Rozprawa Doktorska pod tytułem:

Udział „host immune response” w indukcji zjawiska surowiczooporności po leczeniu kiły wczesnej

ABSTRACT

Syphilis remains a significant global public health concern despite the availability of effective antibiotic therapy. In recent years, a resurgence in the incidence of syphilis has been observed, particularly in developed countries. Benzathine penicillin G remains the standard treatment for early syphilis, and therapeutic success is assessed based on both clinical and serological response.

In a subset of patients, however, the expected decline in non-treponemal antibody titers is not achieved despite appropriate treatment. This phenomenon, referred to as the serofast state, affects approximately 15–20% of patients. The pathogenesis of this condition remains unclear, and its clinical significance is still debated. In particular, it is uncertain whether persistent seroreactivity reflects ongoing infection or dysregulation of the host immune response.

The main aim of this study was to evaluate the role of the host immune response in the pathogenesis of the serofast state following treatment of early syphilis.

The specific objectives included: assessing differences in immune response between patients with an adequate serological response and those with serofast status; analyzing peripheral blood lymphocyte subpopulations and selected immunological parameters over time; determining the association between interferon gamma (IFN-γ) gene polymorphisms and the risk of developing the serofast state; and evaluating the presence and clinical relevance of anticardiolipin antibodies.

In the conducted studies, significant differences in immune response were observed between patients with a normal serological response and those with the serofast state.

Patients with serofast status demonstrated a significantly reduced number of cytotoxic T lymphocytes (CD8+) and total T lymphocytes (CD3+), both before and after treatment, indicating impaired cellular immune response.

At the same time, persistent activation of the humoral immune response was observed, reflected by increased B-cell counts after treatment. These findings suggest an imbalance between cellular and humoral immunity as a potential mechanism underlying persistent seroreactivity.

A significant association was also identified between IFN-γ gene polymorphisms (+874 T/A and +2109 A/G) and the risk of developing the serofast state. Genotypes associated with lower IFN-γ production were linked to a higher likelihood of persistent serological response, highlighting the role of immunogenetic factors in modulating treatment outcomes.

Analysis of anticardiolipin antibodies demonstrated their presence before treatment and complete disappearance after therapy, indicating their transient nature and lack of association with persistent seroreactivity.

Overall, the results indicate that the serofast state is primarily related to dysregulation of the host immune response rather than persistent infection.

The serofast state following treatment of early syphilis represents a complex clinical phenomenon resulting from dysregulation of the host immune response. A key role is played by an imbalance between impaired cellular immunity and sustained humoral activation.

Genetic factors, particularly IFN-γ gene polymorphisms, significantly influence he effectiveness of the immune response and contribute to the risk of developing the serofast state.

These findings provide new insights into the mechanisms underlying persistent seroreactivity and may contribute to the development of more precise diagnostic and therapeutic strategies, including the identification of patients who may benefit from individualized clinical management.