Autoimmune Thyroid Diseases

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Abstract

The thyroid gland is frequently affected by the autoimmune disorders Graves' Disease (GD) and Hashimoto's Thyroiditis (HT), which lead to hyperthyroidism and hypothyroidism, respectively. They have a number of mysterious connections despite their conflicting clinical manifestations. Here, we suggest that GD and HT share a common underlying cause: they both result from an advantageous physiological process known as the autoimmune surveillance of hypersecreting mutants. Mutant cells that over secrete hormones and pose a threat of developing into poisonous nodules are selectively eliminated by autoreactive T cells. In those who are vulnerable, these T cells can set off a humoral response that results in the development of antibodies against thyroid antigens. Despite having opposing clinical phenotypes, HT and GD have similar incidence and risk factors, which can be explained by their shared genesis.

Keywords: Physiological autoimmunity • Graves' disease • Hashimoto's thyroiditis • Autoimmune etiology

Introduction

In GD, the Thyroid-Stimulating Hormone Receptor (TSHR) is stimulated by autoantibodies produced by the immune system. As a result, hyperthyroidism (see Glossary) results from the thyroid gland producing excessive amounts of thyroid hormones. In contrast, HT is a disorder in which the thyroid is destroyed by autoantibodies and autoreactive T cells, resulting in hypothyroidism. The thyroid antigens Thyroglobulin (Tg) and Thyroid Peroxidase (TPO) are the targets of the autoantibodies seen in HT.

It is unclear what causes these similar traits in the two disorders. Although this hypothesis needs more testing, one idea holds that lymphocytic infiltration of the thyroid in GD can cause the TSHR antibody response to additional thyroid autoantigens to spread molecularly, ultimately resulting in HT.

Here, we investigate the physiological process that, in all healthy persons, results in autoreactive TSHR-specific T cells as a potential cause of GD. This procedure stops mutant thyroid cells from growing, which would otherwise result in harmful thyroid nodules. The price of this crucial mechanism is that it can activate B cells and produce TSHR autoantibodies, which can result in GD in those with genetic risk factors like HLA-DR3.

According to this view, autoreactive T cells in healthy people have a physiological function that involves removing mutant cells that are hyper sensitized to a signal that boosts hormone secretion and cell growth. We dubbed this regulatory motif "the secrete-and-grow circuit" (top panel) because it is shared by many endocrine systems. An input signal causes endocrine cells to secrete their hormones and grow. One example of such a circuit is the regulation of the thyroid. The thyroid hormones T3 and T4 are secreted by thyroid follicular cells as a result of thyroid-stimulating hormone (TSH) acting as an input signal. Thyroid hormones create a negative feedback loop by inhibiting the pituitary gland's ability to produce TSH.

Because of their excessive proliferation and potential to develop into poisonous nodules, hypersecreting mutants (bottom left panel) are vulnerable to the secrete-and-grow circuit. Based on their increased autoantigen presentation to maintain homeostasis, autoreactive T cells are expected to eradicate these mutants (bottom middle panel). However, in those who are predisposed, excessive surveillance can result in autoimmune illnesses (bottom right panel).

His circuit has been demonstrated to perform various important tasks. By combining cell development and cell function in one signal, it controls how big the thyroid is. It stabilizes the thyroid's functional mass at a dimension that ensures correct operation, i.e., normal thyroid hormone levels. When levels of thyroid hormone are insufficient, TSH levels increase, causing increased production and, over a longer period of time, growing the thyroid functional mass (a condition known as goiter), which makes sure that levels of thyroid hormones are stable. For instance, goiter develops when iodine levels, which are necessary for the synthesis of thyroid hormones, are low.

The autoimmune surveillance idea provided a method to eradicate such mutations. According to this notion, mutant cells that over secrete the hormone can be specifically eliminated by autoreactive T cells that detect autoantigens in the secretion route. Due to their overexpression phenotype, these over secreting cells may additionally produce faulty protein fragments as antigen in addition to increased autoantigen on their surface MHCs. Because of the increased amount of presenting autoantigens or the changed antigens, T cells are able to identify and destroy these mutant cells. As a result, the mutant cells are selectively disadvantaged and hypersecreting clones are avoided. As a result, the idea gives T cells that are allergic to the thyroid and, in a similar way, a physiological purpose.

The identity of the autoantigens in each disease is also relevant to the autoimmune surveillance theory. It explains why several of the most prevalent autoantigens in endocrine autoimmune disorders come from proteins in hormone secretion pathways. Because these antigens are expressed more strongly in hypersecreting mutants than in wild-type cells, they are excellent for surveillance. Tg and TPO production is the final step in the synthesis of thyroid hormones in HT, and antibodies to these hormones are employed as a clinical diagnostic for the condition. The 21-hydroxylase in autoimmune Addison's disease and other autoantigens, as well as pre-proinsulin and other antigens in type 1 diabetes mellitus, all suit this hypothesis.

However, this physiological anti-mutant technique comes with a price: autoreactive T cells have the ability to cause an autoimmune disease in people who are predisposed to it by inducing a humoral reaction. This triggering most likely happens in people who exhibit particular known HLA alleles, together with outside influences like infections. By selecting cells for death, antibodies contribute to tissue damage. This leads to the production of more autoantigen, which spreads the autoimmune attack and may eventually result in an autoimmune illness.

We incorporate GD into the autoimmune surveillance theory in an effort to pinpoint the source of autoantibodies directed against TSHR. As a result, we propose that TSHR serves as a third autoantigen in the thyroid's autoimmune surveillance system, in addition to Tg and TPO, to identify mutant cells that secrete too much thyroid hormone. This is due to the theory that mutant cells that overexpress TSHR will create more thyroid hormone and multiply, posing the risk of developing into poisonous nodules. Therefore, it is expected that healthy people will have T lymphocytes that are directed against TSHR antigens to destroy these mutant cells. These T cells can cause B cells to create anti-TSHR antibodies in vulnerable people (such as HLA-DR3 carriers), some of which are stimulating antibodies that result in GD. One may anticipate that GD and HT would have comparable age- and sex-dependent incidence profiles if they have the same fundamental etiology. We used data from Claliti, an Israeli health service company, which served over 50 million life years (2002-2020), or almost half of the Israeli population, in order to investigate this and provide examples. The age-dependent incidence of various thyroid problems in both boys and females was calculated using International Classification of Diseases (ICD)-9 codes. The data consisted of N = 27 015 HT patients and N= 9560 GD cases.

Despite the opposite clinical expression of GD and HT, we offer a theory for a common origin of both conditions. Both illnesses may be the result of an advantageous autoimmune surveillance mechanism that was activated by autoreactive T cells against hypersecreting mutant thyroid cells.This theory of autoimmune surveillance for autoimmune disorders needs to be tested, nevertheless. An appropriate test would be to determine whether naturally occurring autoreactive T lymphocytes preferentially destroy hypersecreting cells in healthy endocrine tissues and so perform a mutant surveillance function. We point out that studies using mice may not be appropriate since their tiny thyroids may produce insufficient mutant cells to support the evolutionarily advantageous selection of an autoimmune surveillance mechanism. To avoid the development of poisonous nodules, humans and other large mammals are more likely to need a method of eradicating such mutations. It would be intriguing to investigate whether autoimmune surveillance may be used to treat other autoimmune disorders and autoantibody-mediated diseases.

Conclusion

However, there is a drawback to this physiological anti-mutant strategy: autoreactive T cells have the ability to cause an autoimmune disease in people who are predisposed to it by inducing a humoral reaction. Individuals that exhibit particular known HLA alleles as well as environmental elements like infections are most likely to experience this triggering. Antibodies contribute to tissue damage by destroying certain cells, which releases additional autoantigen and spreads the autoimmune attack further. This vicious cycle might result in an autoimmune illness that is fully developed.

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