Selective immunoglobulin A deficiency is exceptionally associated with multiple sclerosis

El déficit selectivo de inmunoglobulina A excepcionalmente se asocia a esclerosis múltiple

Dear Editor:

It was with great interest that we read the article by Remolina López et al. on one of the few cases in the literature of IgA deficiency in association with multiple sclerosis. In fact, we could only find one other case, that of a Japanese patient, in PubMed. We would like to call attention to the rarity of this association and its possible aetiopathogenic implications.

Selective IgA deficiency is the most common primary immunodeficiency, and its prevalence varies among different ethnic groups. In our own region, our team found the highest prevalence of this deficiency in the general population in the Western world: 1 in 163 individuals. According to the current study protocol for multiple sclerosis, we measure serum Ig levels (IgG, IgA, and IgM), the kinetics of intrathecal immunoglobulin synthesis, and oligoclonal bands in cerebrospinal fluid in almost every patient. Therefore, we do not believe that immunoglobulin deficiencies go undetected or under-diagnosed in this disease. We studied 183 cases of multiple sclerosis in our series of patients, and we have not found any cases of IgA deficiency in the past 18 years. This contrasts with the high prevalence of IgA deficiency in the general population (0.61%) and in conjunction with numerous autoimmune diseases such as coeliac disease (4.1%), systemic lupus erythematosus (1.5%), thyroiditis (2.69%), and diabetes mellitus (4.70%). The absence of an association between multiple sclerosis and selective IgA deficiency may have aetiopathogenic implications. It could indicate a different genetic substratum or a different environmental trigger from those involved in the autoimmune diseases mentioned above. The lack of IgA in secretions fosters antigenic overstimulation, especially in the digestive tract. It is unlikely, however that the digestive tract would play an important role in triggering multiple sclerosis. In this regard, it is particularly revealing that in myasthenia gravis, another autoimmune disease of the nervous system, we also find no association with selective IgA deficiency. Given the above, we believe that the case described by Remolina López et al. should be considered an exception based on the statistical evidence, and due to the high prevalence of both diseases. Discovering a possible causal association or a common origin for the diseases seems less likely.

References


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