LECTURE 7

Inflammation

*is a typical pathologic process which appears as an answer to action of damaging agent and shows in three interrelated reactions – **alteration**, microcirculation disorder together with **exudation**, emigration and **proliferation**.*

clinical presentations of inflammation

- Celse and Halen were the first who described :
- swelling,
- pain,
- reddening,
- temperature increase,
- function disorder.

Virkhov

- showed the significance of cellular reaction in development of inflammatory reaction in parenchyma and stroma of organs.

I.I. Mechnikov

- discovered the phenomenon of **phagocytosis** in the process of inflammation.

D.F. Kongame

- showed that the **vascular reaction** is of great importance in the development of inflammation, as well as vascular penetration increase, outlet of plasma and cellular elements from the vessels which determines swelling.

Etiology of inflammation

- 1-the **biological agents**
- viruses,
- bacteria,
- Rickettsia,
- fungi,
- Protozoa
- helminthes.

Etiology

- 2- **physical causes**
- - traumas,
- - radiant energy,
- - high and low temperatures
- 3- **chemical** ones
- - acids
- -alkalis.

Etiology

- **Internal (endogenous) factors**
- metabolic products
- result of necrosis,
- tumor decay,
- hemorrhage,
- thrombosis,
- salt deposits.
- Immune complexes

Morphologic stage of inflammation

- alteration,
- exudation,
- proliferation.

Alteration

☐*that is damage of tissue - is an initial stage of inflammatory process. Its essence is in local metabolic disorder and* ***dystrophic*** *changes of parenchyma and stroma to the very* ***necrosis***
Mediators of inflammation

Biologically active substances which are accumulated in the damaged area and determine further kinetics of inflammatory process (secondary alteration) support the alteration.
Mediators of inflammation are

☐**Cellular:**
☐ Vasoactive amines:
☐-histamine is synthesized in tissue basophiles (labrocytes)
☐serotonin is synthesized in platelets
☐metabolites of arachidonic acid (prostaglandins, leukotrienes); activation factor of platelets, tumor necrosis factors, interleukins, interferon
☐Lysosome ferments of granulocytes, monocytes, tissue macrophages and basophiles – protease, esterase, collagenase, elastase
☐ **plasmic**
Pathogenesis of inflammation

Consequences of inflammation

☐a) renovation of structure and function of the organ;
☐b) renovation of structure of the organ by means of substitution (cicatrization);
☐c) conversion to chronic form;
☐d) death of vitally important organs and the organism.

Terms of inflammation

☐addition of ending "itis" to the Latin or Greek name of organ or tissue (pleuritis, appendicitis, and conjunctivitis).
☐Sometimes the special term is used (angina, pneumonia).
Classification of inflammation

☐ *According to the etiology*
☐-ordinary inflammation,
☐ -specific one (tuberculosis, syphilis, leprosy, glanders, rhinoscleroma).
☐*According to anamnesis*
☐fulminant,
☐ acute,
☐subacute
☐chronic inflammations.
☐*According to morphological forms*
☐exudative
☐ productive.
Exudative inflammation

☐***Exudative inflammation*** *– the type of inflammation, in which exudation prevails over alteration and proliferation.*
Classification of exudative inflammation
Serous inflammation

☐**Aetiology:**
☐thermal,
☐chemical
☐ biological agents (microbacteria of tuberculosis, diplococci of Franckel, meningococci, shigels),
☐autointoxications (thyrotoxicosis, uremia).
☐e.g., cardiosclerosis, hepatocirrhosis at thyrotoxicosis).
Serous inflammation
☐The exudation contains about 2% of proteins.
☐It is accumulated in serous cavities, between leaves of soft brain tunic,

☐in perisinusoid and perivascular spaces, in the intersticium of the organs,
☐ Shumlyansky - Bowman's capsule,
☐in the epidermis and under, generating vesicles,
☐in alveoles' lumens.
☐**consequence** of serous inflammation
☐-resolution,
☐-the sclerosis appears not so often (
Serous inflammation
Fibrinous inflammation

☐***Aetiology:***
☐-uremia
☐- mercuric
☐-biological agents(diplococcus of Franckel, streptococcus, staphylococcus, microbacteria of tuberculosis, pathogens of diphtheria, dysentery, and influenza)
☐The exudation contains fibrin.
☐It is **accumulated** in mucous and serous membranes, as an exception in the organ (croupous pneumonia).
☐**two subtypes** of the *Fibrinous* inflammation
☐croupous inflammation
☐ diphtheritic inflammation.
Fibrinous inflammation

☐**Croupous inflammation**
☐fibrinous membrane removal easy
☐**Diphtheritic inflammation**
☐fibrinous membrane removal difficult
☐the area of necrosis of mucous or serous membranes is the deeper and bigger
☐At exfoliation of the membrane the ulcers, hemorrhage, bleeding appear
☐always appears on mucous membranes covered by multilayer pavement epithelium: tonsils, esophagus, groin, neck of uterus, on skin
☐At the same time the fibrin threads penetrate between epithelial cells, and it is difficult to remove the membrane.
Croupous (lobar) pneumonia.
☐Lung is grey colour on a cut, fibrinous inflammation develops in parenchyma of organ.
Croupous pneumonia
☐Staining with haemotoxillin-eosin in clearance of alveoli mere is considerable amount of fibrin, among which there are separate leucocytes and alveolar macrophages. Interalveolar membranes are thickened as a result of infiltration by cellular elements.
Diphtheria of respiratory tract.
☐ Clearance of trachea and bronchial tubes is filled with fibrinous pseudomembranes
Fibrinously-ulcerous enteritis.
☐Mucousa is with phenomena of alteration process of necrosis and layings of fibrin are present.
Consequence of ***Fibrinous inflammation***

☐-dissolve
☐-organize
☐-invasion into granulation tissue
☐-commissures generation or cavities obliteration (obliterating pleurisy or pericarditis)
☐ -organs' deformation (stenosis of bowel).
Suppurative inflammation
☐***Aetiology:***
☐staphylococcus,
☐streptococcus, gonococcus, meningococcus diplococci of Franckel, typhoid fever bacteria,
☐microbacteria of tuberculosis,
☐ fungi
Suppurative inflammation
☐The **exudation** contains dead neutrophils (suppurative corpuscles), lysed tissues and cells with mixture of lymphocytes, macrophages and erythrocytes
☐There are next morphological **types** of suppurative inflammation –

☐phlegmon and
☐abscess,
☐empyema.

Phlegmon

☐– is a vast suppurative infiltration, through which the exudation is distributed diffusely between tissue structures dividing them into layers.

☐There are **soft phlegmon** and **hard phlegmon** (with necrosis of tissues)

Suppurative inflammation

☐***Abcess*** – a local suppurative inflammation generating cavity filled with pus

☐***Empyema*** - the suppurative inflammation of hollow organs or serous cavities with pus accumulation

Abscess of lung.

☐Cavity filled by festering exudates is seen on a cut of lung. The wall of in is thickened, sclerosed, consequently it is ***chronic abscess.***

Chronic abscess of liver.

☐The cavity of abscess is partly filled with purulent masses, which are represented by dead neutrophiles, tissue detritus. The wall of abscess is represented by an internal pyogenic membrane, layer of granulative tissue and mature connective tissue. Well-kept hepatic parenchyma there is on periphery.

Flegmonously – ulcerous appendicitis.

☐Ulcers of mucousa are present.

☐The wall of appendix is along the whole length infiltrated by neutrophiles, swollen

consequences of suppurative inflammation

☐are resolution and

☐ scar formation.

☐The chronic suppurative inflammation causes amyloidosis of internal organs.

Hemorrhage inflammation

☐ is mainly acute.

☐***Aetiology****:*

☐-special danger infectious diseases (plague, anthrax, smallpox)

☐ - viral infections

☐The exudation contains erythrocyte that is why it has a rusty tint.

Hemorrhage inflammation

Catarrhal inflammation

☐is developed on mucous membranes.

☐*Aetiology:*

☐-infectious agents,

☐ -thermal and chemical agents,

☐-autointoxication,

☐- allergy.

Catarrhal inflammation

☐The exudation consists of mucus, cast-off epithelium and blood elements.

☐The inflammation has an <u>acute</u> or a <u>chronic</u> form

☐At first one there is a predominance of hypertrophy of mucous membrane (<u>hypertrophic catarrh</u>),

■at second one – atrophy and sclerosis (<u>atrophic catarrh</u>).

Mixt inflammation

☐Fibrinously-festering pericardisis Pericardium is rough covered by fibers of fibrin. It is “pilose heart”.

Productive (proliferative) inflammation

☐Three types of it are distinguished: interstitial, with formation of polypuses and pointed condiloms and granulematous.

☐There are **proliferation of cells** with formation of focal or diffuse infiltrates takes place in the area of damage: polymorphocellular, roundcellular (limphocytic-monocitic), macrophagal, epithelioid or plasmocellular

Interstitial inflammation

☐ is characterized by formation of cellular infiltrates in stroma of organ (interstitial myocarditis, interstitial pneumonia, interstitial nephrite).

☐It can be acute (rheumatism, glomerulonephritis)

☐and chronic diffuse sclerosis (cardiosclerosis).

Interstitial inflammation

Polypuses and pointed condyloms

☐is characterized by simultaneous drawing stroma and epithelium into inflammatory process.

☐**Polypuses** grow in that places, where glandular epithelium (stomach, intestine) is.

☐**Condyloms** are stratified flat epithelium on places near prismatic (anus, genitals) in reply to the permanent irritation at a gonorrhoea or syphilis proliferates, forming together with stroma protuberances

pointed condyloms

Granulematous inflammation

☐is the special form of productive inflammation

☐***Aetiology:***

☐ persistent irritant of organic or inorganic,

☐ often immune nature

☐***Morphology*** cellular accumulations (granulomas) of macrophages and their derivative.

☐***Phases*** of granulomas formation

☐-young mononuclear cells.

☐-Their transformation into macrophages.

☐-Forming of mature granuloma.

Granulematous inflammation

Granulematous inflammation

☐***Classification***

☐**Non-specific**		**Specific**
☐**Acute**	**Chronic**	**Tuberculosis**
☐**Enteric fever**	**Rheumatism**	**Syphilis**
☐ **(typhoid**		
☐**Spotted**	**Brucellosis**	**Leprosy**
☐**Rabies (hydrophobia**	**Tularemia**	**Glanders**
☐	**Sarcoidosis**	
☐		**Rhinoscleroma**

Granulematous inflammation

☐Macroscopically granulomas have sizes from barely perceptible by eye nodes to tumular formations (syphilis, tuberculosis). At presence of necrosis they are yellow, at its absence – grey. Granulomas are formed around vessels or alongside them. The damaged vascular wall and mesenchimal cells are the basic components of node.

☐At a number of diseases (tuberculosis, syphilis, scleroma, leprosy, glanders) granulomas assume specific structural cellular features. In such cases after the whole complex of specific morphologic features it is possible with a certain extent of authenticity to define etiology of disease. Such granulomas are named specific.

Specific granulomas at tuberculosis

☐Morphological signs:

☐presence of epithelioid cells,

☐lymphocytes,

☐single plasmocytes,

☐giant Pirogov-Langhans' cells,

☐necrosis in a center.

Miliary pulmonary tuberculosis.

☐Numerous nodes of white color of millet grain size are seen on dark red ground of section. White color of tuberculous granuloma is caused by caseous (caseation) necrosis presence in the center

Tuberculous granulomas of lung :

caseous necrosis in the center is represented with homogenous red nuclei-free mass, cellular bank is on periphery. In tuberculous gibbus granulation bank Pirogov-Langerhans' giant cells are seen.

Specific granulomas at syphilis

Morphological signs:

presence of epithelioid cells,

lymphocytes,

big number of plasmocytes,

giant Pirogov-Langhans' cells,

vasculitises and necrosis always develops around vessels.

necrosis in a center.

***Syphilitic mesaarthritis*.**

Inflammatory process is observed in aorta wall, which extends from intima side and adventitia on middle membrane. Lymphoid, plasmatic cells accumulation takes place.

Specific granulomas at leprosy

Morphological signs:

presence of epithelioid cells,

lymphocytes,

big number of plasmocytes,

giant Virhovs' cells whith clepsiella Gansen

fibroblasts.

Specific granulomas at glanders

Morphological signs :

presence of epithelioid cells,

neutrophiles and polymorphonuclear leucocytes

microabscesses,

necrosis with kariorrhexis,

granulative tissue.

Specific granulomas at rhinoscleroma

Morphological signs :

presence of epithelioid cells,

lymphocytes,

big number of plasmocytes,

giant Mikulichs' cells with foamy cytoplasm

hyaline spheres.

***Rhinoscleroma*.**

it is represented with vascular component and numerous cellular elements: aggregation of plasma cells with admixture of minor quantity of spindle-shaped epithelioid elements. Among these cells typical large cells with transparent cytoplasm are met, so called Mikulich' cells.

Thanks for your attention