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Short Communication

Association Between Diabetes and Trichinella Spiralis

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Abstract:

Diabetes is widespread and has been reported in 8.5% of the world's population, with an estimated 600 million cases by 2035. Therefore, it is considered a major public health problem. Furthermore, parasitic helminth infections are believed to play a key role in the progression of DM, as studies suggest they mitigate or slow complications. Among these parasites, Trichinella spiralis stands out due to its complex life cycle involving various organ systems of the host, which implies extensive interaction with the immune system in pursuit of its survival. The objective of this document is to present the arguments presented in this regard, based on an analysis of the information available in the global scientific literature.

Key words: diabetes; trichinella spiralis; immune system; autoimmune diseases; antigens

Introduction

Helminths influence the progression of chronic autoimmune diseases, especially in low-income countries. In this regard, it is postulated that substantial improvements in hygiene and environmental sanitation, as well as increased wastewater infrastructure, contribute significantly to reducing the prevalence of helminthiasis and are inversely proportional to the increase in clinical cases of diabetes in these populations (considered a public health problem because it is reported in 8.5% of the world's population, with a trend toward an increase in the number of cases). Based on this hypothesis, several studies have shown that parasitic helminth infections can prevent the onset of autoimmune diseases or their progression to severe complications, whose pathophysiological processes involve parasitic modulation of the host's immune response [1-7].

In the case of *Trichinella spiralis*, which causes trichinosis characterized by the encystment of its larvae in the striated muscle of the heart and musculoskeletal system, as well as in the brain and gastrointestinal tract, its complex life cycle, involving passage through various organ systems (gastrointestinal, pulmonary, circulatory, and muscular, and exceptionally, the brain) with marked interaction with the immune system in its struggle to survive, could be one of the appropriate models to demonstrate the effect of helminths on diabetes (which affects approximately 8.5% of the world's population) [3,7]. Therefore, the objective of this paper is to present arguments in this regard, based on an analysis of existing information on the subject.

T. spiralis the parasite

It is a *Nemathelminthes* of the family *Trichinellidae* that infects a wide range of mammals and humans. Its life cycle does not involve any free-living stage, but rather larvae and adults within the same host (autoheteroxenous). Its life cycle begins with the ingestion of undercooked or raw meat infected with parasite cysts. The free larva then penetrates the wall of the small intestine, where it remains until it reaches adulthood [8-11].

In the small intestine, the adults mate. The males die and are eliminated in the feces. The gravid females release larvae into the intestinal mucosa. These larvae reach the bloodstream and travel to the lungs, from where they can disseminate throughout the body. However, due to positive tropism, they prefer striated muscle, where they encyst and remain viable for many years (between 5 and 10 years) until ingested by a new host [8-11].

The pathogenic mechanisms in the small intestine (duodenum and jejunum) include edema, hyperemia, increased mucus production, and mild eosinophilic infiltration. In skeletal muscle, they cause separation, degeneration, thickening, and hypertrophy. Within the muscle fiber, they cause proliferation and elongation of the nuclei, as well as loss of sarcoplasmic striations or sarcolemma and formation of a capsule derived from the sarcolemma around the larva. Acutely, this parasite causes myocarditis, myositis, and encephalitis [8-11].

Effects of *T. spiralis* on the host immune system that influence the progression of diabetes

Several host mechanisms have been identified as participating in the development of autoimmune diseases, such as the robust immune response against helminth antigens that inhibits the response to weaker antigens (autoantigens and allergens), and the suppressive function of regulatory T lymphocytes (Tregs) in the immune response to helminth antigens [7, 12].

Therefore, regarding *T. spiralis* and the second hypothesis, the following are noted: direct activation of Tregs or modifications in the movement of autoreactive T cells. It is also reported that helminths stimulate the Th2-type immune response, which involves the genesis of eosinophilia, the production of IgE, and the release of specific cytokines (IL-4, IL-5, and IL-13) [7, 8, 13].

In the case of *T. spiralis*, specifically during the progression of infection, its antigens activate the host's immune system (structural or secretory/excretory) towards the negative regulation of the adaptive immune response, that is, from inflammation to anti-inflammation, based on the action of Treg lymphocytes, alternatively activated macrophages, and regulatory B lymphocytes, which influence dendritic cells and T lymphocytes, resulting in greater insulin sensitivity and less inflammation (predominance of Th2 over Th1 immune response) [7, 12].

Blood glucose levels are reduced in individuals infected with *T. spiralis*, directly related to the development of muscle larvae (glucose consumption by these life cycle stages) and the encystment of infected muscle cells. Furthermore, significantly elevated levels of superoxide dismutase, glutathione S-transferase, and peroxidase are reported, accompanied by an increase in antioxidants, including vitamin E [14, 15].

For this reason, the use of *T. spiralis* antigens is currently being proposed for the treatment of autoimmune diseases, such as rheumatoid arthritis, autoimmune encephalitis, and diabetes and its most severe complication, nephropathy, which can lead to end-stage renal disease. It is also being considered for the treatment of inflammatory bowel and respiratory tract disorders [16-18].

Conclusions

The helminth *T. spiralis* can inhibit the development of diabetes and its progression to nephropathy and eventual end-stage renal disease by downregulating the host's adaptive immune response, that is, by promoting a Th2 predominance, with a reduction in blood glucose levels (due to larval consumption) and increased insulin sensitivity. Therefore, some authors suggest its use in the design of new pharmacological intervention strategies to prevent the harmful effects of this pathology.

Conflict of interests

The authors have no conflict of interest to declare. The authors declared that this study has received no financial support.

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