E CONF SERIES

International Conference on Medical Science, Medicine and Public Health
Hosted online from Jakarta, Indonesia
Website: econfseries.com 30th July, 2025

CLINICAL COURSE OF THYROIDITIS AND MODERN TREATMENT METHODS

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Annotation

This thesis explores the clinical features, diagnosis, and modern treatment of thyroiditis, an inflammatory disorder of the thyroid gland. It analyzes the diverse presentations of autoimmune, infectious, and drug-induced thyroiditis, highlighting their distinct mechanisms and clinical courses. The study shows that early recognition, supported by laboratory and imaging tests, allows for targeted therapies that improve outcomes. Current treatments include immunosuppressive drugs, hormone replacement, and biologics targeting specific inflammatory pathways. The research emphasizes standardized diagnostic and therapeutic protocols to preserve thyroid function and guide clinical decisions.

Keywords: thyroiditis, autoimmune thyroid disease, Hashimoto thyroiditis, subacute thyroiditis, postpartum thyroiditis, thyroid function disorders, immunosuppressive therapy, thyroid hormone replacement, inflammatory thyroid conditions, thyroid ultrasound

Today, thyroiditis is a clinically complex endocrine disorder that includes diverse inflammatory conditions impairing thyroid structure and function. Autoimmune thyroiditis alone affects about four percent of the global population, with a higher prevalence in women. Its pathogenesis involves genetic, environmental, and immune factors that trigger thyroid inflammation and dysfunction. Beyond thyroid-related symptoms, it is often linked to other autoimmune diseases and can affect reproduction, cardiovascular health, and metabolism. Modern diagnostics - such as ultrasonography, autoantibody testing, and advanced thyroid function assessment - enable precise subtype identification and treatment monitoring. Therapies have advanced as well, with targeted immunomodulators, improved hormone

replacement, and personalized strategies based on patient profiles and disease severity.

The clinical course of thyroiditis is highly heterogeneous, with distinct subtypes showing characteristic presentations, progression, and outcomes. Hashimoto thyroiditis, the most common autoimmune form, develops gradually with hypothyroidism, thyroid enlargement, and thyroid peroxidase and thyroglobulin antibodies. Patients often report fatigue, weight gain, cold intolerance, and cognitive decline over months to years. This results from lymphocytic infiltration, follicular destruction, and fibrosis impairing hormone production. Subacute thyroiditis presents acutely with neck pain, fever, and thyrotoxic symptoms from sudden hormone release, often after a viral respiratory infection. It typically follows a triphasic course-thyrotoxic, hypothyroid, and recovery-over several months. Examination reveals a very tender enlarged thyroid, elevated erythrocyte sedimentation rate, and low thyroid stimulating hormone in the acute phase. Postpartum thyroiditis affects about ten percent of women in the first year after delivery, showing a biphasic course of thyrotoxicosis followed by hypothyroidism. It results from immune rebound after pregnancy-induced suppression, with thyroid peroxidase antibodies as predictive markers. Diagnosis is challenging due to overlap with normal postpartum changes. Diagnosis combines clinical evaluation, laboratory tests, and imaging. Thyroid stimulating hormone, free thyroxine, and free triiodothyronine tests determine functional status and disease phase. Autoantibodies distinguish autoimmune from non-autoimmune forms, while ultrasonography shows structural and vascular changes to support diagnosis and guide therapy. Treatment is individualized. Hashimoto thyroiditis is managed with levothyroxine, adjusted to normalize thyroid stimulating hormone and relieve symptoms. Selenium may help in mild cases with elevated antibodies. Subacute thyroiditis responds to anti-inflammatory drugs in mild cases and corticosteroids like prednisone in severe ones, which quickly reduce symptoms and inflammation; beta-blockers help manage thyrotoxic symptoms. New therapies for refractory cases include immunomodulatory agents such as rituximab for severe autoimmune variants and tocilizumab for resistant subacute forms. These biologics show promise in early studies but require further research to establish standard protocols.

Thyroiditis requires early recognition, precise diagnosis, and tailored treatment to address its diverse forms. Modern therapies have improved outcomes, while future research should focus on biomarkers and personalized care. Multidisciplinary collaboration remains key to reducing complications and enhancing patient well-being.

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