Volume 3, Issue 1, January 2025 ISSN (E): 2938-3765

THE VALUE OF AUTOANTIBODIES IN THE DIAGNOSIS OF AUTOIMMUNE AND AUTOINFLAMMATORY DISEASES

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Abstract
Autoinflammatory diseases (AInDs) are a variant of autoimmune diseases (AIDs) because they are also characterised by inflammation without an aetiological factor, are associated with immune response genes, and are amenable to immunosuppressive therapy [1,4,6,7].

Keywords: Autoimmune diseases, autoinflammatory diseases, immune response, autoantibodies.

Introduction

In contrast to AID caused by the so-called ‘autoimmune reaction’, the pathogenesis of AInD is based on the pathology of regulation of innate immune responses. More than 100 AID and AInD have been described, and many conditions have an autoimmune pathogenesis. Although traditionally considered rare nosologies, AInD is estimated to account for up to 20% of the general medical pathology. Epidemiological studies show that up to 9% of the population has AInD [8,9,11,12].

Over the last 20 years, there has been a worldwide increase in the number of AID, including a multiple increase in the prevalence of SLE and systemic rheumatic diseases, diabetes mellitus, multiple sclerosis and inflammatory bowel disease. This allows us to speak of an ‘epidemic of autoimmune diseases’, the causes of which include the development of the chemical industry in the second half of the 20th century, the adjuvant theory, which points to the role of immunity adjuvants in the composition of vaccines and exogenous substances, and the hygiene theory, which explains the increase in the number of autoimmune diseases by changes in the microflora of the organism, a decrease in the number of infections and worm infestations [15,16].

All immune mechanisms, including innate and acquired (both humoral and cellular), are involved in the pathogenesis of AID. To date, there are no clinically justified methods for assessing cellular

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immune responses. The detection of autoantibodies, i.e. serological methods, are central to the diagnosis of AID and are part of the criteria for a number of AID. Autoantibodies are immunoglobulin proteins directed against the body's own antigens and are involved in the pathogenesis of many AID. Autoantibodies are often directed against antigens of the same species, which allows them to be grouped into families, e.g. antinuclear antibody family, antimitochondrial antibody family [11,12,13].

In systemic rheumatic diseases, autoimmune liver diseases, autoimmune endocrinopathies, 'cross-syndromes' including symptoms of several AID are observed. Combined detection of the autoantibody spectrum is important for the diagnosis of such conditions. Although autoantibodies are in most cases reliable markers of AID, some of their variants can be detected relatively frequently in healthy individuals, and detection increases with age [14,15,16].

Autoantibodies that may be incidental findings in clinically healthy individuals include antinuclear factor (3-5%), antiphospholipid antibodies (5-10%), rheumatoid factor (3%), thyroperoxidase antibodies (4%), myocardial antibodies (5%), and skeletal muscle antibodies (3%). Therefore, the sensitivity and specificity of a particular test is of great importance in the diagnosis of autoimmune diseases. Sensitivity determines how often antibodies are found in individuals with the disease [4,7,8,910,11].

Specificity, on the other hand, determines the % of no antibodies in a control group with other diseases. For example, if the specificity is 90%, then 10% of the control group has antibodies, i.e. every 10th test will be a false positive. More sensitive tests are useful for screening (i.e. early diagnosis) of diseases, while more specific tests are useful for confirming the diagnosis (i.e. differential diagnosis).

For the practitioner, a useful indicator that assesses the value of a laboratory test is the risk factor, which determines how many times the probability of diagnosing diseases increases with a positive test result. The risk factor is calculated as Sensitivity /(1-Specificity). Thus, if the sensitivity of the test is 90% and specificity 95%, the risk factor will be 18, which means an 18-fold increase in the risk of a given disease in relation to the pre-test probability of the disease. Antibodies are determined in laboratories by several methods, primarily indirect immunofluorescence (nRIF), enzyme-linked immunosorbent assay (ELISA) and immunoblotting [1,2,3].

The indirect immunofluorescence method is the best method for detecting antibodies to insoluble tissue antigens, and antibodies to multiple targets can be detected in a single test, making it the optimal method for screening autoantibodies. If the antigen to which the autoantibodies bind is characterised precisely, enzyme-linked immunosorbent assay (ELISA) or immunoblotting (IB) is used. Autoantibody and immunoglobulin content usually decreases when immunosuppressive therapy is administered and when there is a good response to treatment. However, immunoglobulins are very stable and the half-life of the molecules in the body is about 1 month. Therefore, repeated serological examinations for autoantibody detection should not be performed more often than once every 3 months [1,2,3,4,5].

Autoantibodies form immune complexes and trigger the classical pathway of the complement system, so in many AID there is marked hypocomplementemia, indicating the consumption of complement factors. A special type of autoantibodies are autoantibodies presented to paraproteins, i.e. monoclonal immunoglobulins. So often monoclonal can be rheumatoid factor,

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haemoagglutinins, antibodies in polyneuropathies. In this case, the detection of autoantibodies should be supplemented by immunofixation to determine the clonality of the immunoglobulins. To monitor the treatment of AID with systemic inflammation, the detection of acute-phase response parameters such as ESR and CRP are commonly used.

Due to its high half-life (18 hours), when effective antibiotic/immunosuppressant therapy is administered, CRP concentrations return to normal within 3 days of treatment initiation, whereas a decrease in ESR may take several weeks. New informative biomarkers are useful for assessing systemic inflammation in AInD and AID [6,7,8].

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