Inflammation (lat. - Inflammatio)

a typical pathological process that occurs in response to the influence of a pathological factor, aimed at delineating and eliminating the damaging agent, as well as tissue restoration, accompanied by the phenomena of alteration, exudation and proliferation.

ETIOLOGY

Any damaging agent that exceeds the adaptive capacity of the tissue in strength and duration can cause inflammation.

All factors are divided into exogenous and endogenous.

Exogenous include microorganisms (bacteria, viruses, fungi); animal organisms (protozoa, worms, insects), chemicals (acids, alkalis), mechanical (foreign bodies, trauma) and thermal effects (cold, heat), radiation energy (x-rays, radioactive, ultraviolet rays).

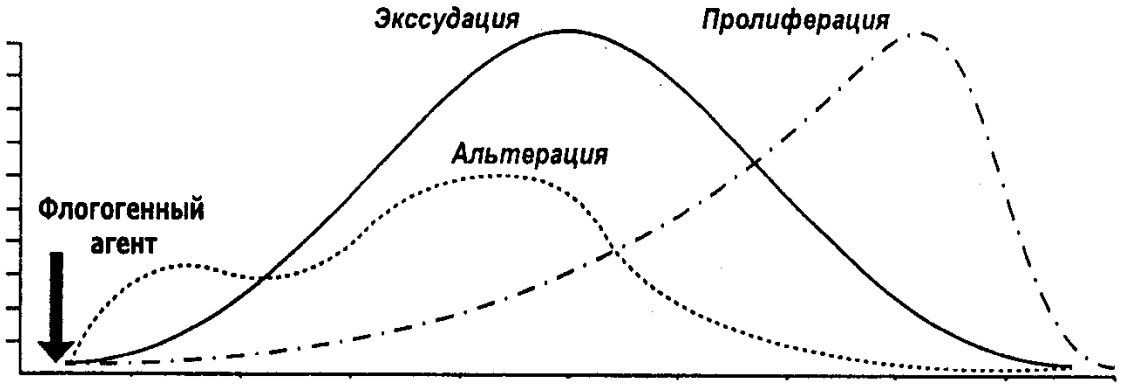
Endogenous factors include those that occur in the body itself as a result of a different disease - a tumor, bile or urinary stones, or thrombosis.

PATHOGENESIS

Inflammation consists of the following components:

I. Alterations – alteratio
II. Exudations – exsudatio
III. Proliferation – proliferatio

The components of inflammation are closely interrelated, complement each other and pass into each other, there are no clear boundaries between them.



The ratio of the components of inflammation

ALTERATION

- the general name of changes in cells, tissues, and organs that are accompanied by a violation of their vital functions.

Alteration is the first and direct consequence of the action of the damaging factor and the initial link in the mechanism of inflammation development.

There are primary and secondary alterations.

Primary alteration is caused by direct contact of the damaging factor with the tissue.

Secondary alterations (self-harm) as a component of inflammation, it is characterized by a change in:

1. structure;
2. metabolism;
3. physical and chemical properties;
4. spectrum of biologically active substances (formation of inflammatory mediators);
5. the functions of the damaged tissue.

Physical and chemical changes in the focus of inflammation

1. Acidosis - due to the accumulation of excess of under-oxidized compounds in the focus of inflammation, violation of their outflow, as well as depletion of alkaline buffer systems.
2. Hyperosmia – an increase in osmotic pressure in the focus of inflammation as a result of the release of intracellular potassium during primary alteration, an increase in the number of H+ions.

3. Hyperonkiya - an increase in oncotic pressure in the focus of inflammation, due to increased hydrolysis of macromolecules and the accumulation of polypeptides and other oncotically active compounds in the focus of inflammation.
4. Reduced surface charge and electrical potentials of cells as a result of damage to cell membranes and ion imbalance in extracellular fluid.

During alterations, the release of biological active substances – inflammatory mediators-occurs.

This is the trigger of inflammation, which determines the development of an inflammatory response.

Inflammatory mediators can be of plasma (humoral) and cellular (tissue) origin.

Mediators of plasma origin (humoral) are found in liquid media – blood plasma and tissue fluid (bradykinin, plasmin).

Mediators of cellular origin are associated with effector cells – mast cells, basophilic leukocytes, platelets.

They are divided into 2 groups:

- preformed,
- newly formed.

Preformed cell mediators are constantly synthesized in cells and, if necessary, are immediately released into the focus of inflammation.

These include:

- histamine,
- serotonin.

Due to their rapid release, these substances change the lumen of microvessels and provide initial microcirculatory disorders in the focus of acute inflammation.

Newly formed cell mediators are produced by white blood cells when they are stimulated.

These include:

- prostaglandins,
- tumor necrosis factor - α,
- lysosomal enzymes,
- active oxygen metabolites,
- cationic proteins,
- catecholamines,
- acetylcholine,
- nitric oxide.

EFFECTS OF MEDIATORS

- increase the permeability of microvessels,
- stimulate phagocytosis,
- they have a bactericidal effect,
- cause secondary alterations,
- include immune mechanisms in the inflammatory response,
- regulate the proliferation and differentiation of cells in the focus of inflammation.

ANTI-INFLAMMATORY AGENTS

With the development of inflammation, substances are released that prevent excessive accumulation and entry into the bloodstream of mediators – anti-inflammatory mediators.

Anti-inflammatory mediators neutralize mediators by reducing their formation, release from cells, binding or destruction.

Among anti-mediators, the most important place is occupied by enzymes that destroy inflammatory mediators:

- histaminase that destroys histamine;
- carboxypeptidase, causing the breakdown of kinins;
- esterase inhibitory components of the complement;
- prostaglandin dehydrogenase, which destroys prostaglandins.

Antimediatory activity have glucocorticoids:

stabilize cell membranes,

reduce the production of kinins and prostaglandins,

weaken the secondary alteration of the vascular reaction,

inhibit proliferation.

EXUDATION

- the process of moving protein-rich fluid, often containing shaped blood elements, from small veins and capillaries to the surrounding inflamed tissues and body cavities.

This manifestation quickly follows the alteration and release of mediators.

Mechanisms of exudation - exudation of blood components (water, proteins, electrolytes) beyond the vascular wall:

1. increased vascular wall permeability;
2. increased hydrostatic pressure in the vessel;
3. increased oncotic pressure in the focus of inflammation;
4. increased osmotic pressure in the focus of inflammation.

VASCULAR REACTIONS IN INFLAMMATION

1. vascular spasm
2. arterial hyperemia
3. venous hyperemia
4. stasis

Mechanism of vascular spasm

Occurs during alteration as a result of the release of substances with a vasoconstrictive effect.

Mechanisms of arterial hyperemia

I. Neurogenic

-increased release of acetylcholine by parasympathetic nerve endings;

-increased sensitivity of cholinoreceptors to acetylcholine in conditions of excess extracellular content of K+ and H+ ions.

II. Humoral

-vasodilatation under the influence of inflammatory mediators.

III. Reduction of basal tone of arterioles.

MECHANISMS OF VENOUS HYPEREMIA

Occurs in the dilated venules and postcapillaries in the slow motion flow of blood.

The transition of arterial hyperemia to venous hyperemia is associated with several mechanisms.

I. changes in the rheological properties of blood and its circulation:

- marginal standing of white blood cells;
- increased blood viscosity due to its thickening due to exudation, loss of albumins, increased globulin content, changes in the colloidal state of proteins;
- swelling and aggregation of red blood cells;
- thrombosis (due to activation of the blood clotting system), leading to narrowing of the lumen of the venules.

II. changes in the vascular wall:

- loss of vascular tone due to paralysis of the neuromuscular system of blood vessels;
- reduced elasticity of the vascular wall;
- swelling of the endothelium and increasing its adhesiveness, as a result of which the vascular lumen narrows, conditions are created for the adhesion of white blood cells to the endothelium.

III. Tissue changes:

- compression of venules and lymphatic vessels with exudate;
- reduced elasticity of connective tissue.

White blood cell emigration

This is the process of leaving white blood cells from the lumen of blood vessels to the focus of inflammation in the tissues.

Emigration consists of a series of consecutive events:

marginal standing (margin) of white blood cells,

adhesion,

penetration of white blood cells through the microvessel wall (leukodiapedesis),

directed movement of white blood cells in tissues to the focus of inflammation.

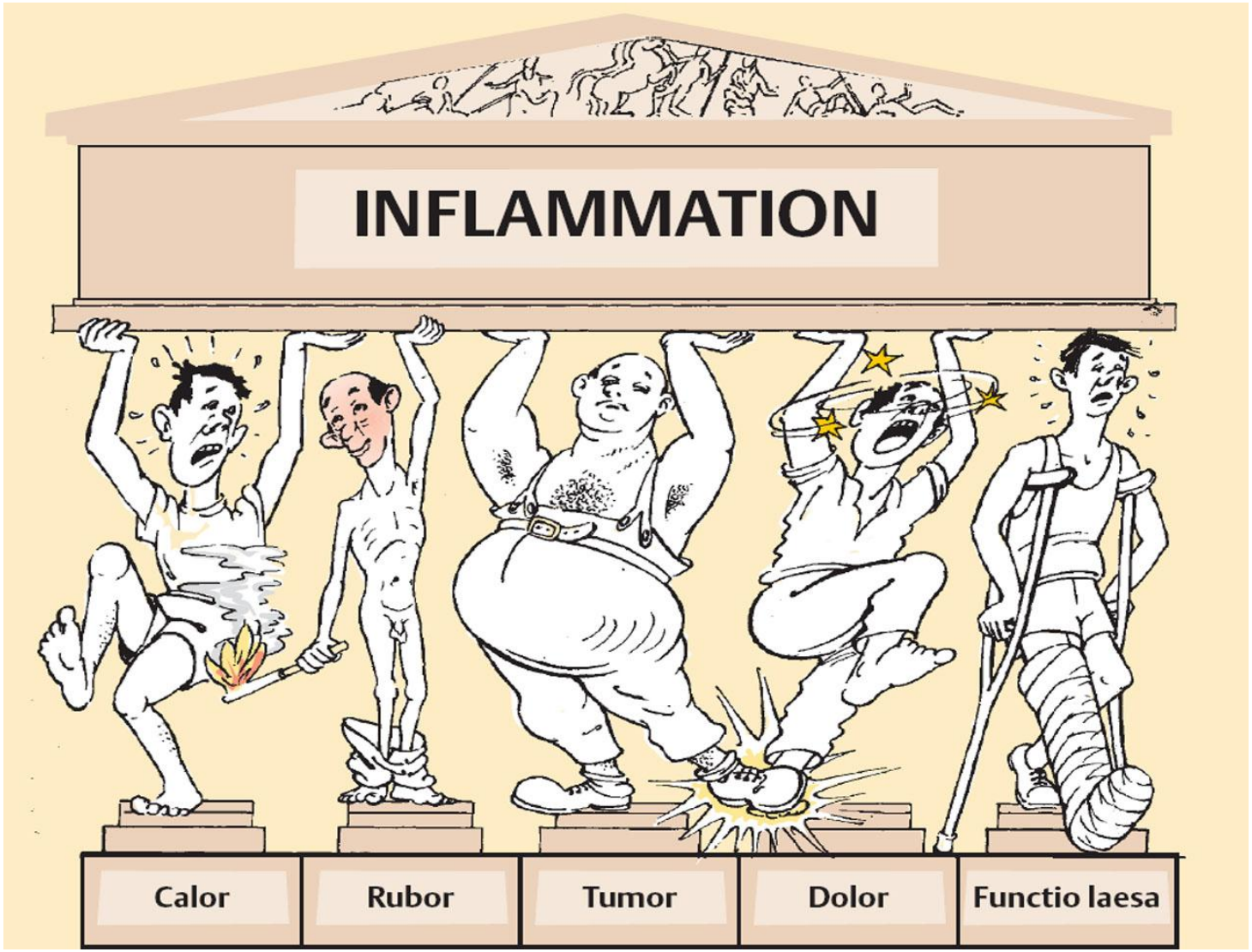
Formation of exudate and cellular infiltrate

Completes the exudation processes described above. Effusion of liquid parts of the blood, emigration of white blood cells, diapedesis of red blood cells lead to the appearance of inflammatory fluid – exudate in the affected tissues or body cavities.

PROLIFERATION

- growth of tissue of the organism through the reproductive cells.

It is the final component of inflammation aimed at restoring damaged tissue.


INFLAMMATION
Calor
Rubor
Tumor
Dolor
Functio laesa

LOCAL SIGNS OF INFLAMMATION

Inflammatory arterial hyperemia causes an increase in temperature (calor) and redness (rubor) of the inflamed area.

The accumulation of fluid in the tissues causes- increase in volume (tumor),

to compression of the nerve endings and pain (dolor), whose appearance in inflammation associated with exposure mediators (bradykinin, histamine, serotonin, certain prostaglandins and leukotrienes), the accumulation of ions K+ and H+,to dysfunction of the tissue or organ (functio laesa).

QUESTIONS FOR SELF-CONTROL OF KNOWLEDGE

1 .To give a definition of inflammation.

2. How to prove the involvement of the nervous system in the mechanism of inflammation?

3. What are the features of the course of inflammation in animals that are in a state of winterhibernation.

4. How to determine the involvement of the nervous and humoral systems in the development of inflammation?

5. How will inflammation occur with hyperfunction of the thyroid gland?

6. What are inflammatory mediators?

7. What is the biological role of exudation?

8. What is the role of biologically active substances in the mechanism of disordersblood circulation?

9. What is expressed in the physical-chemical violations in the inflammation?

10. does any inflammation have all 5 signs of inflammation?

11. What are the main pathogenetic factors in the development of arterial and venous hyperemia in inflammation.

12. What are the signs of inflammation of internal organs?

Tasks

1. Will the course of the inflammatory process in desympathizationa focus of inflammation? Give an explanation.
2. What are the signs that can detect an inflammatory process in lungs?
3. A lethal dose of poison was administered to an experimental animal in the focus of acute inflammation and to a control animal in a similar area of the skin. Which animal will die first and why?
4. acute inflammation was reproduced on the skin of the rabbit's ear, after which it was intravenously injected with a 1% solution of trypan blue. Which ear was more brightly colored in blue: healthy or inflamed? Explain the answer.
5. is the same intensity of the inflammatory response in animals if one of them was previously administered large doses of hydrocortisone for one week, and the other – aldosterone? Give an explanation.

6. a nursing mother had breast pain two weeks after giving birth, milk production noticeably decreased, and her body Temperature rose to 39^0 C. In the gland, a sharply painful dense formation measuring 5x5 cm is palpated. Redness of the skin with increased venous pattern is noted above the focal seal. Enlarged axillary lymph nodes. According to the blood test: the concentration of white blood cells – 12.4 G/l. Name the local and general signs of the inflammatory process.

7. Patient for diagnostic purposes was carried out puncture of the abdominal cavity (paracentesis). The study obtained a turbid yellow-green liquid with a specific gravity of 1029 and a protein content of 0.39 g/l. A significant number of shaped elements were found in the sediment, among which neutrophils with signs of degeneration predominate, as well as microbial flora located intra- and extracellularly. What is the nature of the fluid obtained during the puncture? What are the mechanisms that lead to fluid accumulation in the abdominal cavity?