Microscopic manifestations of inflammation

Alteration

By 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 action of pollutants;
- products of microorganisms or their penetration into cells with subsequent cytolysis;
- immune response directed against infected cells.

Inflammatory changes can also progress to the vessels supplying the inflammatory lesion, creating a secondary alteration - Ischemia necrosis of the entire tissue area.

Exudation

Leakage of fluids and Protein from blood vessels damaged by inflammation (exudate - inflammatory effusion, protein-rich fluid). Later, the output of blood cells is associated, which infiltrate the surrounding tissues (so-called inflammatory cellulite).

The basis of exudation is peristatic hyperemia, which is caused by dilatation of capillaries (dilation of blood vessels is caused by direct tissue damage and chemicals released secondarily from inflammatory tissue). In peristatic hyperemia, the capillaries are dilated to the greatest extent, 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 whole vessel (normally erythrocytes flow through the center of the capillary and plasma with isolated leukocytes flows in the periphery). The blood flow may then stop temporarily or permanently.

Furthermore, there is a c**hange in the permeability of the capillary wall** (the formation of crevices between the endothelium). An increased amount of fluid (inflammatory edema), proteins (according to their size - first albumins, then globulins and finally fibrinogen, which immediately clots into fibrin outside the vessel) and finally cells come out of the blood vessel.

Inflammatory exudate

Fluid containing proteins and cells. The basic types are:

- serous - low fibrin,
- fibrinous - a lot of fibrinogen that clots on fibrin,
- purulent - a number of polynuclear cells,
- hemorrhagic - abundance of erythrocytes,

Its other components are biologically active substances, so-called chemical mediators of inflammation (cytokines, components of complement, kallikrein system, hemocoagulation cascades).

Inflammatory infiltrate

Blood cells first accumulate on the capillary wall (marginalization) or fill its entire lumen (leukostasis), later they are pushed out by amoeboid movement (leukodiapedesis). Neutrophilic granulocytes appear first, then macrophages, and finally lymphocytes.

Components of inflammatory infiltrate: Neutrophilic granulocytes (polynuclear cells) At the site of inflammation within minutes to hours. Chemokines are attracted to the site of inflammation and release a number of pro-inflammatory mediators themselves. They mainly have the function phagocytic (they phagocytose smaller bacteria, especially pyogenic cocci - they are therefore referred to as microphages). They use their enzymes to liquefy fibrin and necrotic tissue (but they only work in an alkaline environment). Due to peristatic hyperemia, there is a lack of oxygen in the inflammatory area - acidic metabolites (lactate) accumulate. Neutrophils thus undergo regressive changes (steatosis - gives the pus a yellowish color) and soon die out.

Macrophages (histiocytes)

At the site of inflammation in a few hours to days. They arise from peripheral blood monocytes. Their basic properties are phagocytosis and degradation of fibrin, necrotic tissue elements, etc. (their proteases are effective in the acidic environment of inflammation). During phagocytosis, it changes its appearance significantly (lipophages, siderophages, granule cells, giant cells from foreign bodies,...). It also serves as an APC presenting portions of absorbed antigens to lymphocytes. They also produce mediators that induce the proliferation of lymphocytes, capillaries and fibrous cells, increase temperature and induce leukocytosis.

Lymphocytes

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

Eosinophilic granulocytes

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

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);
- osmotic lysis mediated by **complement**;
- antibody-mediated cytotoxicity (ADCC) - non-specific cytotoxic response - **NK-cells**.

Specific immunity

- antibody production - **plasma cells**;
- macrophage help (TH1-type immune response);
- help **B-lymphocytes** (TH2-type immune reactions);
- **cell-mediated cytotoxicity**- specific cytotoxic response - TC;
- suppression of the immune response - TS.

Proliferation

Changes **characterized by proliferation of the ligament** (proliferation) and **the formation of new connective tissue** (fibroproduction) are a manifestation **of repair** (i.e. the greater the alterative component, the greater the proliferative changes!).

The basic form of repair is the **formation of non-specific granulation tissue** with scarring. In a sparse intercellular mass with a small amount of collagen fibers and a large number of blood capillaries, fibroblasts multiply which climb along the fibrin fibers and produce collagen. Capillary budding (formation of small lateral buds of the endothelium, which gradually lumines) creates new capillaries. This newly formed connective tissue with blood vessels is referred to as non-specific granulation tissue (in ulcerative inflammation, when viewed from above the exposed inflammatory newly formed tissue of the ulcer base, its surface is slightly granular and bright red. Individual red grains correspond to capillary loops).

In the further course, the granulation tissue fades, becomes stiffer and firmer, blood vessels shrink, collagen fibers increase, cellularity decreases (fibroblasts turn into fibrocytes, or some of them disappear) and often there is a hyaline transformation - a **"scar" (*cicatrix*).** Similar changes occur in the organization of fibrin masses, such as the hematoma or thrombus.

Another form of productive changes are chronic inflammations without the formation of "granulation tissue", in which macrophages and lymphocytes are mainly present in the inflammatory locus. They produce substances (especially macrophages) that cause an increase in connective tissue (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.**

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