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Opinion Article

Role of Environmental Factors in the Pathogenesis of Thyroid Autoimmune Diseases

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DESCRIPTION

Hashimoto's thyroiditis and Graves' disease are two of the most common autoimmune disorders affecting the thyroid in the globe. These conditions are caused by a combination of environmental stimuli and genetic predispositions that cause immune system dysregulation and consequent thyroid tissue destruction. Although the underlying vulnerability is determined by genetics, environmental circumstances can serve as important triggers for the onset or aggravation of many illnesses. Clarifying the pathophysiology of Thyroid Autoimmune Diseases (TADs) and creating prevention measures or therapeutic therapies require an understanding of these environmental factors.

Iodine consumption is a major environmental factor that contributes to thyroid autoimmunity. The formation of TADs has been linked to both higher and lower iodine levels. High iodine intake, which is typical in regions where iodized salt is supplemented, can make thyroglobulin, a key thyroid protein, more antigenic. Autoimmune reactions might result from this increased antigenicity stimulating the immune system. However, in certain developing nations, iodine shortage is still common. It can also cause stress to the thyroid gland, causing compensatory processes that may cause autoimmunity in genetically susceptible individuals. Thyroid health depends on maintaining an ideal iodine intake since either extreme increases the likelihood of autoimmune disease. A trace element with excellent antioxidant qualities, selenium is another important environmental component. Since glutathione peroxidase and other selenium-proteins essential for reducing oxidative stress in the thyroid gland are inhibited by selenium deficiency, there is a higher chance of thyroid autoimmunity. An autoimmune reaction may result from increased thyroid antigen release caused by oxidative damage. The role of environmental contaminants in TADs is becoming better acknowledged. Endocrine-Disrupting Chemicals (EDCs) such phthalates, Bisphenol A (BPA) and Polychlorinated Biphenyls (PCBs) disrupt the immune system and thyroid. In addition to disrupting thyroid hormone production and altering immunological responses, these drugs have the ability to mimic or block thyroid hormones, hence promoting inflammation and fostering autoimmunity. One indicator of autoimmune thyroid disorders, elevated thyroid peroxidase antibodies, has been connected to BPA exposure. Furthermore, oxidative stress caused by heavy metal accumulation in the thyroid gland, such as that caused by mercury and cadmium, is known to exacerbate autoimmune processes. Reducing exposure to these environmental pollutants is essential to lessening the impact of TADs. The pathophysiology of thyroid autoimmunity also heavily relies on infections. Through processes including molecular mimicry, bystander activation and epitope dissemination, bacterial and viral infections can set off autoimmune reactions. For instance, TADs have been linked to Epstein-Barr Virus (EBV) and Yersinia enterocolitica infections. Cross-reactive immune responses are caused by molecular mimicry, which happens when microbial antigens mimic thyroid antigens. Epitope spreading is the diversification of the autoimmune response to encompass various thyroid antigens, whereas bystander activation is the stimulation of autoreactive T cells in the setting of infection. These processes highlight how essential it is to comprehend how infection causes thyroid gland autoimmunity. Physical and psychological stress is another environmental element that affects TADs. Prolonged stress can change the hypothalamic-Pituitary-Thyroid (HPT) axis, which can cause immunological and thyroid Hormone production to become dysregulated. Cortisol and other stress hormones secreted during stressful situations might alter immune cell function and raise the likelihood of autoimmune responses. Furthermore, stress might worsen TADs' clinical course by increasing antibody production and inflammation. Thyroid autoimmunity may be less affected by stress if it is managed with lifestyle changes like exercise and mindfulness. One known environmental component of thyroid disorders, including autoimmune illnesses, is radiation exposure. Ionizing radiation can harm thyroid cells and change their antigenicity, making them targets for immune assault. This might happen as a result of medical procedures, nuclear accidents, or occupational exposure. Thyroid autoantibodies and autoimmune thyroiditis, for example, were more common in people exposed to radioactive iodine during the Chernobyl

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tragedy. Thyroid autoimmunity is influenced by dietary variables other than iodine and selenium. TADs have been linked to the protein gluten, which is present in wheat, barley and rye, especially in those who have celiac disease. This connection might be explained by shared immunological and genetic mechanisms between TADs and celiac disease. In those who are vulnerable, gluten-free diets have demonstrated promise in lowering thyroid antibody levels and enhancing thyroid function. Likewise, goitrogens, which are present in cruciferous vegetables, can disrupt thyroid function, although they have less of an effect on autoimmunity when eating a typical diet.

CONCLUSION

Thyroid autoimmune illnesses are mostly caused by environmental variables, which work in concert with genetic predisposition to affect the start and course of the disease. Dietary variables, radiation, stress, infections, environmental contaminants, micronutrient imbalances and gut microbiota are important contributors. Significant promise for avoiding and controlling TADs exists when these environmental factors are addressed through focused treatments, such as improving gut health, lowering exposure to toxins, managing stress and optimizing nutritional intake.