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AUTOIMMUNE THYROID DISEASES: A COMPREHENSIVE LITERATURE REVIEW OF TREATMENT AND THE CURRENT STATE OF KNOWLEDGE

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ABSTRACT

Introduction and Purpose: Autoimmune thyroid diseases (AITDs) are among the most common organ-specific autoimmune disorders worldwide. The two major forms, Hashimoto's thyroiditis and Graves' disease, result from a complex interaction between genetic susceptibility, environmental triggers, and immune dysregulation. These conditions significantly affect quality of life and are associated with numerous endocrine and non-endocrine complications. The purpose of this review is to summarize current knowledge regarding the epidemiology, pathophysiology, diagnosis, treatment, and emerging therapeutic approaches for autoimmune thyroid diseases.

Description of the State of Knowledge: AITDs develop due to the loss of immune tolerance toward thyroid antigens, particularly thyroid peroxidase (TPO), thyroglobulin (Tg), and the thyroid-stimulating hormone receptor (TSHR). Hashimoto's thyroiditis is characterized predominantly by lymphocytic infiltration and progressive destruction of thyroid tissue, resulting in hypothyroidism, whereas Graves' disease is mediated by stimulating autoantibodies directed against TSH receptors, leading to hyperthyroidism. Current diagnostic strategies rely on clinical evaluation, measurement of thyroid hormone levels, detection of thyroid-specific autoantibodies, and thyroid ultrasonography.

Standard treatment includes levothyroxine replacement therapy for Hashimoto's thyroiditis and antithyroid drugs, radioactive iodine therapy, or thyroidectomy for Graves' disease. Recent research focuses on biological therapies, immunomodulatory agents, and precision medicine approaches aimed at modifying disease progression and improving long-term outcomes.

Conclusion: The understanding of autoimmune thyroid diseases continues to evolve. Although current therapeutic options effectively manage hormonal dysfunction, they do not directly address the underlying autoimmune process. Further research into disease mechanisms and targeted therapies may lead to more personalized and disease-modifying treatment strategies in the future.

KEYWORDS

Hashimoto's Thyroiditis, Graves' Disease, Autoimmunity, Thyroid Peroxidase Antibodies, Thyroid-stimulating Hormone Receptor Antibodies, Levothyroxine

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Introduction

Autoimmune thyroid diseases (AITDs) constitute a major global health burden and represent the most prevalent group of organ-specific autoimmune disorders, affecting millions of individuals worldwide [1,2]. The development of AITDs is multifactorial and involves a complex interaction between genetic susceptibility, environmental factors, epigenetic mechanisms, and dysregulation of both innate and adaptive immune responses. Hashimoto's thyroiditis is characterized by progressive lymphocytic infiltration of the thyroid gland, destruction of thyroid follicular cells, and the gradual development of hypothyroidism. In contrast, Graves' disease is caused by stimulatory autoantibodies directed against the thyroid-stimulating hormone receptor (TSHR), leading to excessive thyroid hormone production and hyperthyroidism [3,9].

Although these disorders differ substantially in their clinical manifestations and effects on thyroid function, they share common autoimmune mechanisms involving autoreactive T lymphocytes, B lymphocytes, and thyroid-specific autoantibodies. Autoimmune thyroid diseases occur significantly more frequently in women than in men, suggesting an important role of sex hormones, X-chromosome-related mechanisms, and genetic susceptibility in disease development [3].

Beyond thyroid dysfunction, AITDs are frequently associated with other autoimmune diseases, including type 1 diabetes mellitus, celiac disease, rheumatoid arthritis, and systemic lupus erythematosus. Untreated thyroid dysfunction may result in significant cardiovascular, metabolic, reproductive, and neuropsychiatric complications, thereby substantially reducing patients' quality of life [3,10].

The aim of this literature review is to summarize current knowledge regarding the epidemiology, pathophysiology, clinical presentation, diagnosis, and treatment of autoimmune thyroid diseases, as well as to discuss emerging therapeutic strategies and future directions in research.

Methodology

A literature search was conducted using PubMed, Scopus, and Web of Science databases. Articles published between January 2015 and February 2026 were screened using combinations of the following keywords: "Autoimmune Thyroid Disease", "Hashimoto's Thyroiditis", "Graves' Disease", "Thyroid Autoimmunity", "Diagnosis", and "Treatment". Original research articles, systematic reviews, meta-analyses, and clinical guidelines published in English were considered eligible. Studies lacking relevance to the scope of the review, duplicate records, and conference abstracts were excluded. Following the screening and eligibility assessment process, the most relevant publications were selected for qualitative synthesis and critical analysis.

Epidemiology

Following the methodological framework described above, the selected studies provided insight into several key aspects of autoimmune thyroid diseases, including their epidemiology, pathogenesis, diagnosis, and management. The epidemiological characteristics of AITDs are discussed below.

Recent epidemiological evidences suggest that the prevalence of autoimmune thyroid diseases has increased over recent decades, potentially due to improved diagnostic capabilities, environmental changes, and growing exposure to immune-modulating factors.

Autoimmune thyroid diseases (AITDs) are among the most prevalent autoimmune disorders worldwide, affecting approximately 5% of the general population, although the prevalence varies depending on the population studied and the diagnostic criteria used. Hashimoto's thyroiditis (HT) is the most common autoimmune thyroid disorder and the leading cause of hypothyroidism in regions with sufficient iodine intake[1].

The global prevalence of Hashimoto's thyroiditis is estimated to range from 7% to 13% in the adult population, with higher rates observed when thyroid autoantibodies and ultrasonographic findings are included in the diagnostic criteria. Graves' disease (GD), the most common cause of autoimmune hyperthyroidism, affects approximately 1–2% of the population and accounts for 60–80% of hyperthyroidism cases in iodine-sufficient regions [2,4,11].

Both Hashimoto's thyroiditis and Graves' disease occur significantly more frequently in women than in men. The female-to-male ratio is estimated to be approximately 7–10:1 for Hashimoto's thyroiditis and 5–10:1 for Graves' disease, suggesting a substantial influence of sex hormones, X chromosome-related factors, and immune regulatory mechanisms on disease susceptibility [3,12].

The incidence of autoimmune thyroid diseases increases with age, particularly in middle-aged and older women, although both disorders may occur in children and adolescents. Geographical variation also plays an important role in the epidemiology of AITDs [1].

Higher prevalence rates have been reported in North America and Europe, whereas lower rates are generally observed in certain regions of Africa and Asia. These differences are partly related to genetic background, iodine intake, environmental exposures, and healthcare accessibility [11].

Pathophysiology

The pathogenesis of autoimmune thyroid diseases is multifactorial and results from a complex interplay between genetic susceptibility, environmental exposures, epigenetic influences, and immune dysregulation. Genetic predisposition plays a crucial role in the development of AITDs, and numerous studies have demonstrated associations with specific human leukocyte antigen (HLA) alleles, particularly HLA-DR3, HLA-DR4, and HLA-DR5. In addition to HLA genes, polymorphisms in immune-regulatory genes such as CTLA-4 (Cytotoxic T-Lymphocyte Associated Protein 4) and PTPN22 (Protein Tyrosine Phosphatase Non-Receptor Type 22) contribute significantly to disease susceptibility by affecting T-cell activation and immune tolerance mechanisms [13].

However, genetic predisposition alone does not fully explain disease onset, highlighting the critical role of environmental and epigenetic modifiers. Several environmental factors have been implicated in the

development of AITDs, including excessive iodine intake, viral and bacterial infections, cigarette smoking, chronic stress, selenium deficiency, vitamin D deficiency, and exposure to endocrine-disrupting chemicals [3].

A key event in the pathogenesis of AITDs is the loss of immune tolerance to thyroid antigens, leading to the activation of autoreactive T and B lymphocytes.

Following the breakdown of immune tolerance, both cellular and humoral immune responses become activated and contribute to the development of chronic thyroid inflammation.

Autoreactive CD4⁺ T lymphocytes recognize thyroid antigens presented by antigen-presenting cells and initiate an inflammatory response through the secretion of pro-inflammatory cytokines, including interferon-gamma (IFN- γ), interleukin-17 (IL-17), and tumor necrosis factor-alpha (TNF- α). This process promotes the recruitment of additional immune cells into thyroid tissue and contributes to the maintenance of chronic inflammation.

Increasing evidence indicates that an imbalance between pro-inflammatory Th17 lymphocytes and regulatory T cells (Tregs) plays a central role in the pathogenesis of autoimmune thyroid diseases. Reduced Treg activity may contribute to the breakdown of immune tolerance, whereas excessive Th17 responses promote chronic thyroid inflammation and tissue damage [13].

Simultaneously, activated B lymphocytes differentiate into plasma cells that produce autoantibodies directed against thyroid-specific antigens.

The most important autoantibodies detected in Hashimoto's thyroiditis are thyroid peroxidase antibodies (TPOAb) and thyroglobulin antibodies (TgAb). Thyroid peroxidase antibodies are present in more than 90% of patients with Hashimoto's thyroiditis and represent the most sensitive serological marker of the disease [3].

In Graves' disease, thyroid-stimulating hormone receptor antibodies (TRAb) play a central role by binding to TSH receptors on thyroid follicular cells and stimulating excessive thyroid hormone synthesis and secretion.

These immune-mediated mechanisms ultimately result in progressive thyroid tissue destruction and hypothyroidism in Hashimoto's thyroiditis or persistent hyperthyroidism in Graves' disease [2].

Clinical presentation

The immunological mechanisms described above translate directly into the diverse clinical manifestations observed in patients with autoimmune thyroid diseases. Consequently, the clinical presentation of AITDs varies depending on the underlying disorder and the degree of thyroid dysfunction.

The clinical manifestations of autoimmune thyroid diseases vary depending on the underlying disorder and the degree of thyroid dysfunction. Hashimoto's thyroiditis is typically associated with hypothyroidism, whereas Graves' disease is characterized by hyperthyroidism and its systemic effects [2,3,6,8].

Hashimoto's thyroiditis

Hashimoto's thyroiditis usually presents with nonspecific symptoms that develop gradually over time. Among the clinical manifestations of hypothyroidism associated with Hashimoto's thyroiditis, fatigue remains one of the most prevalent symptoms and is recognized as a major contributor to reduced quality of life and impaired daily functioning [3].

Patients commonly experience weight gain due to a reduction in basal metabolic rate associated with thyroid hormone deficiency. Cold intolerance is another characteristic manifestation, resulting from decreased thermogenesis and impaired heat production [14]. Many individuals also develop neuropsychiatric symptoms, including depression, cognitive slowing, impaired concentration, and memory disturbances.

Additional clinical features may include dry skin, constipation, bradycardia, muscle weakness, and menstrual irregularities [3].

Graves' disease

Graves' disease is the most common cause of hyperthyroidism and results from excessive stimulation of the thyroid gland by thyroid-stimulating hormone receptor antibodies (TRAb).

Unintentional weight loss despite normal or increased appetite is a common symptom caused by an increased metabolic rate [2]. Cardiovascular manifestations are frequently observed, with tachycardia being one of the hallmark clinical signs of the disease. Patients often present with nervousness, irritability, emotional lability, anxiety, and hyperactivity as a consequence of excessive thyroid hormone activity [2]. A distinctive feature of Graves' disease is thyroid eye disease, also known as Graves' orbitopathy, which is characterized by orbital inflammation, proptosis, eyelid retraction, diplopia, and, in severe cases, vision impairment. The

severity of orbitopathy varies considerably among patients and is strongly associated with smoking and high serum TRAb concentrations [15].

Other common manifestations of Graves' disease include heat intolerance, excessive sweating, tremor, increased bowel frequency, muscle weakness, and sleep disturbances. Beyond the direct manifestations of thyroid dysfunction, autoimmune thyroid diseases are frequently associated with a broad spectrum of other autoimmune conditions.

Associated conditions

Autoimmune thyroid diseases (AITDs) frequently coexist with other autoimmune disorders, reflecting shared genetic susceptibility, common immunological pathways, and overlapping environmental risk factors. Patients with Hashimoto's thyroiditis and Graves' disease have a significantly increased risk of developing additional autoimmune conditions compared with the general population [3].

Type 1 diabetes mellitus (T1DM) is one of the most common autoimmune diseases associated with AITDs. The coexistence of these disorders is largely attributed to shared genetic factors, particularly HLA-related susceptibility genes, and patients with T1DM are routinely screened for thyroid autoimmunity [16].

Celiac disease is another well-recognized comorbidity of autoimmune thyroid diseases. Both conditions share common genetic predispositions, including HLA-DQ2 and HLA-DQ8 haplotypes, and studies have demonstrated a higher prevalence of celiac disease among patients with autoimmune thyroid disorders compared with the general population [17].

Addison's disease frequently occurs in association with autoimmune thyroid diseases as part of autoimmune polyglandular syndromes, particularly autoimmune polyglandular syndrome type 2. The coexistence of adrenal insufficiency and thyroid dysfunction may complicate diagnosis and management, requiring careful endocrine evaluation [1].

Vitiligo, characterized by autoimmune destruction of melanocytes, has also been linked to thyroid autoimmunity. Several studies have reported an increased prevalence of thyroid autoantibodies and thyroid dysfunction among patients with vitiligo, supporting the existence of shared autoimmune mechanisms [18].

Rheumatoid arthritis (RA) is another autoimmune condition commonly associated with AITDs. Chronic systemic inflammation, similar genetic predisposition, and immune dysregulation contribute to the frequent coexistence of these disorders [19].

Systemic lupus erythematosus (SLE) has also been associated with an increased prevalence of autoimmune thyroid diseases. Patients with SLE demonstrate a higher frequency of thyroid autoantibodies and thyroid dysfunction than healthy individuals, suggesting overlapping pathogenic pathways involving B-cell hyperactivity and loss of immune tolerance [20].

Due to the increased risk of coexisting autoimmune disorders, clinicians should maintain a high index of suspicion and consider appropriate screening strategies in patients diagnosed with autoimmune thyroid diseases [3].

Diagnosis

Given the heterogeneity of clinical manifestations and the frequent overlap with other autoimmune disorders, accurate diagnosis requires a comprehensive assessment combining clinical, biochemical, immunological, and imaging findings [3].

Serum thyroid-stimulating hormone (TSH) concentration is considered the most sensitive initial test for assessing thyroid function. Elevated TSH levels are typically observed in hypothyroidism associated with Hashimoto's thyroiditis, whereas suppressed TSH levels are characteristic of hyperthyroidism in Graves' disease.

Measurement of free thyroxine (fT4) and free triiodothyronine (fT3) is essential for determining the severity of thyroid dysfunction and distinguishing between overt and subclinical disease [2].

The detection of thyroid-specific autoantibodies plays a crucial role in confirming the autoimmune nature of thyroid disorders. Anti-thyroid peroxidase antibodies (anti-TPO) are the most sensitive serological markers of Hashimoto's thyroiditis and are present in the majority of affected patients. Anti-thyroglobulin antibodies (anti-Tg) may also be detected and can provide additional diagnostic value, particularly in anti-TPO-negative individuals [3].

In Graves' disease, thyroid-stimulating hormone receptor antibodies (TRAb) represent the most specific immunological marker and are useful for confirming the diagnosis, assessing disease activity, and predicting the risk of relapse [2].

Thyroid ultrasonography is a widely used imaging modality that provides valuable information regarding gland size, echogenicity, vascularization, and structural abnormalities. In Hashimoto's thyroiditis, ultrasound typically reveals a diffusely hypoechoic and heterogeneous gland, whereas Graves' disease is often characterized by increased vascularity, commonly referred to as the "thyroid inferno" pattern [3].

Fine-needle aspiration biopsy (FNAB) is not routinely required for the diagnosis of autoimmune thyroid diseases. However, it may be indicated in selected cases, particularly when thyroid nodules, suspicious ultrasound findings, or concerns regarding malignancy are present [14].

A comprehensive diagnostic approach combining biochemical, immunological, and imaging assessments allows for accurate diagnosis and appropriate management of autoimmune thyroid diseases.

Once the diagnosis has been established, treatment strategies should be tailored according to the specific autoimmune thyroid disorder and the degree of thyroid dysfunction.

Treatment

The management of autoimmune thyroid diseases (AITDs) depends on the underlying disorder and the severity of thyroid dysfunction. Current treatment strategies primarily focus on restoring and maintaining normal thyroid hormone levels, alleviating symptoms, and preventing long-term complications [2,3].

Hashimoto's thyroiditis

Levothyroxine (L-T4) replacement therapy is the standard treatment for Hashimoto's thyroiditis and remains the most effective approach for managing hypothyroidism. The primary goal of treatment is to normalize serum TSH levels and restore euthyroidism, thereby improving clinical symptoms and preventing complications associated with thyroid hormone deficiency [3].

The dosage of levothyroxine should be individualized based on factors such as age, body weight, severity of hypothyroidism, cardiovascular status, and pregnancy. Regular monitoring of TSH levels is recommended to ensure adequate treatment and optimize therapeutic outcomes.

Graves' disease

In Graves' disease, treatment is aimed at reducing excessive thyroid hormone production and controlling symptoms. Antithyroid drugs are generally considered the first-line treatment in many patients [2].

Methimazole is the preferred antithyroid medication due to its efficacy, longer duration of action, and lower risk of adverse effects compared with other agents. It inhibits thyroid hormone synthesis by blocking thyroid peroxidase activity and is recommended for most patients with Graves' disease [2,4,5].

Propylthiouracil (PTU) is an alternative antithyroid drug that inhibits thyroid hormone synthesis and additionally reduces the peripheral conversion of thyroxine (T4) to triiodothyronine (T3). Its use is generally reserved for specific situations, such as the first trimester of pregnancy, thyroid storm, or intolerance to methimazole [2].

Radioactive iodine (RAI) therapy is a well-established treatment option that destroys thyroid tissue through the uptake of radioactive iodine-131. It is widely used in adults with Graves' disease, particularly in cases of relapse after antithyroid drug therapy or when long-term medical treatment is not desired [2,5,6].

Thyroidectomy provides a definitive treatment for Graves' disease and may be indicated in patients with large goiters, suspected thyroid malignancy, severe compressive symptoms, or contraindications to radioactive iodine therapy. Although highly effective, surgical treatment carries potential risks, including recurrent laryngeal nerve injury and hypoparathyroidism [2,7].

Treatment selection should be individualized and guided by disease severity, patient characteristics, comorbidities, and patient preferences. A multidisciplinary approach is often necessary to achieve optimal long-term outcomes and improve quality of life.

Research into new treatment methods

While conventional therapies remain highly effective in correcting thyroid hormone abnormalities and controlling symptoms, they do not directly target the autoimmune mechanisms responsible for disease initiation and progression. Consequently, increasing research efforts are focused on the development of disease-modifying therapies aimed at regulating immune responses and preventing thyroid tissue damage [13].

One of the most extensively studied biological therapies is rituximab, a monoclonal antibody targeting the CD20 antigen expressed on B lymphocytes. By reducing the number of B cells involved in autoantibody

production, rituximab has demonstrated potential benefits in selected patients with Graves' orbitopathy and other autoimmune disorders [15].

Teprotumumab, a monoclonal antibody directed against the insulin-like growth factor 1 receptor (IGF-1R), represents a major advancement in the treatment of thyroid eye disease associated with Graves' disease. Clinical trials have shown significant improvements in proptosis, diplopia, and quality of life, making teprotumumab the first targeted therapy approved specifically for thyroid eye disease [21].

Mycophenolate mofetil, an immunosuppressive agent that inhibits lymphocyte proliferation, has also emerged as a promising treatment option for moderate-to-severe Graves' orbitopathy. Studies suggest that combination therapy with glucocorticoids may improve treatment outcomes compared with conventional therapy alone [22].

Additional investigational approaches include biologic agents targeting B-cell signaling pathways and regulatory T-cell (Treg) therapies aimed at restoring immune tolerance [13].

Personalized immunotherapy is also gaining attention as a future therapeutic strategy. Advances in genomics, immunophenotyping, and biomarker identification may allow clinicians to tailor treatment according to individual genetic and immunological profiles, improving efficacy while reducing adverse effects [9].

Although many of these approaches remain under investigation, they represent promising avenues for the development of therapies capable of modifying the natural course of autoimmune thyroid diseases rather than simply correcting hormonal abnormalities. Continued research is necessary to establish their long-term safety, efficacy, and clinical applicability.

Despite promising results, most biologic therapies remain limited by high treatment costs, restricted availability, and insufficient long-term safety data. Furthermore, evidence regarding their effectiveness in modifying the underlying autoimmune process remains inconclusive, highlighting the need for larger randomized clinical trials.

Monitoring

Regardless of the therapeutic approach employed, long-term follow-up remains an essential component of patient care due to the chronic nature of autoimmune thyroid diseases and the risk of relapse or progression. Regular monitoring allows clinicians to assess treatment effectiveness, maintain euthyroidism, and identify disease-related complications at an early stage. Serum thyroid-stimulating hormone (TSH) remains the most important parameter for long-term follow-up and treatment adjustment. Measurement of free thyroxine (fT4) is also recommended, particularly during the initiation of therapy and in patients with unstable thyroid function [3].

The assessment of thyroid-specific autoantibodies may provide additional information regarding disease activity. Anti-thyroid peroxidase antibodies (anti-TPO) and anti-thyroglobulin antibodies (anti-Tg) are commonly detected in Hashimoto's thyroiditis, whereas thyroid-stimulating hormone receptor antibodies (TRAb) are useful in monitoring Graves' disease and estimating the risk of relapse [2].

Thyroid ultrasonography is an important imaging tool that allows evaluation of gland size, echogenicity, vascularization, and the presence of nodules. Periodic ultrasound examinations may be particularly useful in patients with structural abnormalities or progressive disease [3,4].

In patients receiving levothyroxine therapy, serum TSH should generally be reassessed 6–8 weeks after dose adjustment and subsequently every 6–12 months once stable euthyroidism has been achieved.

A combination of biochemical, immunological, and imaging assessments enables comprehensive monitoring and supports optimal long-term management of autoimmune thyroid diseases.

Complications

Although most patients achieve satisfactory disease control with appropriate treatment, autoimmune thyroid diseases may lead to significant complications when diagnosis is delayed or therapeutic targets are not achieved. These complications may affect multiple organ systems and substantially increase morbidity and mortality [3].

Myxedema is a rare but life-threatening complication of severe hypothyroidism, typically associated with advanced Hashimoto's thyroiditis. Conversely, thyroid storm represents the most severe complication of hyperthyroidism and is most commonly observed in patients with Graves' disease. Both conditions require immediate medical intervention due to their high mortality rates [2,6].

Cardiovascular complications are common in both hypothyroidism and hyperthyroidism and may include hypertension, arrhythmias, heart failure, and an increased risk of cardiovascular events.

Long-term thyroid dysfunction may also adversely affect bone metabolism, increasing the risk of osteopenia and osteoporosis, particularly in patients with prolonged hyperthyroidism. Additionally, autoimmune thyroid diseases may contribute to infertility, menstrual irregularities, recurrent miscarriages, and adverse pregnancy outcomes [3].

Severe thyroid eye disease may lead to vision impairment and permanent ocular complications if left untreated [15].

Early diagnosis, appropriate treatment, and regular follow-up are essential for preventing complications and improving long-term patient outcomes.

Conclusions

Autoimmune thyroid diseases remain a major clinical and public health challenge due to their high prevalence, chronic nature, and frequent coexistence with other autoimmune disorders. Although current treatment strategies effectively correct hormonal abnormalities, they do not eliminate the underlying immune dysregulation responsible for disease development. Recent advances in immunology, molecular medicine, and targeted biological therapies have expanded our understanding of disease mechanisms and may pave the way toward personalized and disease-modifying treatment approaches. Future research should focus on identifying reliable biomarkers, optimizing risk stratification, and developing therapies capable of restoring immune tolerance, thereby shifting the management of autoimmune thyroid diseases from symptom control toward genuine disease modification.

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