Manifestations of inflammation

Macroscopic manifestations of inflammation

Four so-called Celsus signs are derived from the macroscopic manifestations of skin inflammation , to which Virchow added a fifth:

1. **rubor** – redness of the inflammatory focus due to hyperemia;
2. **calor** – heating of the inflammatory focus;
3. **dolor** – soreness of the inflammatory focus due to irritation of peripheral nerve endings by acidic pH and cytokines ;
4. **tumor** – swelling of the inflammatory focus due to the expansion of capillaries and inflammatory edema ;
5. **functio laesa** – attenuation or, on the contrary, increased function (e.g. hypersecretion of mucus by an inflamed mucous membrane).

General manifestations of inflammation

1. Elevated temperature;
2. leukocytosis ;
3. increased sedimentation ;
4. increase in acute phase protein levels . [✎ edit embedded article]

Microscopic manifestations of inflammation

Alterations

By the term alteration we mean **regressive changes during** inflammation . From simple metabolic disorders (dystrophy) to necrosis of various extents.

The causes of alteration can be threefold:

- directly by the impact of the pollutant;
- products of microorganisms or their penetration into cells with subsequent cytolysis;
- an immune response directed against the attacked cells.

Inflammatory changes can also spread to the vessels supplying the inflammatory focus and a secondary alteration occurs - ischemic necrosis of the entire tissue area.

Exudation

Leakage of fluids and proteins from vessels damaged by inflammation (exudate - inflammatory effusion, fluid rich in proteins). Later, the output of blood cells that infiltrate the surrounding tissues (so-called inflammatory cellularization) is associated.

The basis of the exudation is peristatic hyperemia , which is conditioned by dilation of capillaries (dilation of blood vessels is caused by direct damage to the tissue, and by chemical substances released secondarily from the inflamed tissue). In peristatic hyperemia, the capillaries are maximally dilated, but the supply arterioles are relatively narrow. The blood flow in the capillaries slows down, the axial flow of erythrocytes slows down and fills the entire vessel (under normal circumstances, erythrocytes flow through the center of the capillary, and plasma with isolated leukocytes flows on the periphery). The blood flow can then stop temporarily or permanently.

Furthermore, there is **a change in the permeability of the capillary wall** (formation of slits between the endothelium). An increased amount of fluid (inflammatory edema), proteins (depending on their size – first albumins, then globulins and finally fibrinogen, which immediately clots into fibrin outside the vessel) and finally cells, emerge from the vessel.

Inflammatory exudate

Fluid containing proteins and cells. The basic types are:

- serous - little fibrin,
- fibrinous – a lot of fibrinogen that clots into fibrin,
- purulent - the amount of polynuclear,
- hemorrhagic - abundance of erythrocytes,

Another of its components are biologically active substances, so-called chemical mediators of inflammation (cytokines, components of complement, kallikrein system, hemocoagulation cascade).

Inflammatory infiltrate

Blood cells first accumulate on the wall of the capillary (margination) or fill its entire lumen (leukostasis), later they are forced out by amoeboid movement (leukodiapedesis). Neutrophil granulocytes appear first , then macrophages and lastly lymphocytes.

Components of the inflammatory infiltrate:

Neutrophil granulocytes (polynuclear)

In the place of inflammation within a few minutes to hours. Chemokines are attracted to the site of inflammation, and they themselves release a number of pro-inflammatory mediators. They mainly have a phagocytic function (they phagocytose smaller bacteria, especially pyogenic cocci - they are therefore called microphages). With their enzymes, they liquefy fibrin and necrotic tissue (however, they only work in an alkaline environment). In the inflammatory focus, the effect of peristatic hyperemia is a lack of oxygen - acidic metabolites (lactate) accumulate . Neutrophils thus undergo regressive changes (steatosis - gives the pus a yellowish color) and soon die.

Macrophages (histiocytes)

In the place of inflammation in a few hours to days. They arise from peripheral blood monocytes. Their basic property is phagocytosis and breakdown of fibrin, necrotic tissue elements, etc. (their proteases are effective in the acidic environment of inflammation). During phagocytosis, they change their appearance significantly (lipophages, siderophages, granule cells, giant cells from foreign bodies, ...). They also serve as APCs presenting parts of ingested antigens to lymphocytes. They also produce mediators inducing proliferation of lymphocytes, capillaries and fibrous cells, increasing temperature and inducing leukocytosis.

Lymphocytes

They appear at the latest (with inflammations of viral etiology even in the initial stages!). In addition to exudated lymphocytes from peripheral blood, lymphocytes from regional nodes are also involved in inflammation. They differentiate into T-lymphocytes (acting on macrophages and other lymphocytes) and B-lymphocytes , whose final effectors are immunoglobulin-producing plasma cells. They occur mainly perivascularly in chronic inflammation and their cytoplasm is filled with eosinophilic Russel bodies (corresponding to the accumulation of immunoglobulins in the GER cisterns). Histiocytes, lymphocytes and plasma cells together form the so-called **round cell (mononuclear, lymphoplasmacytic) infiltrate** .

Eosinophilic granulocytes

They appear quite late in the exudate and their main functions are phagocytosis of immune complexes and participation in allergic and parasitic diseases (they secrete substances, especially basic protein, which is toxic to parasites but also to epithelia).

Basophilic granulocytes, heparinocytes (mast cells, mast cells)

It serves as a source of heparin and especially histamine and serotonin (early mediators of inflammation, causing vasodilation and increased capillary permeability).

Platelets, endothelium, fibroblasts, erythrocytes.

Immune events

Non-specific immunity

- phagocytosis – **phagocytes** (neutrophils, eosinophils, macrophages);
- **complement**- mediated osmotic lysis ;
- antibody-mediated cytotoxicity (ADCC) - non-specific cytotoxic reaction - NK-cells**NK-cells** .

Specific immunity

- production of antibodies - **plasma cells** ;
- help **macrophages** (TH1-type immune response);
- help **B lymphocytes** (TH2 type immune response);
- **cell-mediated cytotoxicity** – specific cytotoxic reaction – TC;
- suppression of the immune response - TS.

Proliferation

Changes **characterized by the proliferation of tissue** (proliferation) **and the creation of new tissue tissue** (fibroproduction) are a manifestation **of reparation** (ie, proliferative changes are greater, the greater the alterative component!).

The basic form of reparation is **the formation of non-specific granulation tissue** with the formation of a scar. In the sparse intercellular mass with a small amount of collagen fibers and a large amount of blood capillaries, fibroblasts multiply and climb along the fibrin fibers and produce collagen. Budding of capillaries (formation of small lateral buds of the endothelium that gradually luminesce) creates new capillaries. This newly formed connective tissue with blood vessels is referred to as non-specific granulation tissue (in case of ulcerative inflammation, when looking from above at the exposed inflammatory newly formed tissue of the base of the ulcer, its surface is slightly granular (granulated) and bright red. The individual red grains correspond to loops of capillaries). In the further course, the granulation tissue fades, becomes stiffer and firmer, vessels decrease,

collagen fibers increase, cellularity decreases (fibroblasts turn into fibrocytes, or some of them disappear) and a hyaline transformation often occurs - it formsa scar (*cicatrix*). Similar changes occur during the organization of fibrin masses, e.g. in a hematoma or thrombus.

Another form of productive changes is chronic inflammation without **the formation of granulation tissue** , in which macrophages and lymphocytes are found in the inflammatory focus. They produce substances (mainly macrophages) causing tissue proliferation (fibrosis). This mechanism is used, for example, in liver cirrhosis and pulmonary fibrosis.

A special form of productive changes is **the formation of specific granulation tissue** in the form of epithelioid granulomas, which are formed *by modified macrophages* (epithelioid and Langhans cells) and, unlike non-specific granulation tissue, **are avascular .**

[✎ edit embedded article]

Links

related articles

- Macroscopic manifestations of inflammation
- Microscopic manifestations of inflammation

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