process on the mucous membranes, there is a hyperproduction of mucus, which is mixed with the inflammatory exudate, which determines the development of a special type of exudative inflammation - catarrhal.
There are various types of exudate, differing in their protein and cellular composition.
• serous exudate contains up to 2% protein, single neutrophilic leukocytes, desquamated damaged epithelial cells;
• fibrinous exudate is rich in fibrin;
• purulent exudate is rich in proteins and contains not only a large number of neutrophilic leukocytes, but also tissue detritus, formed as a result of tissue lysis;
• hemorrhagic exudate is characterized by a predominance of erythrocytes;
• putrefactive exudate is associated with putrefactive flora;
• mixed forms.
Inflammation outcomes.
Full resolution. Complete tissue repair is observed with small amounts of damage. Resolution of inflammation includes neutralizing chemical mediators, restoring normal vascular permeability, stopping leukocyte infiltration, and finally removing edematous fluid, protein, leukocytes, foreign agents, and tissue detritus.
Healing with connective tissue replacement (fibrosis). The process begins after partial destruction of tissue or the development of inflammatory damage in tissues that are unable to regenerate, as well as with massive exudation of fibrin. In the event that the fibrinous exudate in the tissue or serous cavities (on the pleura and peritoneum) is not completely absorbed (resorbed), the connective tissue invades the exudate, turning it into a mass of fibrous tissue. This process is called organization.
The following are the adverse outcomes of inflammation.
The progression of an acute inflammatory response to various forms of chronic inflammation occurs when the acute inflammatory response is not resolved, there is a persistence of the damaging agent, or a violation of the organization process. For example, a bacterial infection of the lung begins as a focus of acute inflammation (pneumonia), but the melting of damaged tissue can lead to a cavity in which inflammation continues, resulting in a chronic lung abscess.
The spread of inflammation to adjacent tissues. In this case, fistulas and fistulas are formed and even generalization of the infection is observed (as is the case with sepsis), as well as tissue melting with perforation of the walls of organs and vessels, which is the cause of the spread of inflammation to the peritoneum, pleura, as well as internal bleeding.

Immune inflammation, which usually becomes chronic, underlies such serious human diseases as glomerulonephritis, viral hepatitis, idiopathic fibrosing alveolitis, tuberculosis, bronchial asthma, rheumatic diseases, systemic vasculitis, etc.
There are a number of acquired and hereditary diseases caused by impairments of various functions of leukocytes and manifested in the predisposition of patients to infectious diseases and the development of chronic forms of inflammation.

Lesson plan.

Explore and describe:

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

2. To study focal purulent inflammation in a microscopic picture. Describe the **micropreparation** "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.

3. To study diffuse purulent inflammation according to the macroscopic picture. To 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 appearance of the convolutions and furrows.

4. 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 tissues of the membranes in the affected area and adjacent brain tissue.
5. 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 - the color, appearance and density of connection with the underlying tissues.

6. To study croupous inflammation on a microscopic picture. Describe the **micropreparation** "Croupous pneumonia" (staining with hematoxylin and eosin, for fibrin - according to Shueninov). 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.

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 replacing the mucous membrane of the large intestine.

8. To study diphtheria inflammation in a microscopic picture. Describe the **micropreparation** "Diphtheritic colitis" (staining with hematoxylin and eosin). Pay attention to the condition of the intestinal mucosa, the thickness, composition and localization of the fibrinous film, changes in the underlying tissues.

9. To study diphtheria inflammation in a microscopic picture. Describe the **micropreparation** "Diphtheritic inflammation of the throat in diphtheria" (staining with hematoxylin and eosin). Pay attention to the condition of the mucous membrane in the area of crypt, the thickness, composition and localization of the fibrinous film, changes in the underlying tissues.

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

11. Examine the granulation tissue on a microscopic picture. Describe the **micropreparation** "Granulation tissue" (staining with hematoxylin and eosin). Pay attention to the number and characteristics of blood vessels, name the cells of granulation tissue.

12. To study large-focal cardiosclerosis on a macroscopic picture. Describe the **macropreparation** "Large focal cardiosclerosis". Pay attention to the localization, size, color and shape of the lesion, wall thickness and the state of the surrounding myocardium.

13. To study large-focal cardiosclerosis by microscopic picture. Describe the **micropreparation** "Large focal cardiosclerosis" (staining with hematoxylin and eosin, picrofuchsin). Pay attention to the shape and structure of the lesion, the state of adjacent myocardiocytes, as well as the difference in the color of the lesion and myocardiocytes when stained with picrofuchsin.

Test QUESTIONS

Choose one correct answer
1. Local, complex, vascular-mesenchymal reaction in response to injury.
a) necrosis,
b) adaptation,
c) inflammation,
d) thrombosis,
e) plethora.

Select all correct answers
2. Inflammation has the following phases:
a) alteration,
b) exudation,
c) proliferation,
d) reparation.

Select all correct answers
3. The main components of the exudation phase:
a) alteration,
b) a change in blood flow,
c) the formation of inflammatory edema,
d) proliferation,
e) cell emigration and phagocytosis.

Select all correct answers
4. The emigration of leukocytes to the inflammatory focus has the following stages:
a) marginal standing in the bloodstream,
b) diapedesis,
c) chemotaxis,
d) phagocytosis.

Select all correct answers
5. Mediators of inflammation involved in emigration
leukocytes in the field of inflammation:
a) adhesive molecules on the surface of leukocytes,
b) adhesive molecules on the surface of the endothelium,
c) integrins CD11 / CD18, VLA-4, L-selectin,
d) immunoglobulins ICAM-1, VCAM-1,
e) IL-1 and TNF.

Establish compliance
6. Composition of the infiltrate: Pathological process:

1) neutrophilic leukocytes - a) repair,

2) mononuclear cells b) acute inflammation, c) atrophy d) chronic inflammation.
Select all correct answers
7. Components of the plasma protease system:
a) the complement system,
b) the TNF family,
c) kinin system,
d) blood coagulation system,
e) membrane attacking complex.

Select all correct answers
8. Types of exudative inflammation:
a) granulomatous,
b) abscess,
c) catarrhal,
d) chronic.
Select all correct answers
9. Catarrhal inflammation is characterized by the following
signs:
a) can be diphtheritic,
b) fibrin is always a part of the exudate,
c) a very large amount of exudate,
d) the resulting films are tightly bound to the underlying tissues,
e) outcome - complete restoration of tissues.
Select all correct answers
10. Fibrinous pericarditis is characterized by the following
signs:
a) often occurs with uremia,
b) the figurative name "hairy heart",
c) can be with transmural myocardial infarction,
d) there are adhesions in the cavity of the heart shirt,
e) accompanied by a pleural friction noise,
f) diphtheria inflammation.
Select all correct answers
11. Emigration of leukocytes (leukodiapedesis) is characterized by the following features:
a) leukocytes come out interendothelially,
b) the basement membrane is overcome by the thixotropy mechanism,
c) leukocytes form pseudopodia,
d) leukocytes enter the field of inflammation following monocytes,
e) leukocytes go beyond the vascular wall through the mechanism of pinocytosis.

List of recommended literature:
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 (2^{th}).
5. "Histology for Pathologist" Ed. S.S.Sternberg – Philadelphia: Lippincott Raven Publ, 1997 (2^{th}).
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 (14^{th}).
8. "Pathology" Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3^{th}).

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).

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