Short communication

Hormones, food, stress and infections: their etiological role in autoimmune diseases

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ABSTRACT

Introduction: The etiology of autoimmune diseases is still unknown, but different causes arise.

Objective: To describe the role of hormones, diet, stress and infections in the etiology of autoimmune diseases.

Methods: A bibliographic review was made using the Google Scholar and articles of free access in the Pubmed and Scielo database from 2015 to 2019. The search terms were used according to the DeCS and MeSH descriptors.

Development: It is well known that female hormones increase the risk of autoimmune diseases. Stress can maintain low-grade chronic inflammatory responses that cause tissue damage, initiating or aggravating the clinical manifestations of autoimmunity. An adequate diet allows the guests of the intestinal microbiota to maintain the homeostasis of

the immune system. Today, glutamate is used as a flavor enhancer, especially in developed countries. Perhaps it is one of the causes of the higher incidence of autoimmune diseases in these regions.

Conclusions: Autoimmune diseases are more frequent in women. Adequate nutrition allows the gut microbiota not to be altered and to maintain immunological homeostasis. Infections and stress situations can trigger or exacerbate the clinical manifestations of autoimmunity.

Keywords: autoimmunity; hormones- autoimmunity; stress- autoimmunity; food- autoimmunity; infections- autoimmunity.

INTRODUCTION

Failures in tolerance cause autoantibodies and autoreactive lymphocytes that cause autoimmune diseases. These trigger immune responses that are harmful to the body. Even when genetics is a predisposing factor, diet, hormones, stress, among others, are necessary to trigger them. However, the initiation of these mechanisms is unknown.[1,2,3]

Evolutively, since the females are responsible for perpetuating the species, their immune system is stronger, which is why they are more prone to develop autoimmune diseases due to the action of the female sex hormones.[2,4]

It has been seen that stress can cause autoimmune diseases. Psychoneuroendocrinoimmunology is a science that explains the role of these phenomenons in these systems. In addition, infectious diseases can trigger, through different mechanisms, failures in the tolerance to self-reactive activity.[3]

Due to the lack of understanding of the complexity of these issues, the present review was conducted with the objective of describing the role of hormones, nutrition, stress and infectious diseases in the development of autoimmune diseases.

HORMONES AND AUTOIMMUNITY

Why are autoimmune diseases more common in women than in men? Estrogens are hormones considered immunostimulatory while progesterone and androgens have immunomodulatory effects due to changes in Th1 / Th2 patterns.[5]

Its role in cellular homeostasis is complex and depends on the physiological context. They modulate the immune response, control gene expression and modify the expression of proteins by interacting with the genome through nuclear and extranuclear pathways. An imbalance in these mechanisms can trigger autoimmune diseases.[1,6]

Then one might ask how does a woman tolerate the fetus that is a semialograft? A balance has been achieved during pregnancy. The predominance of the inflammatory pattern (Th1) is necessary during fertilization, implantation, which returns around the third trimester for delivery. The regulatory Th2 response predominates during the rest of the pregnancy. Changes in this balance are associated with implantation problems, recurrent abortions, preterm delivery and infertility.[7]

FEEDING AND AUTOIMMUNITY

It is one of the topics currently studied and of which there are still many puzzles to elucidate. Alterations in diet cause changes in the intestinal microbiome and are associated with autoimmune diseases and inflammatory disorders. Diets with a predominance of saturated fatty acids promote the Th1-Th17 response and activate the inflammasome, which can induce tissue lesions. While the ingestion of unsaturated fatty acids inhibits the aforementioned substances. The mechanisms that cause these alterations are not fully understood.[7]

In type 1 diabetes mellitus, the production of autoantibodies against pancreatic islets has been related to exposure to toxic chemicals, the consumption of milk in childhood and the decrease of commensal bacteria of the gastric microbiota such as Bifidobacterium.[7,8]

An imbalance of the intestinal anaerobic germs is associated with stimuli for the proliferation of Th17 lymphocytes in the inflammatory disease of the colon, while the presence of the commensal species facilitates the development of regulatory T-

lymphocytes and the production of IL-10. In autoimmune neuroinflammatory pathologies, microbiota dysbiosis can increase production of autoreactive lymphocytes.(7)

Monosodium glutamate (MSG), a monosodium salt of glutamic acid, is used to enhance the taste of food. There is evidence that increasing their intracerebral concentration increases the levels of proinflammatory cytokines. This has been proven in traumatic brain injuries, neurodegenerative diseases and central nervous system infections. However, the causal mechanism has not yet been fully elucidated.(9)

STRESS AND AUTOIMMUNITY

The psychoneuroendocrine-immune system (PNEI) encompasses numerous organ systems and their interactions. Today, almost all the population is subjected to prolonged stressful situations that cause depletion and imbalance of the PNEI system. For this reason, stress is considered a pandemic related to the increase of obesity, hypertension and atherosclerosis and autoimmune diseases.(3)

Many patients as a result of stress suffer systemic inflammatory diseases that affect different organ systems such as cancer, cardiovascular diseases, heart attacks, Parkinson's disease, psychiatric diseases, severe chronic fatigue, among others. In diabetics, stress increases the probability of morphofunctional abnormalities in the organogenesis of the fetus.(3,4)

INFECTIONS AND AUTOIMMUNITY

It is one of the most known and studied aspects in relation to the etiology of autoimmune diseases. For that reason in the present work we will limit ourselves to mention the mechanisms that are known.

Infectious agents can trigger the immune system to be autoreactive by cross-reactivity between aggressor structures and the host, by non-specific activation when pathogens break tolerance in the inflammatory response, by presentation of cryptic antigens or superantigens that activate many lymphocytes that can be self-reactive For example, in animal models, viruses can trigger exacerbation of the clinical manifestations of lupus.

Persistent infections with enteroviruses increase the damage in the islets of the pancreas, since they break the tolerance and activate lymphocytes that damage this tissue.[10]

CONCLUSIONS

Autoimmune diseases are more frequent in women. Adequate nutrition allows the gut microbiota not to be altered and to maintain immunological homeostasis. Infections and stress situations can trigger or exacerbate the clinical manifestations of autoimmunity.

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