ZAPOROZHZHIAN STATE MEDICAL UNIVERSITY

The department of pathological anatomy and forensic medicine with basis of law

Acute & chronic inflammation

Lecture on pathological anatomy

Inflammation

It is a local and biologically expedient reaction of organism as a reply on the damage.

Classic clinical signs of inflammation:

- Heat (calor).
- Redness (rubor).
- Edema (tumor).
- Pain (dolor).
- Loss of Function (functio laesa).

The agents causing inflammation:

- Physical agents (heat, cold, radiation, mechanical injury).
- Chemical agents (organic and inorganic poisons).
- Infective agents (bacteria, viruses, parasites).
- Immunological agents (cell-mediated and antigen-antibody reactions).

Principles of classification:

According to the duration of process:

- acute (during 2 weeks),
- subacute (more than 1month),
- chronic (months and years).

According to the reason of development:

a) banal (unspecific) – there are any factors of external environment: - physical agents
- toxic chemical agents
- microbiological agents
- immunological agents

b) specific - there are certain infections, such as tuberculosis, Syphilis & so on.

According to morphological features:

a) exudative,

b) proliferative (productive).

Pathogenesis:

Phases of inflammatory process:

1. Alteration
2. Exudation
3. Cell proliferation and renewal of the damaged tissue

The main components of acute and chronic inflammatory responses are:

- circulating cells and proteins,
- cells of blood vessels,
- cells and proteins of the extracellular matrix

I. Alteration

Alteration is the rough damage, degeneration and necrosis of vessel wall, mucous and serous membranes, selective damage of parenchyma by biological, physical or chemical factors.

II. Exudation

– it is a formation of exudate.

The major local manifestations of acute inflammation in the stage of exudation are:

1) Vascular dilation and increased blood flow (causing erythema and warmth),
2) extravasation and deposition of plasma fluid and proteins (edema),
3) leukocyte and blood cell emigration and accumulation in the site of injury,
4) phagocytosis
5) formation of exudation
6) formation of inflammatory infiltration.

Components of an exudate:

1. liquid part: plasma albumens (2-9%), water, ions.
2. cells of blood and immune cells (red corpuscles, leucocytes, monocytes)
3. tailings of the blasted tissues
4. bacteria, which cause inflammation.

III. Phase of cellular proliferation and renewal of the damaged tissue

1. The process begins from complete completion of mechanisms of damage, exudation and the action of all destroying mediators are repressed.
2. The stimulation of cambial and special cells (their reproduction) began – it is a reparative proliferation under constraint factors of growth of macrophages, thrombocytes, fibroblasts, lymphocytes, endothelium.
3. Renewal of tissue or organ architectonics. A process is regulated by hormones (architectonics) and immune cells.

According to prevailing one of these phases, inflammation is classified into exudative and proliferative inflammation.

Exudative inflammation usually develops as acute, proliferative inflammation – as chronic.

EXUDATIVE INFLAMMATION

It is characterized by predominance of vascular reaction and predominance of exudation (2-3 weeks)

CLASSIFICATION ON MORPHOLOGY OF EXUDATE (on a prevailing component):

1.Serous inflammation - a lot of liquid and albumens of plasma (3-8%)

Reasons of development:

- bacteria, chemical factors, physical factors

Localization: skin, serouse shells, mucous membranes (more rarely)

Outcomes:

favorable, renewal of tissues.

Morphology: erythema and swelling lead to mucosal edema with risk of stenosis.

2.Fibrinous inflammation

It is acute inflammation with exudation of fibrin on the mucous surface (oral, respiratory, bowel) and serous membranes.

Fibrinous exudate contains large amount of fibrin, neutrophils, and macrophages.

Reasons of development: bacteria, viruses, toxins

Localization: mucous, serous covers, lungs

Types of fibrinous inflammation:

A. Croupous inflammation – superficial alteration, tapes or filaments of fibrin loosely related to subject tissues, easily becomes separated from tissue without formation of ulcers and erosions.

B. Diphtheroid inflammation – it is characterized by the deep damage with the formation of the densely soldered tapes of tissue with appearance of ulcerous defects.

It appeared on the surfaces, covered by a squamous or intermediate epithelium.

Outcomes of fibrinous inflammation:

it is incomplete restoration with formation of joints and partial obliteration of cavity;

In tubular organs:

In cases of Diphteroid thracheitis fibrin is not protractedly tear away, scars appear after tearing away

In cases of Crupouse thracheitis fibrin is easily torn away and is cleared one's the throat; at children they close the road clearance of bronchyoles ☐ asphyxia.

3.Purulent inflammation

– is inflammation with exudate which consists primarily of neutrophils, cellular debris (fragments of the blasted tissue), bacteria and plasma albumens

Macroscopically it is yellow-green pus which covers the edges of wound.
Arises up under influencing of bacteria:
Staphylococci
Streptococci
Mushrooms

Localization of purulent inflammation: in any organs

A) an abscess is the local, limited hearth of festering inflammation.

Example: Pulmonary abscesses occur after pulmonary infarction or lobar pneumonia.

B) phlegmon is the diffuse unreserved festering inflammation;

Morphology: the exudate primarily consists of granulocytes and proteolytic serum components.

Purulent inflammation:

C) a carbuncle is inflammation of one hair follicle;
D) a furuncle is inflammation of group of hair follicles;

F) empyema – suppurative inflammation in a body‘s serous cavity or in a hollow organ.

Outcomes:

- Renewal of tissue
- Formation of fistula
- Formation of chronic abscess
- Bacteryemia
- Sepsis (great number of abscess)

4. Putrid inflammation

– putrid transformation of exudates with bed smelling.

Reason: Anaerobes, Escherichia coli, Proteus

Localization and **Outcomes** as the same as by festering inflammation

5. Catarrhal inflammation

is formation of exudates rich with mucus

Reason: Bacteria, viruses, temperature, physical and chemical factors

Localization: mucous membranes rich by serous-mucous glands.

Outcomes:

Renewal of structure of the damaged tissue

At the chronic flow ☐ atrophy

6. Hemorragic inflammation

The exudate is rich by red corpuscles.

Reasons: viruses, bacteria which cause the damage of endotelium and sharp increase of permeability of vascular wall (flu, plague, anthrax)

Outcomes: mortal because of exciter action

7. Mixed inflammation - combination of exudates

Acute inflammation may have one of four outcomes:

1. *Complete resolution.*
2. *Healing by connective tissue replacement (fibrosis).*
3. *Abscess formation*, which occurs particularly in infections with pyogenic organisms.
4. An acute inflammation that fails to heal may become *chronic inflammation*.

Proliferative (productive) inflammation.

Three major groups of reasons of development can be identified:

1. Persistent infections by certain intracellular microorganisms, such as tubercle bacilli and certain fungi.

   The inflammatory response often takes a specific pattern called a granulomatous reaction.

Proliferative (productive) inflammation.

2. Prolonged exposure to nondegradable inanimate material. For example: the silica particles, which after being inhaled for a prolonged period set up a chronic inflammatory response in the lungs. It is called silicosis.
3. Under certain conditions, immune reactions are set up against the individual's own tissues, leading to autoimmune diseases. In these diseases, autoantigens evoke a self-perpetuating immune reaction that results in several important chronic inflammatory diseases, such as rheumatoid

Proliferative (productive) inflammation

is characterized by formation of infiltrates consists of:

1. mesenchymal cells (endothelia, fibroblasts, cambial cells),
2. immune cells (T and B-cells, plazmocytes, monocytes cells),
3. cells of blood.

Classification of infiltrates:

On prevalence:

a) hearth,

b) diffuse.

On localization:

a) around-vessels,

b) periductal,

c) interstitial (in stroma of organ between the specialized structures),

d) around the areas of necrosis.

Originally:

a) banal – caused by viruses, fungi, simplest, soluble toxins, foreign bodies,

b) specific.

Forms of banal proliferate inflammation.

- Formation of polyps and pointed condylomas.
- Interstitial inflammation.
- Granulomatous inflammation.

It is localized: in additional sinuses and mucous membrane of nose, from the protracted inflammatory reaction

Morphology of polypus:

It is proliferation of vascular-mesenchymal components and epithelium in reply to the irritation. The vascular leg and components of stroma of normal mucous membrane is formed.

It is necessary to distinguish inflammatory polyps from tumors, as tumor polyps are excrescence of tumor cells.

Outcomes: regression after the removal of reason.

Reasons: viruses, bacteria (pale treponema), papilloma-virus.

Localization: on the border of mucous membrane and skin (nasal, cervical, colorectal polyps are common).

Outcomes:

1. regression,
2. transformation in a malignant new formation (papilloma-virus).

Morphology of Pointed condylomas:

Localization: on the genital organs or the perineal area.

Condyloma is the growth of squamous cell epithelium and connective tissue of the skin with appearance of numerous small papillae on the surface. In stroma there are dilated vessels, infiltrates of lymphocytes and plasma cells with admixture of leukocytes.
Reasons: viruses, the most frequent Papillomaviruses (also have a carcinogenic effect).
Outcomes:
1. regression,
2. transformation in a malignant tumor.

Interstitial inflammation

is appearance in organs stroma of inflammatory immune-cellular infiltrates.

Localization: stroma of myocardium, liver, kidney, pancreas

Reasons: fixed on basal membranes viruses, toxins, medicines, autoantigens.

Morphology: lymphocytes, plazmocytes, macrophages, eosinophils prevails, mast cells, neutrophils (at the beginning of process).

Lymphocytes and macrophages, fibroblasts and fibrocytes prevails then.

Outcomes:

complete renewal of organs

tissues (viral diseases),

diffuse sclerosis,

cirrhosis of organ.

Granulomatous inflammation

is formation in tissues of small knots by a diameter from 1 to 5 sm.

Localization of granuloma:

around microvessels,

in stroma of organs,

in parenchyma.

Morphological types of granulomas:

1. granulomas from the immune damage of infection, that activates the immune system (viruses, rickettsia, fungi, bacteria),
2. granuloma of foreign bodies – at presence of foreign bodies in tissue (dust, stitch material). Foreign bodies are surrounded by macrophages and are formed granuloma.

Granulomatous inflammation

Stages of forming of granuloma:

- damage of tissue and migration of lymphocytes and monocytes, which are transformed in macrophages,
- epithelioid cells (transformed monocytes are macrophages of secretor type) surround the areas of necrosis,
- many-nuclear macrophages which phagocytized necrotic tissues.

Granuloma Cells:

- Macrophages
- Epithelioid cells
- Multinucleated giant cells
- Necrosis may be a feature of some granulomas
- Fibrosis

Specific inflammation

It is proliferative-granulomatous inflammation which is caused by:

- mycobacterium tuberculosis,
- mycobacterium lepry,
- pale treponema (Syphilis),
- the Volkovicz-Frish's or klebsyela stick (rhinoscleroma).

Morphology of tuberculous granuloma:

- caseous necrosis centrally,
- domination of epithelioid cells and presence of Langhans' giant cells,
- vessels are absent (or very small amount of capillaries),
- miliary and multiple,
- outcome is soft sclerosis.

Morphology of syphilis granuloma:

- caseous necrosis centrally,
- domination of lymphocytes and plasmocytes,
- large amount of capillaries,
- solitary,
- outcome is gross sclerosis.

Specific inflammation

Flow: protracted with the periods of intensification (necrotic granulomas) and periods of fading of process (epithelioid-macrophage's granulomas).

Outcomes: scars, deformations of organs, organ insufficiency.