IMMUNOLOGICAL FACTORS IN THE PATHOGENESIS OF DEVELOPMENT OF DESTRUCTIVE PNEUMONIA IN CHILDREN

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Annotation. The paper presents the results of a review of the literature on the pathogenesis of the development of destructive forms of pneumonia in children, taking into account immunological changes in the body.

Keywords: pneumonia, bacterial destruction of the lungs, cellular and humoral immunity, cytokines

It is known that the phenomenon of an acute inflammatory process during bacterial destruction of the lungs is localized mainly in the distal parts of the lungs, which are the most vulnerable area of the organs of the bronchoalveolar tree [1,8,17]. This process is associated with the anatomical and physiological features of the structure of organs, as well as lymph and blood circulation in this area. Along with this, the distal areas have a better developed protection system [3,6]. There are also such factors of natural protection of the respiratory tract as the presence of the mucociliary clearance process, class A immunoglobulins, surfactant, the activity of natural killers and T cells, as well as the complement system, which are involved in maintaining the sterility of the bronchoalveolar tree and its resistance to infection [2, 6 ,9,10]. At the same time, a violation of some of these links or their complex dysfunction can lead to infection of the lower parts of the respiratory system. The structure and pathogenic properties of the microorganism are directly related to the development of pneumonia [6,9,10,15]. With the development of pneumonia in bronchoalveolar tissues and in the lymphoid system, dysfunction is observed on the part of the peripheral circulatory and lymphatic systems, arising from the activation of all components of the immune system, in which changes in immunocompetent cells of the T-cell phenotype are observed with an increase in the synthesis of CD8 + cells, an increase in their activities as a result of activation complement systems and phagocytosis, as well as an increase in the number of immature cells [4,5,12]. At the same time, with The structure and pathogenic properties of the microorganism are directly related to the development of pneumonia [6,9,10,15]. With the development of pneumonia in bronchoalveolar tissues and in the lymphoid system, dysfunction is observed on the part of the peripheral circulatory and lymphatic systems, arising from the activation of all components of the immune system, in which changes in immunocompetent cells of the T-cell phenotype are observed with an increase in the synthesis of CD8 + cells, an increase in their activities as a result of activation complement systems and phagocytosis, as well as an increase in the number of immature cells [4,5,12]. At the same time, with The structure and pathogenic properties of the microorganism are directly related to the development of pneumonia [6,9,10,15]. With the development of pneumonia in bronchoalveolar tissues and in the lymphoid system, dysfunction is observed on the part of the peripheral circulatory and lymphatic systems, arising from the activation of all components of the immune system, in which changes in immunocompetent cells of the T-

cell phenotype are observed with an increase in the synthesis of CD8 + cells, an increase in their activities as a result of activation complement systems and phagocytosis, as well as an increase in the number of immature cells [4,5,12]. At the same time, with arising from the activation of all components of the immune system, in which there are changes in immunocompetent cells of the T-cell phenotype with an increase in the synthesis of CD8+ cells, an increase in their activities as a result of the activation of complement systems and phagocytosis, as well as an increase in the number of immature cells [4,5,12]. At the same time, with arising from the activation of all components of the immune system, in which there are changes in immunocompetent cells of the T-cell phenotype with an increase in the synthesis of CD8+ cells, an increase in their activities as a result of the activation of complement systems and phagocytosis, as well as an increase in the number of immature cells [4,5,12]. At the same time, withit is necessary to pay attention to the increase in the content of such serum cytokines as -TNF-α, IL-1, IL-4, IL-6, IL-10, MCP-1 [13].

The amount of these cytokines regulates the degree of manifestation of inflammation processes, by regulating the production of proteins (CRP), fibrinogen, the degree of leukocytosis, the values of the erythrocyte sedimentation rate, the degree of formation of IL-8 chemokines, the slow-reacting substance of anaphylaxis, which increase the coupling values and the level of activity of white blood cells [11 ,14,20]. The listed indicators of body protection are of great importance in the manifestation of pneumonia, showing the severity of a high level of mortality and mortality [14,23,24].

During the period of inflammation in the place of the inflamed area of the lung tissue, the activity of cytotoxic T-lymphocytes, T-helpers and antibodies belonging to different groups is noted. [19]. Due to the process of synergy of these cytokines with complement proteins, the phagocytic response of neutrophils begins [22,23]. Natural killers belonging to the subpopulation of T-helpers secrete IL-4, due to which the transition of T-helpers to the second type occurs [18,21]. The number of T-helpers is the main factor in the outcome of acute pneumonia. An increased number of T-helpers of the first type indicates an intracellular infectious process [19,20]. The main cytokines are interferon-γ and IL-2 [20,24]. Under the influence of these cytokines, a delayed-type hypersensitivity reaction is provoked by macrophages, while there is a release of IgG3 instead of IgM due to B-lymphocytes. T-helpers of the second type secrete IL-10, which stops this reaction [22]. B-lymphocytes and eosinophils carry out an immune response to extracellular infection. An increase in the secretion of T-helpers of the second type occurs due to the release of IL-4, IL-6, IL-13 secreted by mast cells and T-helpers, due to this, the process of transition from the synthesis of IgM to IgG4, IgE, IgA, IgG2 is also carried out; this process is suppressed by interferon-γ [14,15,21]. due to this, the process of transition from the synthesis of IgM to IgG4, IgE, IgA, IgG2 is also carried out; this process is suppressed by interferon-γ [14,15,21]. due to this, the process of transition from the synthesis of IgM to IgG4, IgE, IgA, IgG2 is also carried out; this process is suppressed by interferon-γ [14,15,21].

Cytokines provoke a number of processes that are typical for lung tissue inflammation, and these manifests itself in dysfunction of the microvasculature, the presence of oxygen starvation of tissues, fluid accumulation in the alveolar and interstitial tissue, and the manifestation of dysfunction of the metabolic function of the lungs [12,20]. The variation in the amount of cytokines in peripheral vessels shows the severity, the end result of inflammation of the lung tissue, and also helps to assess the quality of the treatment used

[13,22]. In the first 3-10 days of pneumonia, there is an increase in the amount of pro-inflammatory cytokines IL-6, 8, 12, the amount of which indicates the quality of treatment and the possible end of the disease [7,22]. This number may vary depending on the type of pathogen. Strong activation of alveolar macrophages and T-lymphocytes is observed in pneumococcal pathogen. And in the first 24 hours there is an increase in the number of IL-1β, TNF-α, IL-6, IL-10. The value of interferon-γ and IL-6 remain high for 7 days. With a viral causative agent of pneumonia, an increase in the concentration of pro-inflammatory cytokines is observed. In such a situation, it is possible to increase the release of Th2 cytokines (IL-4, 5, 13) leading to the lung tissue of eosinophils, which provokes the occurrence of bronchial obstruction [7,12,16]. During inflammation of the lung tissue caused by pneumococcal infection, the release of TNF-α and interferon-γ increases, which has a protective effect, while the release of IL-10, which reduces the inflammatory process, leading to a decrease in protective indicators [1]. There is a possibility that each pathogen causes a change in T-helpers in the direction of Th2, which leads to the failure of the immune response. As an example, we can consider inflammation caused by Mycoplasma pneumoniae, which leads to a decrease in IL-4 and interferon-γ, leading to chronic inflammation due to an insufficient immune response [12]. Cytokines have a bactericidal effect. It has been proven that TNF-α and interferon-γ inhibit the exotoxins of Gr+ bacteria - streptolysin "O" and tetanolysin [16,17]. The severity and outcome of pneumonia is determined by how effective the processes of isolation of IL-12, 6, 18 and interferon-γ are. It was also noted that with a decrease in the synthesis of IL-12, which has the properties of antibacterial protection, repeated cases of pneumonia were observed. These properties of IL-12 are based on such interferon-γ dependent processes as stimulation of cytokine activity, T-cell infiltration and increased NO synthesis. IL-4 and interferon-γ are involved in regenerative processes in the epithelial tissue of the respiratory tract against the background of inflammatory processes in the lungs. At the same time, the main role in the outcome of the inflammatory process of the lungs belongs to IL-18, which is a synergist of IL-12 and works in conjunction with interferon-γ [19,20,21,22]. However, from another point of view, the ability of interferon-γ and TNF-α to induce the expression of CD54+/ICAM-1 can lead to attachment of cells to the endothelium, as well as aggregation of fibrin particles, which ultimately lead to hemodynamic disturbances in capillaries, an increase in their permeability and an increase in edema in the lung tissue against the background of inflammatory processes [19]. The outcome, the dynamics of the inflammatory process, as well as its degree depends on the activity of T- and B-lymphocytes, the activity of cytokines and antibodies [24]. Cytokines in this process can act,

In this way, Based on the above, we can conclude that the study of the factors of humoral and cellular immunity allows us to understand the course of pathological processes in community-acquired pneumonia, and also makes it possible to individually predict the patient's condition.

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