THE IMMUNE SYSTEM IN BURN INJURY

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Currently, three phases are identified in the course of burn injury: the phase of immunosuppression, the phase of compensation, and in cases of severe burns, the phase of late immunosuppression. In cases of relatively mild burns, after the first phase of immunosuppression, a compensatory period follows, characterized by the normalization of immunological reactivity. Hematopoiesis of stem cells in the bone marrow and their migratory activity are enhanced, myelopoiesis is restored, and the number of B- and T-lymphocytes, including T-helper cells, increases. Concurrently, the suppressor activity of various cellular populations decreases, while the antigen-presenting and phagocytic functions of innate immune cells are enhanced [5,6,9].

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The use of laboratory methods to study both cellular and humoral immune deficiencies can serve as significant diagnostic and prognostic criteria for the severity of burn injury.

The consequences of burn injury primarily affect keratinocytes, leading to the activation of receptor complexes on their surface. The influence of pro-inflammatory cytokines-IL-1, IL-6, TNF, as well as IFN-y, IL-4, and IL-17-allows for antigen presentation by keratinocytes, which do not possess this function in their inactive state. The activation of keratinocytes increases the number of receptors for adhesion molecules on their surface, facilitating contact with immune cells. Epithelial cells acquire antigen-presenting properties through the expression of CD80, CD86, and CD40. Additionally, the production of cytokines like IL-1, TNF, and IL-6 by keratinocytes attracts granulocytes, macrophages, and lymphocytes to the wound area [11,12,13].

Langerhans cells (LC) are the primary antigen-presenting population of intraepidermal macrophage-like cells and express HLA class I and II molecules. Dendritic cells (DC) play a role in regulating antigen-presenting functions and in

activating CD4+ T-lymphocytes and cytokine production via Toll-like receptors.

Directly in the zone of injury after burn, neutrophils produce enzymes into the extracellular environment, disrupting the intercellular matrix and destroying necrotic tissues. Due to the depression of the cytotoxic function of macrophages, patients with burn injuries show an escalation of natural cytotoxicity levels in granulocytes.

In the first day after a burn injury, there is a disruption of phagocyte chemotaxis associated with the disturbance of polymerization and depolymerization of actin filaments, suppressing their migration, which may be considered an unfavorable predictor of infectious complications. This process gradually peaks on days 3-5 and correlates with the extent of the damage [8,9,10].

Macrophages phagocytize antimicrobial components from neutrophils, such as myeloperoxidase and cationic proteins. They play a central role in regulating the transition from the vascular-exudative stage to the reparative stage. Macrophages are classified as resident (tissue) and recruited, regulating immune responses. Activated macrophages are represented by three subpopulations with specific functions. M1 macrophages synthesize pro-inflammatory cytokines and exhibit phagocytic activity. In contrast, the M2 subpopulation synthesizes large amounts of the anti-inflammatory cytokine IL-10 and participates in phagocytosis, angiogenesis, reparative, and immunomodulatory reactions. Mox macrophages express COX-2 and IL-1, participating in antioxidant protection. It is also known that the phagocytic activity of the Mox subpopulation is significantly lower compared to M1 and M2. There is evidence of M1 macrophages transitioning to reparative M2 macrophages due to adhesion to the extracellular matrix.

As a result of burn injury, neuro-sensory peptides are released from the peptide-dergic nerve fibers involved in nociception, stimulating cytokine production in macrophages. The activity of macrophages in burn wounds largely determines the development of immune dysfunction, influenced by the modulation of immune response types (Th1 and Th2) and changes in the activity of YS T-lymphocytes [7,8,9].

T-cells participate in reparative processes by releasing mediators from T-helper cells (CD3+CD4+) that activate macrophages.

Platelets also influence tissue repair processes in burn injuries by forming a hemostatic plug and releasing platelet-derived growth factor (PDGF), attracting and activating macrophages and fibroblasts.

In addition to innate immunity, adaptive immunity plays a significant role in the pathogenesis of immune reactivity in burn injuries. The functioning of all immune components is closely interconnected, sustaining the inflammatory process.

In the human epidermis, lymphocytes account for 1-4% of the total number. The majority are T-lymphocytes, while B-lymphocytes are found in the middle and deep layers of the dermis [4,5,6].

In the first days following severe burn injuries, there is significant suppression of the cellular immune component, along with dystrophic changes in the thymus. The total number of T-lymphocytes and their subpopulations decreases, altering functional activity, which correlates with the severity of the burn.

In the initial days post-burn, there is a reduction in the number of cells expressing CD2, CD4, and CD8 antigens, with T-helper cells (CD3+CD4+) decreasing more significantly than cytotoxic T-cells (CD3+CD8+), leading to a nearly threefold reduction in the CD4/CD8 ratio.

During the acute period of burn injury, there is an increase in the expression of low-affinity receptors for IL-2 (CD25) and transferrin receptors (CD71) by lymphocytes [1,2,3].

The literature describes two theories of T-lymphocyte dysfunction in burn injuries. One pathway suggests the suppression of T-lymphocyte activity due to increased concentrations of HMGB1 (high mobility group box-1) in plasma, which is inversely proportional to the ratio of T-helper to cytotoxic T-lymphocytes. The second pathway recognizes T-cell function depression due to an increase in the anti-inflammatory component of the cytokine balance.

The B-cell component is more resilient during burn injury. On days 1-3 post-burn, a decrease in IgM and IgA levels is observed, while in cases of deep burns, there is an increase in IgG levels. This process during burns is influenced by plasma loss and the death of B-lymphocytes due to proteolytic enzymes. There is evidence of a direct dependence of increased IgM and IgG levels on the extent of burn damage [20].

Given the significant role of the immune system throughout burn injury and skin defect healing, it is advisable to conduct laboratory evaluations of immunological parameters. Understanding the mechanisms by which the immune system regulates the processes of burn injury and skin defect restoration will help optimize the therapy for these patients in the pre-, intra-, and postoperative periods.

The most clinically significant parameters of immune status include the subpopulational composition of lymphocytes, encompassing the analysis of regulatory and effector subpopulations, activation markers, and pro-inflammatory and anti-inflammatory cytokines. According to several authors, studying the levels of matrix metalloproteinases and assessing fibroblast activity is the most informative for characterizing the processes of tissue repair [17,18,19].

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functional activity, which correlates with the severity of the burn [14,15,16].

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