Review Article

Impacts of Viral Infections on Autoimmune Thyroid Diseases: A Narrative Review

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Abstract

Autoimmune thyroid diseases (AITDs), primarily Hashimoto's thyroiditis (HT) and Graves' disease (GD), represent the most prevalent organ-specific autoimmune disorders worldwide. Their development results from a multifactorial interaction among genetic susceptibility, immune dysregulation, hormonal influences, and environmental triggers. Among environmental factors, viral infections have gained major attention because of their ability to disrupt immune tolerance and initiate autoreactive immune responses against thyroid antigens. This narrative review summarizes current evidence regarding the contribution of viral infections to the pathogenesis and progression of AITDs, with emphasis on immunopathogenic mechanisms, epidemiological associations, and clinical implications. A structured literature search was conducted using PubMed, Scopus, and Google Scholar databases. Priority was given to systematic reviews, meta-analyses, cohort studies, mechanistic investigations, and landmark original articles published in peer-reviewed journals. Evidence indicates that Epstein–Barr virus (EBV) and hepatitis C virus (HCV) demonstrate the strongest associations with thyroid autoimmunity, whereas SARS-CoV-2, cytomegalovirus (CMV), human herpesvirus-6 (HHV-6), enteroviruses, and parvovirus B19 show variable but biologically plausible associations. Proposed mechanisms include molecular mimicry, bystander activation, epitope spreading, aberrant HLA class II expression, polyclonal B-cell activation, apoptosis-mediated release of thyroid antigens, and chronic cytokine-driven inflammation. Viral infections appear to function primarily as environmental triggers in genetically predisposed individuals rather than independent causative agents. In Iraq and other regions with persistent infectious disease burden, understanding virus-associated thyroid autoimmunity may improve early detection and preventive strategies. Further prospective multicenter studies integrating virological, immunological, and genetic analyses are required to establish causality and guide targeted interventions.

Keyword: Autoimmune thyroid disease, Hashimoto thyroiditis, Graves' disease, Epstein–Barr virus, Hepatitis C virus, SARS-CoV-2, Thyroid autoimmunity, Viral infection.

Introduction

The thyroid gland plays a central role in metabolic regulation, neurodevelopment, cardiovascular function, and cellular homeostasis through secretion of thyroxine (T4) and triiodothyronine (T3). Thyroid hormone synthesis and release are regulated by thyroid-stimulating hormone (TSH), which serves as the most sensitive biochemical marker of thyroid function [1–3]. Autoimmune thyroid diseases (AITDs) are classical organ-specific autoimmune disorders characterized by immune-mediated destruction or stimulation of thyroid tissue. The two major forms are Hashimoto's thyroiditis (HT), which commonly causes hypothyroidism, and Graves' disease (GD), which leads to hyper-

thyroidism through stimulating autoantibodies against the TSH receptor [4,5]. AITDs are particularly common in women and typically occur between 30 and 50 years of age [6]. The pathogenesis of AITDs is multifactorial and involves interactions among genetic susceptibility, hormonal influences, environmental exposures, and immune dysregulation [7]. Environmental factors implicated in disease initiation include iodine imbalance, smoking, stress, medications, radiation exposure, and infectious agents [8]. Among these, viral infections have emerged as important contributors because of their capacity to alter innate and adaptive immune responses and disrupt self-tolerance [9]. Several viruses have been associated with thyroid autoimmunity, including Epstein–Barr virus (EBV), hepatitis C virus (HCV), SARS-CoV-2, cytomegalovirus (CMV), human herpesvirus-6 (HHV-6), enteroviruses, and parvovirus B19 [10–14]. Although causal relationships remain incompletely established, increasing molecular and immunological evidence suggests that viral infections may trigger or exacerbate thyroid autoimmunity in genetically susceptible hosts. This review summarizes current evidence regarding the role of viral infections in AITDs, discusses the principal immunopathogenic mechanisms involved, and highlights the clinical and epidemiological implications of virus-associated thyroid autoimmunity.

Materials and Methods

A structured narrative review methodology was adopted. A comprehensive literature search was conducted between January 2025 and March 2026 using the PubMed, Scopus, and Google Scholar databases. The search strategy combined the following keywords: autoimmune thyroid disease, Hashimoto thyroiditis, Graves' disease, viral infection, Epstein–Barr virus, hepatitis C virus, COVID-19, SARS-CoV-2, cytomegalovirus, human herpesvirus 6, and parvovirus B19. A total of 215 records were initially identified. After duplicate removal and screening of titles and abstracts, 86 full-text articles were assessed for eligibility. Ultimately, 42 studies that met the predefined inclusion criteria were included in this narrative review. Eligible publications comprised systematic reviews, meta-analyses, cohort studies, case–control studies, mechanistic investigations, and landmark original research articles published in English-language peer-reviewed journals. Studies focusing exclusively on non-autoimmune thyroid disorders, non-peer-reviewed publications, or articles lacking scientific relevance to virus-induced thyroid autoimmunity were excluded. Priority was given to studies providing robust mechanistic evidence, epidemiological associations, and clinically relevant findings regarding the role of viral infections in autoimmune thyroid diseases.

Immunopathogenic Mechanisms Linking Viral Infection and Thyroid Autoimmunity

The initiation of AITDs requires breakdown of immune tolerance in genetically predisposed individuals. Viral infections may contribute through several interconnected immunological pathways.

Molecular Mimicry

Molecular mimicry occurs when viral peptides share structural similarity with thyroid autoantigens such as thyroglobulin (Tg), thyroid peroxidase (TPO), or the TSH receptor. Cross-reactive immune responses generated against viral antigens may subsequently target thyroid tissue, initiating chronic autoimmune inflammation [15,16].

Bystander Activation and Epitope Spreading

Virus-induced tissue injury results in local cytokine release and activation of antigen-

presenting cells, leading to non-specific activeation of autoreactive lymphocytes. This mechanism, termed bystander activation, amplifies local inflammation and promotes autoimmune responses [17]. Persistent inflammation may subsequently induce epitope spreading, whereby the immune response extends from an initial target antigen to additional thyroid self-antigens [18].

Aberrant HLA Class II Expression

Under inflammatory conditions, thyrocytes may aberrantly express HLA-DR molecules and function as non-professional antigen-presenting cells. This abnormal antigen presentation facilitates activation of autoreactive T lymphocytes and perpetuates autoimmune thyroid injury [19].

Cytokine Dysregulation and Chronic Inflammation

Viral infections induce prolonged activation of interferon signaling pathways and proinflammatory cytokines including IL-6, IL-17, TNF-α, and CXCL10. These mediators are strongly implicated in AITD pathogenesis and contribute to Th1/Th17 polarization, recruitment of immune cells, and maintenance of chronic thyroid inflammation [20]. Collectively, these mechanisms support the concept that viruses act primarily as environmental triggers rather than direct etiological agents.

Major Viral Infections Associated with Autoimmune Thyroid Diseases

Epstein–Barr Virus (EBV)

EBV is a ubiquitous γ-herpesvirus infecting more than 90% of adults worldwide. Following primary infection, EBV establishes lifelong latency within B lymphocytes and exerts potent immunomodulatory effects [21]. EBV has been strongly implicated in autoimmune disorders because of its capacity to induce chronic B-cell activation and immune dysregulation. The viral latent membrane protein-1 (LMP1) mimics CD40 signaling pathways, promoting survival and proliferation of autoreactive B cells [22]. Additionally, EBV-encoded RNA (EBER) and LMP1 expression have been identified in thyroid tissues of patients with Hashimoto's thyroiditis and, less frequently, Graves' disease [23]. EBV reactivation may also stimulate Toll-like receptor pathways and enhance local cytokine production, thereby amplifying thyroid immune responses [24]. Current evidence therefore supports a biologically plausible relationship between EBV persistence and thyroid autoimmunity.

Hepatitis C Virus (HCV)

HCV is among the most consistently associated viral infections linked to thyroid autoimmunity. Patients with chronic HCV infection demonstrate significantly higher prevalence of anti-thyroid antibodies and hypothyroidism compared with healthy controls [25]. Mechanistically, HCV may infect thyrocytes through CD81 receptor interactions, leading to local inflammatory responses and cytokine production [26]. Molecular mimicry between HCV proteins and thyroid antigens has also been proposed [27]. Interferon-α therapy, historically used for HCV treatment, has been strongly associated with induction or exacerbation of autoimmune thyroid disease, including both HT and GD [28]. Although direct-acting antiviral therapy has reduced treatment-associated thyroid dysfunction, HCV-associated thyroid autoimmunity remains clinically relevant.

SARS-CoV-2 (COVID-19)

Since the COVID-19 pandemic, increasing evidence has linked SARS-CoV-2 infection to thyroid dysfunction and autoimmune thyroiditis. Reported manifestations include subacute thyroiditis, Graves' disease, autoimmune hypothyroidism, and non-thyroidal illness syndrome

[29]. The virus may directly infect thyroid tissue through ACE2 and TMPRSS2 receptors, while systemic hyperinflammation and cytokine storm syndromes promote immune dysregulation and autoantibody production [30]. Several cohort studies and case series have documented both new-onset and relapsing autoimmune thyroid disease following COVID-19 infection [31]. Although long-term causality remains uncertain, SARS-CoV-2 demonstrates how acute viral inflammation may transiently disrupt thyroid immune tolerance.

Cytomegalovirus (CMV) and Human Herpesvirus-6 (HHV-6)

CMV and HHV-6 are latent herpesviruses capable of persistent immune activation and modulation of cytokine responses. Both viruses may interfere with antigen presentation and alter local immune homeostasis [32]. Detection of viral DNA within thyroid tissue and serological associations with AITDs have been reported; however, findings remain inconsistent and largely observational [33]. Consequently, these viruses are currently regarded as secondary co-factors rather than primary triggers of thyroid autoimmunity.

Enteroviruses and Parvovirus B19

Enteroviruses and parvovirus B19 have also been investigated as potential environmental triggers of thyroid autoimmunity. Viral nucleic acids and antibodies have been detected in subsets of patients with Graves' disease and Hashimoto's thyroiditis [34]. Nevertheless, evidence remains heterogeneous and insufficient to establish direct causality. Their contribution likely depends on host genetic susceptibility and concurrent immune dysregulation.

Genetic Susceptibility and Host Factors

Genetic predisposition plays a central role in AITD development. Major susceptibility loci include HLA-DR polymorphisms, CTLA-4, PTPN22, CD40, and genes involved in T-cell regulation and immune tolerance [35,36]. Host-related factors including female sex, estrogen-mediated immune modulation, smoking, selenium status, iodine intake, stress, and aging further influence disease susceptibility and progression. Viral infections interact with these genetic and environmental determinants rather than acting independently. This multifactorial threshold model explains why viral exposure alone is insufficient to induce autoimmune thyroid disease in most individuals.

Clinical and Public Health Implications

Recognition of viral contributions to thyroid autoimmunity has important clinical implications. Patients with chronic viral infections, particularly HCV and EBV, may benefit from periodic thyroid function assessment and thyroid autoantibody screening, especially women and individuals with family history of autoimmune disease [37]. Early diagnosis of thyroid dysfunction may prevent complications including infertility, dyslipidemia, cardiovascular disease, neurocognitive impairment, and reduced quality of life. In Iraq and other regions with high infectious disease burden, integrating thyroid screening into infectious disease and hepatology clinics may improve early detection of virus-associated thyroid disorders. Additionally, multicenter epidemiological studies are needed to determine the true prevalence and clinical impact of viral-induced thyroid autoimmunity within Middle Eastern populations.

Future Perspectives and Research Directions

Future research should focus on large multi-center prospective cohort studies integrating molecular virology, thyroid autoantibody profiling, immunogenetics, and longitudinal

endocrine follow-up. Advanced molecular approaches such as transcriptomics, single-cell RNA sequencing, and cytokine network analysis may further clarify the mechanisms linking viral exposure with thyroid immune dysregulation. Additional studies are also needed to determine whether antiviral therapies, vaccination strategies, or immune-modulating interventions can reduce the risk of autoimmune thyroid disease in susceptible populations. Particular attention should be directed toward identifying predictive biomarkers that may allow early detection of virus-associated thyroid autoimmunity before irreversible thyroid damage occurs. In developing countries, including Iraq, establishing national autoimmune registries and strengthening collaborations between endocrinology, immunology, and infectious disease centers may significantly improve epidemiological surveillance and clinical management.

Discussion

The complex and multifactorial relationship between viral infections and autoimmune thyroid diseases (AITDs), particularly Hashimoto's thyroiditis and Graves' disease. Current evidence indicates that viral pathogens are unlikely to function as direct etiological agents; instead, they act as environmental triggers capable of initiating or amplifying autoimmune responses in genetically susceptible individuals. This concept is consistent with the contemporary model of autoimmunity, in which genetic susceptibility interacts with environmental and immunological factors to disrupt immune tolerance and promote disease development [37]. Among the investigated viral pathogens, Epstein–Barr virus (EBV) and hepatitis C virus (HCV) demonstrate the strongest epidemiological and mechanistic associations with thyroid autoimmunity. EBV contributes through latent infection of B lymphocytes, chronic immune activation, immune evasion, and molecular mimicry. In particular, latent membrane protein-1 (LMP1) mimics CD40 signaling pathways, thereby enhancing survival and proliferation of autoreactive B-cell clones [38]. Furthermore, the detection of EBV-encoded RNA (EBER) and LMP1 within thyroid tissues supports the possibility of localized immune activation in Hashimoto's thyroiditis, indicating that persistent latent viral infection may sustain chronic inflammatory responses within thyroid tissue. Similarly, HCV has been strongly associated with thyroid dysfunction through both direct and indirect immunopathogenic mechanisms. HCV may interact with thyrocytes through CD81 receptor-mediated pathways, resulting in local cytokine production, recruitment of inflammatory immune cells, and cytokine-mediated immune dysregulation [39]. Molecular homology between HCV proteins and thyroid antigens may additionally contribute to loss of self-tolerance. Moreover, the increased incidence of autoimmune thyroid dysfunction observed during interferon-α therapy further supports the role of immune-mediated virus–thyroid interactions rather than isolated drug toxicity alone [40,41]. The emergence of SARS-CoV-2 has provided additional evidence supporting the role of viral inflammation in thyroid autoimmunity. Clinical observations of subacute thyroiditis, Graves' disease, and autoimmune hypothyroidism following COVID-19 infection suggest that acute systemic inflammation and cytokine storm syndromes may transiently disrupt immune tolerance [42]. In addition, thyroid expression of ACE2 and TMPRSS2 receptors may facilitate direct viral interaction with thyroid tissue. Although current evidence supports a biological association, long-term prospective studies remain necessary to determine whether COVID-19 significantly

increases the lifetime risk of chronic autoimmune thyroid disease. Other viral agents, including cytomegalovirus (CMV), human herpesvirus-6 (HHV-6), enteroviruses, and parvovirus B19, demonstrate weaker and less consistent associations with AITDs. These viruses may contribute indirectly through immune modulation, persistent cytokine activation, and alteration of antigen presentation pathways [43]. However, available evidence remains insufficient to establish definitive causality, suggesting that their pathogenic contribution is likely dependent on host susceptibility, immune status, and coexisting environmental factors. Despite the diversity of implicated viral pathogens, several common immunological mechanisms appear to converge in the pathogenesis of virus-associated thyroid autoimmunity. These include activation of Toll-like receptors, Th1/Th17 immune polarization, regulatory T-cell dysfunction, aberrant HLA class II expression, and persistent production of inflammatory cytokines such as IL-6, IL-17, TNF-α, and CXCL10 [44]. The convergence of these pathways may explain why distinct viral infections can produce similar autoimmune thyroid phenotypes. Importantly, viral exposure alone appears insufficient to induce autoimmune thyroid disease. Genetic susceptibility factors, particularly HLA class II alleles, CTLA-4 polymorphisms, and PTPN22 variants, together with hormonal and environmental influences, remain essential determinants of disease expression [45]. These findings support a threshold-based multifactorial model in which disease develops only when cumulative genetic and environmental risk exceeds immune tolerance capacity. In the Iraqi context, these findings are particularly relevant due to the coexistence of chronic viral infections, environmental stressors, and variable healthcare accessibility. Widespread EBV seropositivity, persistent HCV burden in certain populations, smoking prevalence, iodine variability, and psychosocial stress may collectively increase autoimmune susceptibility. However, the absence of national autoimmune registries and limited molecular epidemiological studies restrict accurate assessment of virus-associated thyroid disease burden within Iraq. Therefore, multicenter Iraqi studies integrating virological, endocrinological, and immunogenetic methodologies are urgently needed.

Limitations of study

Despite growing evidence supporting viral involvement in autoimmune thyroid disease, several limitations remain. First, many implicated viruses, particularly EBV, are highly prevalent in the general population, making direct causal associations difficult to establish. Second, substantial heterogeneity exists among studies regarding diagnostic criteria, study design, and patient selection. Third, most available data derive from observational studies, case reports, or small cohorts, limiting generalizability. Furthermore, the chronic and slowly progressive nature of autoimmune thyroid diseases complicates identification of the initial triggering events. Limited availability of longitudinal virological and immunological data also restricts mechanistic interpretation.

Conclusions

Autoimmune thyroid diseases arise from complex interactions between genetic susceptibility, immune dysregulation, and environmental triggers; viral infections contribute to disease development through mechanisms such as molecular mimicry, bystander activation, and cytokine-mediated immune disturbance; among studied viruses, Epstein–Barr virus and hepatitis C virus show the strongest evidence of association with

thyroid autoimmunity, while SARS-CoV-2 may have a transient effect on thyroid immune regulation in susceptible individuals; however, viral exposure alone is insufficient for disease development, emphasizing the essential role of host genetic and environmental factors; therefore, further multicenter prospective studies are required to clarify causality and improve early detection and prevention strategies.

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