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Commentary

A Commentary on MicroRNA Profiling in Behçet's Disease

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DESCRIPTION

Behçet's disease (BD) is a rare and chronic multisystem disease characterized by a triple-symptom complex of recurrent oral aphthous ulcers, genital ulcers, and uveitis. Moreover, manifestations of vascular, articular, neurologic, urogenital, gastrointestinal, pulmonary, and cardiac involvement may occur. Hippocrates described BD in the fifth century BCE. In 1930, the Greek ophthalmologist Benediktos Adamantiades reported a patient with inflammatory arthritis, oral and genital ulcers, phlebitis, and iritis.

As virtually no unique histological or laboratory features have been identified to help in the diagnosis of the disease, clinical features are used to define and diagnose Behçet syndrome. An international study group on Behçet's disease has recently revised the criteria for the classification/diagnosis of BD.

There are sporadic cases of BD all around the world, but it is most frequently seen along the ancient Silk Route because of its frequency in the Middle East and far-east Asia (prevalence of 14–20/100,000 inhabitants), and these regions have traditionally been considered the endemic areas for the condition.

BD is a sporadic disease, but a familial aggregation is well known. Carriers of HLA-B51/HLA-B5 have an increased risk of developing Behçet's disease compared with noncarriers. HLA-B51 is the strongest associated genetic factor, and it has been shown to be more prevalent in Turkish, Middle Eastern, and Japanese populations, with a higher prevalence of Behçet's disease in these populations. Non-HLA genes also contribute to the development of BD . Genome-wide association studies have shown that polymorphisms in genes encoding for cytokines, activator factors, and chemokines are associated with increased BD susceptibility. Among cytokines, IL-10 polymorphisms cause a reduction in the serum level of IL-10, an inhibitory cytokine that regulates innate and adaptive immune responses; on the other hand, IL-23 receptor polymorphism, which reduces the response to IL-23 stimulation, is associated with protection from BD. Recent data reported also associations with CCR1, STAT4, and KLRC4 encoding for a chemokine receptor, a transcription factor implicated in IL-12 and IL-23 signaling and a natural killer receptor. Finally, susceptibility genes involved in the innate immune response to microbial exposure have recently been identified by Immunochip analysis.

Increased Th1, CD4+ and CD8+ T cell, γδ+ T cell, and neutrophil activities have been found both in the serum and in inflamed tissues of BD patients, suggesting the involvement of innate and adaptive immunity in the pathogenesis of BD. Studies on T lymphocytes have suggested a Th1-predominant response. Both CD4+ and CD8+ lymphocytes are higher in the peripheral blood, with increased levels of IL-2 and interferon-(INF-) γ cytokines. The cytokine Th17 may also play an important role in the pathogenesis of the disease. This hypothesis is supported by the observation of high IL-21 and IL-17 levels in sera of patients affected by BD with neurologic involvement. Another study has reported a higher Th17/Th1 ratio in peripheral blood of patients with BD compared to healthy controls, and this ratio was higher in patients with uveitis or folliculitis compared with patients without these manifestations. MicroRNAs (miRNAs) are small noncoding RNAs that play an important role in the regulation of several biological processes through their interaction with cellular messenger RNAs. Inflammatory responses have an impact on miRNA expression, regulating their biogenesis by altering the transcription and processing of precursor transcripts or influencing the stabilization of mature miRNAs. In recent years, the number of miRNAs implicated in immune system development and function has dramatically enhanced, and there has been widespread discussion of their potential use as therapeutics for immunological diseases. Indeed, the aberrant expression of miRNAs frequently occurs in human diseases, including hematological disorders and autoimmunity.

The concept that miRNAs participate in the pathogenesis of diseases, especially refractory diseases with unidentified mechanisms, might lead to a novel effective treatment. A number of studies have reported a differential expression of miRNAs in several inflammatory autoimmune diseases, such as in rheumatoid arthritis (RA), multiple sclerosis, systemic lupus erythematosus, psoriasis, and systemic sclerosis. These studies highlighted a deep implication of miRNAs as regulatory molecules in autoimmunity and the intriguing possibility to use miRNAs as disease biomarkers in these immunological disorders.

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