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Determination of Immunoglobulin E and G Levels and Complement Activity Relatively in Blood and Lymph

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INTRODUCTION

Type I hypersensitivity reactions underlie anaphylactic shock, bronchial asthma, drug-disease, food allergy, eczema, and other common human pathological cases. These reactions are induced by certain types of antigens (allergens and are characterized by all signs of a normal humoral immune response). The development of the reaction consists of recognition, processing, and presentation of allergen, as well as the cooperation of T and B lymphocytes with the formation of the clone of antibody-forming plasma cells and cells of immune "memory". Their only difference from humoral reactions of other types is the production of Class E (IgE) specific immunoglobulins having a high affinity for Fc receptors of mast cells and blood basophils. High-affinity receptors allow cells to bind IgE even at its low concentration. The increase in interleukin (IL) 4 levels in atopic reactions results in increased secretion of IgE-antibodies and the connection of a cascade of IgE mediated allergic reactions involving mast cells, basophils and eosinophils. Studies have shown that in immune response several isotype antibodies are synthesized, most of which are IgG-anthelium [1]. It has shown that induction of IgG - antibodies to the main allergen to reduce IgE production. And also, it is believed, in connection with reagents not only with IgE but also with subclasses of IgG. Antibodies of G3 subclasses play a key role in mechanisms of immunological recognition of foreign antigens, which during immune response connect cascade of mechanisms leading to destruction and elimination of foreign antigens and pathogens [2]. G4 antibodies participate in mechanisms of allergic inflammation but do not provide destruction and removal of allergens and atopenes from the body. Mast cell degranulation can also be induced by anaphylatoxins (C3a, C4a, C5a), various drugs (synthetic ACTH, codeine, morphine) and compounds inducing Ca flow into the cell. Cross-binding of fixed IgE on mast cells and Fc R1 activation leads to a chain of biochemical processes with methylation of membrane phospholipids, resulting in increased mobility of the plasma membrane and formation of Ca^{++} channels. This leads to an increase in Ca^{++} concentration within the cell, which through activation of phospholipase A2 stimulates the formation of arachidonic acid converted into 2 classes of mediators: prostaglandins and leukotrienes. A rapid reduction in cAMP is an important condition for degranulation. Vasoactive amines released by platelets, basophils and mast cells cause endothelial cell contraction, increase vascular permeability and create the possibility of immune complexes deposition [3]. Immune complexes, in turn, are able to trigger the mechanisms of inflammatory processes by activating the complement system to form anaphylatoxins C3a and C5a, which stimulate the release of vasoactive amines, including histamine and 5-hydroxytryptamine [4]. Immune complexes take part in the pathogenesis of disease development, if deposited in tissues, cause diffuse or local damage of their count of complement system activation, platelet aggregation, and phagocytosis enhancement. It is proven that circulating immune complexes to be deposited after administration of substances causing the release of vasoactive amines, including histamine [4]. It has also been found that prolonged administration of vasoactive amine antagonists to test rabbits reduces the deposition of immune complexes. In recent years, there has been evidence that patients with atopic diseases generally show an increase in the number of plasma cells and the level of secreted antibodies (IgM, IgE and IgG), an increase in the number of circulating immune complexes (CIC) [2]. The deposition and damaging effect of

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IR depends on the class of immunoglobulin included in the complexes. Thus, NZB/NZW (New Zealand mice black/New Zealand mice white) lines in mice, with ages, the IgM class is switched to IgG2 and coincides with the onset of kidney damage, indicating the value of the class of antibodies in the deposition of immune complexes in tissues. The rate of excretion of immune complexes also depends on the class of immunoglobulins included therein. In some diseases, for example as rheumatoid arthritis, rheumatoid factor IgG anti-IgG is produced by synovial tissue plasma cells; then these antibodies join together and cause an inflammatory response. The evidence indicates that the complement system can affect the course of many immune processes [5]. The change in complement profile indicates the existence of various types of complement, confirms the activation of complement cascade with signs of both classical and alternative and lectin pathways. Among the three ways of activating the complement system, the alternative way has a low level of basic activity, so it requires control from regulatory molecules. Disruption of these regulatory molecules on cell membranes results in complement amplification, accompanied by an increase in the synthesis of pro-inflammatory mediators such as C3a and C5a, which effect dynamic regulation of the immune response. They (mainly C5a) cause accumulation and activation of polymorphic - nuclear leukocytes leading to the release of lysosomal enzymes and peroxidation products, reduction of smooth muscle cells, release of histamine from mast cells and increase of vascular permeability [4]. There are a number of regulators (e.g. factor H) controlling complement. In the absence of sufficient binding of H factor and its protective properties, tissues may be exposed to complement, as a result, immune and inflammatory responses are formed. Some authors note a correlation between the blood content of the CIC, the complement system and the severity of the course of atopic diseases.

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