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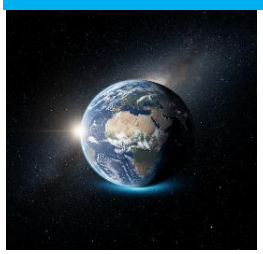

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Hashimoto's Thyroiditis: An Overview of Current Evidence from the Literature

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SUMMARY

Introduction and purpose

Hashimoto's thyroiditis is the most prevalent autoimmune disorder of the thyroid and a primary cause of hypothyroidism. It occurs predominantly in women and often manifests with vague, nonspecific complaints, which may also be present in euthyroid patients, leading to delayed recognition. Although levothyroxine remains the cornerstone of therapy, a subset of patients continues to report ongoing symptoms. This review outlines current evidence regarding the epidemiology, pathophysiology, clinical presentation, diagnostic approach, and both established and emerging therapeutic options for this condition.

A brief description of the state of knowledge

Hashimoto's thyroiditis is a frequent autoimmune disease and the leading cause of hypothyroidism in many populations. Its pathogenesis involves an interplay of genetic predisposition, environmental factors, and dysregulation of the immune response. Clinical

manifestations are heterogeneous and may include fatigue, depressive symptoms, and cognitive difficulties, even in the presence of normal thyroid hormone levels. Diagnosis is based on clinical evaluation supported by laboratory assessment of thyroid hormones and antibodies, as well as ultrasound imaging. While levothyroxine effectively normalizes biochemical parameters in most cases, persistent symptoms are commonly reported. This has led to growing interest in adjunctive interventions such as selenium, vitamin D, and inositol supplementation, acupuncture, and lifestyle modification. Increasing attention is also being paid to the influence of the gut–thyroid axis, including the potential role of probiotics in immune modulation. An individualized and more comprehensive treatment strategy may benefit patients with an incomplete response to standard therapy.

Conclusions

Hashimoto's thyroiditis is a multifactorial autoimmune condition that often necessitates an individualized therapeutic approach. Persistent symptoms despite appropriate treatment are common. Integrating conventional management with supportive measures may enhance clinical outcomes and improve patients' quality of life.

Keywords

hashimoto disease; autoimmune thyroiditis; hypothyroidism; thyroid disease

Introduction and purpose

Hashimoto's thyroiditis (HT) is the most prevalent autoimmune thyroid disorder and a leading cause of hypothyroidism globally. It primarily affects women and involves the progressive destruction of thyroid tissue through autoimmune mechanisms, often resulting in thyroid dysfunction even in individuals with normal hormone levels [31, 32].

Although research has significantly advanced our understanding of the disease, its pathogenesis remains incompletely defined. Current evidence supports a multifactorial origin involving genetic susceptibility and environmental triggers such as excessive iodine intake, stress, and infections [6, 10, 14].

Patients may present with a wide range of symptoms, including fatigue, mood disturbances, cognitive impairment, and reduced quality of life - even before overt hypothyroidism is diagnosed [1, 14, 16, 32]. The often subtle and chronic onset can delay diagnosis, as nonspecific complaints are frequently overlooked, and routine screening is not consistently implemented [1, 12].

Levothyroxine remains the first-line treatment for overt hypothyroidism; however, many patients report persistent symptoms despite normalized TSH levels, suggesting that hormone replacement may not fully address the underlying autoimmunity [1, 16, 25]. In response, alternative and adjunctive strategies - such as micronutrient supplementation, dietary interventions, and acupuncture - are increasingly being explored to improve outcomes and target immune pathways [4, 20, 32].

This review aims to synthesize and evaluate the current body of knowledge on HT, including its epidemiology, pathophysiology, clinical presentation, diagnostic approaches, conventional treatments, and emerging therapeutic options.

Material and Methods

This review was based on the analysis of 32 peer-reviewed articles concerning Hashimoto's thyroiditis, selected to represent a broad and current overview of the disease. The included publications comprise clinical trials, review papers and epidemiological studies published in recent years. Articles were identified through targeted searches of biomedical databases, primarily PubMed and Google Scholar. The selection focused on studies that directly addressed clinical, pathological, or therapeutic aspects of Hashimoto's thyroiditis.

Description of the state of knowledge

Epidemiology and Risk Factors

Hashimoto's thyroiditis is among the most common endocrine disorders, with its prevalence rising globally. It primarily affects women and most frequently manifests between the ages of 30 and 60, although cases are also observed in children and older adults [1, 13, 26, 30].

Thyroid peroxidase antibodies (TPOAb), a hallmark of the condition, are detected in a significant portion of the general population. Their prevalence is notably higher in women and tends to increase with age. However, only a fraction of seropositive individuals progress to clinical hypothyroidism [13, 14, 16].

Genetic predisposition plays a central role in disease development. Variants in immune-regulatory genes such as HLA, CTLA-4, and PTPN22 have been linked to increased susceptibility, supported by evidence from familial clustering and twin studies [2, 11, 14].

Environmental contributors - including excessive iodine intake, deficiencies in selenium and vitamin D, smoking, infections, stress, and exposure to certain chemicals - are believed to influence disease onset, especially in genetically predisposed individuals [1, 8, 14].

Notably, geographical patterns reveal a higher prevalence of autoimmune thyroiditis in iodine-sufficient and iodine-excess regions, suggesting that iodine may act as a triggering factor [2, 14, 16]. Additionally, its prevalence appears to be higher in both low-middle-income and high-income countries compared to upper-middle-income regions. These disparities may reflect differences in healthcare access, diagnostic practices, environmental exposures, and sociocultural factors [18].

Physiological conditions such as pregnancy and the postpartum period also appear to elevate risk. Fluctuations in regulatory T cells, shifts in cytokine profiles, and fetal microchimerism have been proposed as mechanisms contributing to post-pregnancy thyroid autoimmunity [12, 16, 25].

Lifestyle-related factors - particularly chronic psychological stress and proinflammatory dietary patterns - may additionally modulate immune function. Stress-related dysregulation and increased intestinal permeability (the so-called "leaky gut") have been associated with the development of thyroid autoimmunity [5, 6, 22].

Pathophysiology and Immunological Mechanisms

The pathophysiology of Hashimoto’s thyroiditis involves a complex interplay between innate and adaptive immunity, with autoreactive CD4+ T cells playing a central role in initiating the autoimmune cascade. These cells activate cytotoxic CD8+ T lymphocytes and B lymphocytes, leading to the release of cytotoxic molecules and autoantibodies that contribute to thyroid tissue destruction. Key mediators in this process include Th1 and Th17 cells, which produce proinflammatory cytokines such as IFN-γ, IL-17, and IL-23. These cytokines enhance immune cell recruitment, upregulate MHC class II expression on thyrocytes, and promote apoptosis through Fas receptor signaling. Impaired function or reduced numbers of regulatory T cells (Tregs), often marked by low FOXP3 expression, also contribute to the loss of immune tolerance within the thyroid gland [1, 2, 6].

Both cell-mediated and humoral immune mechanisms are responsible for thyroid gland destruction in Hashimoto’s thyroiditis. B lymphocytes play a dual role: they produce antithyroid

antibodies and act as antigen-presenting cells, thereby stimulating T helper cells and enhancing the autoimmune response. The main autoantigens in Hashimoto's thyroiditis are thyroperoxidase (TPO) and thyroglobulin (Tg), which trigger antibody-dependent and complement-mediated cytotoxicity. Apoptosis of thyrocytes is further mediated by the Fas/FasL pathway, promoted by proinflammatory cytokines such as IFN-γ and IL-1β, and by decreased expression of anti-apoptotic proteins like Bcl-2. These processes lead to irreversible damage to thyroid follicular cells [1, 6, 11].

Recent insights into the disease pathogenesis highlight the role of follicular helper T cells (Tfh), altered microRNA expression, and aberrant antigen presentation. Tfh cells are elevated in the thyroid tissue of patients with Hashimoto's thyroiditis and correlate with higher levels of anti-Tg antibodies, indicating their role in B cell activation and sustained autoantibody production. Dysregulation of microRNAs such as miR-451 and miR-296 has been shown to influence apoptosis and thyroid function, contributing to disease progression. Furthermore, abnormal MHC class II expression on thyrocytes facilitates inappropriate antigen presentation and perpetuates the autoimmune response [1, 2, 6].

In summary, the immunopathogenesis of Hashimoto's thyroiditis is multifactorial, driven by a dysregulated interaction between cellular and humoral immune components. Autoreactive T and B cells, proinflammatory cytokines, and defective regulatory mechanisms orchestrate the progressive destruction of thyroid tissue. Core mechanisms include cytotoxicity driven by Th1 and Th17 cells, failure of Treg-mediated immune tolerance, autoantibody production, and thyrocyte apoptosis. Novel findings such as Tfh involvement, microRNA dysregulation, and abnormal antigen presentation further broaden our understanding of this complex autoimmune disease.

Clinical Presentation

Patients with Hashimoto's thyroiditis may exhibit a variety of non-specific symptoms even when thyroid hormone levels remain within the normal range. During the euthyroid or subclinical phase, commonly reported complaints include fatigue, mood instability, impaired concentration, hair loss, dry skin, and weight gain. These symptoms are often subtle and may go unrecognized for extended periods, contributing to diagnostic delays [1, 14, 16].

As the disease progresses into overt hypothyroidism, patients often develop a wide range of systemic symptoms, including dry and cold skin, hoarseness, facial and peripheral edema, constipation, bradycardia, and continued weight gain. In addition to endocrine disturbances, Hashimoto's thyroiditis is associated with neuropsychiatric manifestations, which may appear

even in euthyroid individuals. Commonly reported symptoms include fatigue, mood swings, depression, anxiety, and cognitive impairments such as reduced concentration and memory deficits. These manifestations may result from underlying autoimmune activity rather than hormone deficiency alone and can significantly impair quality of life [1, 14, 16]. The severity of symptoms varies, and in rare cases, severe hypothyroidism may progress to life-threatening myxedema coma [16].

Rare clinical manifestations include Hashimoto's encephalopathy, a steroid-responsive condition characterized by subacute cognitive decline, behavioral disturbances, seizures, and variable neuroimaging abnormalities. Although thyroid hormone levels are typically normal, this syndrome is associated with high levels of antithyroid antibodies and may present with abnormal EEG or MRI findings [14, 16, 30].

Musculoskeletal involvement is also frequently reported and may significantly interfere with daily functioning. Patients may experience muscle cramps, reduced muscle contractility, and delayed tendon reflexes, often linked to myxedematous infiltration and hypothyroidism. Accumulation of mucopolysaccharides in muscle tissue may lead to stiffness and discomfort. A rare, painful variant of Hashimoto's thyroiditis (p-HT) has also been described, characterized by persistent neck pain unresponsive to analgesics or corticosteroids, with thyroidectomy sometimes being the only effective treatment [7, 16].

Some studies suggest that Hashimoto's thyroiditis (HT) may be associated with an increased risk of developing thyroid carcinoma, particularly papillary thyroid carcinoma (PTC). Observational data have indicated that patients with coexisting HT and PTC often present with more favorable tumor characteristics, including reduced rates of extrathyroidal extension, lymph node metastasis, and recurrence, as well as lower prevalence of the BRAFV600E mutation [15]. However, more recent Mendelian randomization analyses, which assess causality through genetic markers, have not confirmed a direct causal link between HT and thyroid cancer. These findings suggest that the observed associations may be influenced by shared risk factors, diagnostic biases, or heightened surveillance in HT patients rather than a true pathogenic connection [27].

Diagnosis

The diagnosis of Hashimoto's thyroiditis is primarily based on a combination of clinical features, laboratory findings, and imaging. Measurement of serum antithyroid antibodies, particularly anti-thyroid peroxidase antibodies (TPOAb) and anti-thyroglobulin antibodies (TgAb), is fundamental. TPOAb are detected in approximately 90–95% of patients, and TgAb

in 60–80%. However, about 5–10% of patients with Hashimoto's thyroiditis may be seronegative, making imaging, especially thyroid ultrasound, an important complementary diagnostic tool [1, 16, 25, 30].

In addition to standard serological tests, several hematologic indices derived from complete blood count have been investigated as potential diagnostic markers in Hashimoto's thyroiditis. Recent studies indicate that markers such as neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), and pan-immune-inflammation index (PII) are significantly elevated in HT patients compared to healthy controls, regardless of thyroid function status. Although not specific, these indices may serve as supplementary indicators of systemic inflammation and aid in the diagnostic process [19].

Thyroid ultrasound is particularly valuable in cases where antithyroid antibodies are absent. Common sonographic features include reduced echogenicity, parenchymal heterogeneity, increased vascularity, fibrous septa, and hypoechoic pseudonodular lesions. These findings reflect underlying lymphocytic infiltration and fibrotic remodeling. Parenchymal heterogeneity has been associated with elevated antibody levels and higher levothyroxine requirements. Additionally, emerging evidence suggests a link between IgG4-related thyroiditis and diffuse hypoechogenicity, indicating possible subtypes within the Hashimoto's disease spectrum [16, 28].

Fine-needle aspiration biopsy (FNA) is not routinely indicated in all patients with Hashimoto's thyroiditis but becomes essential when thyroid nodules are present or malignancy is suspected. Characteristic cytological findings include dense lymphocytic infiltration and Hürthle cell changes, which reflect ongoing autoimmune inflammation. FNA can be particularly helpful in seronegative cases or when ultrasound features are atypical. The presence of lymphocytes in direct contact with follicular cells supports the diagnosis of autoimmune thyroiditis. Furthermore, studies have shown that inflammatory changes may mimic or coexist with malignancy, highlighting the importance of integrating FNA results with clinical and imaging findings to ensure accurate diagnosis and appropriate management [1, 3, 14, 23].

Treatment

Levothyroxine (LT4) is the standard therapy for overt hypothyroidism in patients with Hashimoto's thyroiditis. The primary goal of treatment is to restore biochemical euthyroidism by normalizing serum thyroid-stimulating hormone (TSH) levels. The recommended daily dose of LT4 typically ranges from 1.4 to 1.8 μg per kilogram of body weight, although some sources suggest 1.5–1.7 μg/kg depending on factors such as age, lean body mass, and residual thyroid

function. Regular monitoring and dose adjustments every 6 to 8 weeks are advised until target TSH levels are achieved [1, 12, 16].

While tablet formulations of levothyroxine are most commonly prescribed, alternative preparations such as soft-gel capsules and liquid solutions may offer improved absorption in patients with gastrointestinal disorders, elderly individuals, or those taking interfering medications. These alternative forms can help achieve stable hormone levels in cases of unexplained TSH variability [12].

Despite normalization of TSH, many patients continue to report persistent symptoms such as fatigue, weight gain, or cognitive dysfunction. These may be attributed to irreversible tissue changes, coexisting conditions, or sustained autoimmune activity. Studies indicate that up to one-third of treated patients fall outside the optimal TSH range, and 5–10% experience ongoing symptoms despite biochemical control. Additionally, LT4 therapy may influence immunological parameters such as interleukin-12 and contribute to reductions in antithyroid antibody levels [1, 16, 25].

Adjunctive therapies including selenium, vitamin D, and myo-inositol have attracted growing interest in the management of Hashimoto's thyroiditis. Selenium, as a key component of deiodinases and glutathione peroxidases, supports thyroid hormone metabolism and antioxidant defense. Meta-analyses and clinical trials have shown that selenium supplementation may reduce TPOAb levels and modestly improve thyroid function, particularly in selenium-deficient individuals [4, 20].

Vitamin D has also been implicated in thyroid autoimmunity. Observational and interventional studies reveal an inverse relationship between vitamin D status and antithyroid antibody levels. Supplementation with moderate doses of vitamin D appears to lower both anti-TPO and anti-Tg antibodies and may aid in modulating the immune response [20, 24].

Myo-inositol, especially when used in combination with selenium, has demonstrated promising effects in patients with subclinical hypothyroidism or early-stage Hashimoto's disease. Clinical trials have been shown to reduce TSH and antithyroid antibody levels, likely through modulation of TSH signaling and a decrease in proinflammatory chemokines [20, 25].

Although pharmacological therapy remains the mainstay of Hashimoto's thyroiditis management, thyroidectomy may be considered in selected cases. Surgery is typically reserved for patients with compressive symptoms, suspected malignancy, or severe pain unresponsive to medical treatment. Painful Hashimoto's thyroiditis (p-HT) is a rare variant that may lead to chronic, drug-resistant neck pain. In such cases, total thyroidectomy has been shown to provide significant symptom relief, even when corticosteroids and analgesics are ineffective [7, 14].

Recent randomized controlled trials have also evaluated thyroidectomy in euthyroid patients with persistent symptoms despite optimized levothyroxine therapy. One such study demonstrated significant improvements in fatigue, quality of life, and a reduction in anti-TPO antibody levels 12 to 18 months after surgery. These findings support thyroidectomy as a potential therapeutic option for selected patients with refractory symptoms and high antibody titers [16, 25]. However, it is important to note that surgery in the context of Hashimoto's thyroiditis carries an elevated risk of complications due to dense fibrosis and altered glandular anatomy. For this reason, surgical intervention should be reserved for well-selected patients and performed by experienced thyroid surgeons [14].

Complementary and Alternative Therapies

In recent years, complementary and alternative therapies have gained attention as supportive strategies in the management of Hashimoto's thyroiditis. These approaches are especially considered for patients who continue to experience symptoms despite adequate pharmacological treatment or for those with subclinical thyroid dysfunction.

Acupuncture is one of the best-studied complementary interventions. A systematic review and meta-analysis of 14 randomized controlled trials showed that acupuncture can significantly lower levels of TPOAb and TGAb, while also improving thyroid hormone profiles (TSH, FT3, FT4) and quality of life. Patients reported fewer symptoms such as fatigue and mood changes [21]. Additionally, an ongoing clinical trial is evaluating the effectiveness of penetration needling along the Hand-Yangming meridian. This study is not only measuring hormonal and immunological changes but also assessing outcomes like anxiety, depression, and quality of life using validated psychological tools. Together, these findings reflect growing interest in integrating acupuncture into individualized care models for Hashimoto's disease [21, 32].

Lifestyle and dietary changes are also recognized as important elements of holistic management. A balanced diet, good sleep hygiene, and regular physical activity support immune regulation and thyroid health. Some patients benefit from removing specific food components like gluten or lactose, while others focus on increasing their intake of antioxidant-rich foods and nutrients such as selenium, vitamin D, and iron [1, 25]. Dietary patterns like the Mediterranean and paleolithic diets may offer additional benefits. For example, the Mediterranean diet is thought to lower oxidative stress, while the paleolithic diet may improve general well-being due to its high nutritional density. On the other hand, diets high in processed meats have been associated with a higher risk of thyroid autoimmunity [5, 25]. Importantly, excess body weight is also a risk factor, and weight management has been linked to better outcomes in people with

Hashimoto's. Targeted lifestyle changes - including smoking cessation, improved sleep, and increased physical activity - can play a meaningful role in relieving symptoms, particularly in those with subclinical hypothyroidism [25].

Another area of growing interest is the gut–thyroid connection. Many patients with Hashimoto's show signs of altered gut microbiota, increased intestinal permeability ("leaky gut"), and reduced microbial diversity. Elevated serum zonulin levels and shifts in key bacterial species such as *Bacteroides*, *Bifidobacterium*, and *Lactobacillus* have been observed. These changes may contribute to immune dysregulation by weakening the gut barrier and triggering systemic inflammation [22, 26]. Recent sequencing data further confirm that gut microbial composition becomes progressively more altered as Hashimoto's disease advances, with shifts influenced by hormone levels and gender, and involving both beneficial and proinflammatory bacterial genera [29].

To address this, probiotics have been studied for their ability to restore microbial balance and reinforce the intestinal lining. Strains of *Lactobacillus* and *Bifidobacterium* may help stabilize thyroid function, and their metabolic byproducts - known as postbiotics - have immunomodulatory effects. These include short-chain fatty acids (SCFAs), bacterial lysates, and other compounds that can influence the behavior of immune cells. Animal studies have shown that SCFAs can promote regulatory T cell responses and reduce proinflammatory cytokines. In addition, the gut microbiota appears to shape the development of Treg and Th17 cells, both of which are deeply involved in the pathogenesis of autoimmune thyroiditis. Although research in humans is still limited, these findings point to microbiota-targeted strategies - such as probiotics, prebiotics, and dietary modifications - as potentially valuable additions to standard care [9, 17, 22].

Conclusions

Hashimoto's thyroiditis is a multifaceted autoimmune disease shaped by genetic, environmental, and immune-endocrine factors. While our understanding of its pathogenesis has grown - particularly in terms of lymphocyte activity, cytokine signaling, and thyroid-specific autoantibodies - many aspects remain unresolved. Clinical symptoms can range from barely noticeable to deeply disruptive, sometimes affecting patients' well-being even when hormone levels appear normal.

Diagnosis typically combines clinical evaluation, thyroid function tests, antibody levels, and ultrasound imaging. In more complex cases - such as seronegative presentations or suspicious nodules - fine-needle aspiration or histological analysis may be helpful.

Levothyroxine remains the first-line treatment for hypothyroidism in Hashimoto's, but many patients continue to struggle with fatigue, cognitive issues, or mood changes despite achieving target TSH levels. As a result, interest has grown in supportive strategies like selenium, vitamin D, and inositol supplementation, as well as acupuncture and lifestyle-based interventions. For some individuals with persistent symptoms, surgery may even be considered.

Ultimately, managing Hashimoto's thyroiditis requires an individualized, patient-centered approach that goes beyond lab values. Future research should focus on identifying biomarkers of disease activity, improving treatment personalization, and helping patients feel better - not just look better on paper.

Disclosure

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References

1. Wrońska K, Hałasa M, Szczuko M. The Role of the Immune System in the Course of Hashimoto's Thyroiditis: The Current State of Knowledge. *IJMS*. 2024;25(13):6883. doi:10.3390/ijms25136883

2. Ajjan RA, Weetman AP. The Pathogenesis of Hashimoto's Thyroiditis: Further Developments in our Understanding. *Horm Metab Res*. 2015;47(10):702-710. doi:10.1055/s-0035-1548832

3. Kust D, Matesa N. The impact of familial predisposition on the development of Hashimoto's thyroiditis. *Acta Clinica Belgica*. 2020;75(2):104-108. doi:10.1080/17843286.2018.1555115

4. Huwiler VV, Maissen-Abgottspon S, Stanga Z, et al. Selenium Supplementation in Patients with Hashimoto Thyroiditis: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. *Thyroid*®. 2024;34(3):295-313. doi:10.1089/thy.2023.0556

5. Chen S, Peng Y, Zhang H, Zou Y. Relationship between thyroid function and dietary inflammatory index in Hashimoto thyroiditis patients. *Medicine*. 2023;102(46):e35951. doi:10.1097/MD.0000000000035951

6. Jin B, Wang S, Fan Z. Pathogenesis Markers of Hashimoto's Disease—A Mini Review. *Front Biosci (Landmark Ed)*. 2022;27(10):297. doi:10.31083/j.fbl2710297

7. Rotondi M, Capelli V, Locantore P, Pontecorvi A, Chiovato L. Painful Hashimoto's thyroiditis: myth or reality? *J Endocrinol Invest*. 2017;40(8):815-818. doi:10.1007/s40618-017-0655-5

8. Vargas-Uricoechea H. Molecular Mechanisms in Autoimmune Thyroid Disease. *Cells*. 2023;12(6):918. doi:10.3390/cells12060918

9. Zhu X, Zhang C, Feng S, He R, Zhang S. Intestinal microbiota regulates the gut-thyroid axis: the new dawn of improving Hashimoto thyroiditis. *Clin Exp Med*. 2024;24(1):39. doi:10.1007/s10238-024-01304-4

10. Qiu K, Li K, Zeng T, et al. Integrative Analyses of Genes Associated with Hashimoto's Thyroiditis. Duan L, ed. *Journal of Immunology Research*. 2021;2021:1-9. doi:10.1155/2021/8263829

11. Jabrocka-Hybel A, Skalniak A, Piątkowski J, et al. How much of the predisposition to Hashimoto's thyroiditis can be explained based on previously reported associations? *J Endocrinol Invest*. 2018;41(12):1409-1416. doi:10.1007/s40618-018-0910-4

12. Ragusa F, Fallahi P, Elia G, et al. Hashimotos' thyroiditis: Epidemiology, pathogenesis, clinic and therapy. *Best Practice & Research Clinical Endocrinology & Metabolism*. 2019;33(6):101367. doi:10.1016/j.beem.2019.101367

13. Bothra N, Shah N, Goroshi M, et al. Hashimoto's thyroiditis: relative recurrence risk ratio and implications for screening of first-degree relatives. *Clinical Endocrinology*. 2017;87(2):201-206. doi:10.1111/cen.13323

14. Ralli M, Angeletti D, Fiore M, et al. Hashimoto's thyroiditis: An update on pathogenic mechanisms, diagnostic protocols, therapeutic strategies, and potential malignant transformation. *Autoimmunity Reviews*. 2020;19(10):102649. doi:10.1016/j.autrev.2020.102649

15. Xu J, Ding K, Mu L, et al. Hashimoto's Thyroiditis: A "Double-Edged Sword" in Thyroid Carcinoma. *Front Endocrinol*. 2022;13:801925. doi:10.3389/fendo.2022.801925

16. Klubo-Gwiezdzinska J, Wartofsky L. Hashimoto thyroiditis: an evidence-based guide: etiology, diagnosis and treatment. *Polish Archives of Internal Medicine*. Published online March 3, 2022. doi:10.20452/pamw.16222

17. Virili C, Fallahi P, Antonelli A, Benvenga S, Centanni M. Gut microbiota and Hashimoto's thyroiditis. *Rev Endocr Metab Disord*. 2018;19(4):293-300. doi:10.1007/s11154-018-9467-y

18. Hu X, Chen Y, Shen Y, Tian R, Sheng Y, Que H. Global prevalence and epidemiological trends of Hashimoto's thyroiditis in adults: A systematic review and meta-analysis. *Front Public Health*. 2022;10:1020709. doi:10.3389/fpubh.2022.1020709

19. Bozdag A, Gundogan Bozdag P. Evaluation of systemic inflammation markers in patients with Hashimoto's thyroiditis. *J Int Med Res*. 2024;52(9):03000605241280049. doi:10.1177/03000605241280049

20. Peng B, Wang W, Gu Q, Wang P, Teng W, Shan Z. Effects of different supplements on Hashimoto's thyroiditis: a systematic review and network meta-analysis. *Front Endocrinol*. 2024;15:1445878. doi:10.3389/fendo.2024.1445878

21. Wang X, Li Y, Xie H, et al. Effect of acupuncture on Hashimoto thyroiditis: A systematic review and meta-analysis. *Medicine*. 2024;103(9):e37326. doi:10.1097/MD.0000000000037326

22. Cayres LCDF, De Salis LVV, Rodrigues GSP, et al. Detection of Alterations in the Gut Microbiota and Intestinal Permeability in Patients With Hashimoto Thyroiditis. *Front Immunol*. 2021;12:579140. doi:10.3389/fimmu.2021.579140

23. Hu X, Wang X, Liang Y, et al. Cancer Risk in Hashimoto's Thyroiditis: a Systematic Review and Meta-Analysis. *Front Endocrinol*. 2022;13:937871. doi:10.3389/fendo.2022.937871

24. Chahardoli R, Saboor-Yaraghi AA, Amouzegar A, Khalili D, Vakili A, Azizi F. Can Supplementation with Vitamin D Modify Thyroid Autoantibodies (Anti-TPO Ab, Anti-Tg Ab) and Thyroid Profile (T3, T4, TSH) in Hashimoto's Thyroiditis? A Double Blind, Randomized Clinical Trial. *Horm Metab Res*. 2019;51(05):296-301. doi:10.1055/a-0856-1044

25. Tywanek E, Michalak A, Świrska J, Zwolak A. Autoimmunity, New Potential Biomarkers and the Thyroid Gland—The Perspective of Hashimoto's Thyroiditis and Its Treatment. *IJMS*. 2024;25(9):4703. doi:10.3390/ijms25094703

26. Gong B, Wang C, Meng F, et al. Association Between Gut Microbiota and Autoimmune Thyroid Disease: A Systematic Review and Meta-Analysis. *Front Endocrinol*. 2021;12:774362. doi:10.3389/fendo.2021.774362

27. Zhang Q, Lan X. Assessment of causal association between autoimmune thyroiditis and thyroid cancer: A Mendelian randomization study. *Medicine*. 2025;104(9):e41633. doi:10.1097/MD.0000000000041633

28. Kenarlı K, Bahçecioğlu AB, Aksu ÖB, Güllü S. Are sonographic characteristics of Hashimoto's thyroiditis related with immunologic parameters? A cross-sectional study. *J Endocrinol Invest*. 2024;47(7):1701-1709. doi:10.1007/s40618-023-02286-y

29. Liu J, Qin X, Lin B, et al. Analysis of gut microbiota diversity in Hashimoto's thyroiditis patients. *BMC Microbiol*. 2022;22(1):318. doi:10.1186/s12866-022-02739-z

30. Weetman AP. An update on the pathogenesis of Hashimoto's thyroiditis. *J Endocrinol Invest*. 2021;44(5):883-890. doi:10.1007/s40618-020-01477-1

31. Dove AE, Marathe PH, Gao HX, Close KL. American Association of Clinical Endocrinologists 2017. *Journal of Diabetes*. 2017;9(9):817-820. doi:10.1111/1753-0407.12573

32. Wang S, Zhao J, Zeng W, et al. Acupuncture for Hashimoto thyroiditis: study protocol for a randomized controlled trial. *Trials*. 2021;22(1):74. doi:10.1186/s13063-021-05036-8