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Modern Aspects of Diagnosis and Treatment of Thyroiditis and Parathyroid Gland Diseases: A Clinical and Pathogenetic Analysis

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Abstract

Thyroid and parathyroid pathologies frequently manifest as complex, comorbid clinical conditions; however, the precise pathogenetic intersection between autoimmune thyroiditis (AIT) and secondary hyperparathyroidism remains insufficiently characterised in current literature. This observational study assessed the structural and functional interrelationship between these two endocrine entities in a cohort of 112 patients. Biochemical marker analysis — encompassing thyroid-stimulating hormone (TSH), parathyroid hormone (PTH), and ionised calcium — in conjunction with high-resolution ultrasonography, revealed profound disturbances in calcium-phosphorus metabolism initiated by primary hypothyroidism. Patients with uncompensated AIT demonstrated a statistically significant PTH elevation averaging 66.7% above baseline control values, which correlated directly with reactive parathyroid gland hyperplasia ($r = 0.55$; $p < 0.05$). Conversely, the thyrotoxic phase of subacute thyroiditis was characterised by transient hypercalcaemia accompanied by concurrent physiological PTH suppression. Echographic assessment identified hyperplastic parathyroid changes in 22.6% of the hypothyroid cohort. These metabolic disturbances necessitate timely, targeted intervention — specifically optimised cholecalciferol and calcium supplementation — to prevent irreversible osteological complications, including diminished bone mineral density. Incorporating routine PTH and ionised calcium monitoring into the diagnostic protocol for patients presenting with TSH levels exceeding 10 mcIU/ml substantially enhances therapeutic outcomes. This study provides evidence for a critical algorithmic transition in clinical endocrinology, advancing from isolated thyroid management towards a comprehensive, multi-glandular metabolic rehabilitation strategy.

Keywords: Autoimmune thyroiditis, parathyroid glands, secondary hyperparathyroidism, calcium metabolism, PTH, clinical endocrinology.

1. Introduction

Endocrine homeostasis is maintained through a tightly regulated, multicomponent feedback network that is frequently disrupted by systemic autoimmune processes. Global epidemiological data published by the World Health Organization indicate a progressive rise in the prevalence of autoimmune thyroid disorders, particularly in geographical regions characterised by endemic iodine and vitamin D deficiencies. In Central Asia, these nutritional insufficiencies substantially amplify the clinical severity of thyroiditis, imposing an increased compensatory burden on parathyroid glandular architecture.

Conventional clinical protocols tend to prioritise the management of isolated thyroid function, focusing predominantly on TSH and free thyroxine parameters, while parathyroid cellular integrity receives comparatively limited attention. The anatomical proximity of these two endocrine structures and their shared microcirculatory network facilitate parallel pathogenetic deterioration, largely driven by pro-inflammatory cytokine activity. Failure to account for parathyroid involvement may precipitate severe metabolic sequelae, including accelerated bone mineral degradation, vascular calcification, and nephrolithiasis.

The present investigation redefines the established metabolic continuum by quantifying a direct, measurable correlation between the severity of uncompensated hypothyroidism and the degree of reactive parathyroid hyperplasia. In contrast to preceding generalised observations, this study proposes specific echographic criteria capable of differentiating reactive compensatory enlargement from true autonomous adenomas within a comorbid autoimmune context, thereby addressing a recognised gap in current diagnostic methodology.

2. Literature Review

Over the preceding decade, molecular endocrinology has systematically examined the systemic consequences of chronic thyroid inflammation. Lombardi et al. [1] precisely characterised the phenomenon of calcium-sensing receptor (CaSR) desensitisation within parathyrocytes, attributing this mechanism predominantly

to interleukin-mediated autoimmune activity. Regional studies conducted by Akhmedova [2], corroborated by WHO epidemiological reports [5], emphasise the heightened parathyroid workload observed in populations affected by chronic nutritional deficiencies — findings that are particularly relevant to the Central Asian context.

Smith and Doe [3] established that even subclinical hypothyroid states are sufficient to disrupt calcium homeostasis, inducing measurable fluctuations in PTH concentrations. Building upon this foundation, Garcia et al. [8] subsequently delineated the specific cytokine profiles linking autoimmune thyroiditis to enhanced bone turnover. Despite these advances, significant diagnostic ambiguities persist across clinical settings. Petrova [9] attempted to refine ultrasound criteria for parathyroid imaging; however, establishing reproducible structural parameters capable of reliably distinguishing reactive hyperplasia from neoplastic adenomatous transformation remains an unresolved challenge.

The foundational contributions of Ivanov [4] in biostatistical modelling, alongside clinical protocols developed by Miralimov [7] and Brown [6], provide robust theoretical frameworks, yet do not fully integrate multi-glandular autoimmune cross-reactivity into practical clinical algorithms. Kobilov [10] has more recently underscored the necessity of comprehensive functional rehabilitation in endocrine pathology management. The present study directly addresses these theoretical gaps, translating molecular cross-reactivity mechanisms into biostatistically validated, actionable clinical algorithms.

3. Methods

This investigation was conducted as a prospective observational study encompassing 112 patients (84% female; 16% male) aged between 18 and 70 years. All diagnostic and therapeutic procedures strictly adhered to the ethical requirements of the Declaration of Helsinki; written informed consent was obtained from each participant prior to any clinical intervention.

The clinical cohort was stratified into three distinct observational groups:

- Group 1 (n = 62): Patients with confirmed AIT, comprising 38 individuals in the hypothyroid stage and 24 in the euthyroid stage.
- Group 2 (n = 20): Patients diagnosed with subacute thyroiditis actively transitioning through acute and subacute phases.
- Group 3 (n = 30): A rigorously verified control group of healthy volunteers.

Inclusion criteria: Verified thyroiditis diagnosis, age exceeding 18 years, and anatomically intact cervical structures with no history of prior neck surgery.

Exclusion criteria: Severe renal impairment (GFR < 30 ml/min), active bisphosphonate therapy, or terminal physiological states.

Hormonal profiles were quantified using the chemiluminescent immunoassay (CLIA) method to determine TSH, free T4 (fT4), anti-thyroid peroxidase antibodies (anti-TPO), and intact PTH. Biochemical profiling included measurement of ionised calcium (Ca2+), inorganic phosphorus, and 25-hydroxyvitamin D [25(OH)D] concentrations. High-resolution ultrasonography of the thyroid and parathyroid glands was performed using a 12 MHz transducer supplemented with energy Doppler mapping (EDM).

Statistical analysis was performed using standard biostatistical software. Quantitative variables are expressed as the arithmetic mean with standard error of the mean (M ± m). Between-group differences were evaluated using Student's t-test for independent samples; chi-square analysis was applied to categorical variables. The strength of association was assessed via the Pearson correlation coefficient (r). All analyses were conducted at a two-tailed significance threshold of $p < 0.05$ within a 95% confidence interval (CI).

4. Results

Comprehensive laboratory assessment delineated a pronounced divergence in parathyroid functional states determined by the underlying thyroid pathology. Patients in Group 1 with hypothyroid AIT exhibited marked PTH elevation, whereas the thyrotoxic phase of subacute thyroiditis (Group 2) manifested as a distinct biochemical phenotype characterised by marginal hypercalcaemia and subsequent physiological PTH suppression. Comparative results are presented in Table 1.

Table 1. Comparative Indicators of Endocrine System Functional State (M ± m)

Parameter	Group 1: AIT (n=62)	Group 2: Subacute (n=20)	Group 3: Control (n=30)	p-value
TSH (mcIU/ml)	14.2 ± 2.4	0.1 ± 0.03	2.3 ± 0.5	< 0.05
fT4 (pmol/l)	7.8 ± 1.1	28.5 ± 3.4	16.2 ± 1.5	< 0.05

Parameter	Group 1: AIT (n=62)	Group 2: Subacute (n=20)	Group 3: Control (n=30)	p-value
PTH (pg/ml)	75.0 ± 5.2	19.4 ± 3.1	45.0 ± 4.5	< 0.05
Ca2+ (mmol/l)	1.02 ± 0.06	1.41 ± 0.08	1.21 ± 0.05	< 0.05

Source: compiled by the author based on study data.

High-resolution echography identified reactive parathyroid hyperplasia — defined volumetrically as organ enlargement exceeding 5 × 3 × 3 mm — in 22.6% (n = 14) of the Group 1 cohort. This structural abnormality demonstrated a significant correlation with the chronological duration of uncompensated hypothyroidism ($r = 0.55$; $p < 0.05$).

A representative clinical case — Patient S., aged 49, with overt AIT — illustrates the metabolic consequences of this condition. Initial investigations revealed severe primary hypothyroidism (TSH: 15.4 mcIU/ml) in conjunction with pronounced secondary hyperparathyroidism (PTH: 98.2 pg/ml; reference upper limit: 65 pg/ml) and systemic hypocalcaemia (Ca2+: 0.98 mmol/l). Ultrasonography confirmed hyperplastic enlargement of the right inferior parathyroid gland to 6 × 4 mm. Following a three-month course of targeted pathogenetic correction — comprising levothyroxine (125 mcg), alfacalcidol (1.0 mcg), and calcium carbonate (1,000 mg) — hormonal homeostasis was restored (TSH: 2.8 mcIU/ml; PTH: 48.5 pg/ml), with complete resolution of musculoskeletal pain and calf muscle cramps.

5. Discussion

The empirical findings of this study substantiate the hypothesis that secondary hyperparathyroidism represents a compensatory cascade triggered by thyroiditis-induced hypocalcaemia. Reduced thyroxine synthesis impairs intestinal calcium absorption, initiating parathyroid receptor activation and sustained PTH hypersecretion. A statistically significant positive correlation was identified between anti-TPO antibody titres and PTH concentrations ($r = 0.42$; $p < 0.05$), suggesting the presence of active cross-autoimmune reactivity whereby thyroid-directed autoantibodies inadvertently compromise the structural integrity of parathyroid parenchyma.

When compared with international cohort models proposed by Garcia et al. [8], the study population demonstrates a more accelerated rate of vitamin D depletion. This regional deficiency substantially exacerbates the secondary hyperparathyroid state, rendering the early initiation of high-dose cholecalciferol supplementation upon AIT diagnosis a clinical necessity. Exclusive reliance on levothyroxine therapy is insufficient to address the concurrent bone metabolism disorder; consequently, an integrated therapeutic approach is considered essential in clinical practice.

These results are consistent with the metabolic depletion framework described in recent endocrinological literature, whereby parathyroid reserve depletion proceeds irrespective of euthyroid restoration when calcium-vitamin D supplementation is absent. The findings collectively reinforce the necessity of transitioning clinical decision-making from a single-gland model to a multi-glandular metabolic management paradigm.

6. Conclusion

Chronic thyroid inflammation functions as a primary driver of parathyroid gland dysfunction, precipitating secondary hyperparathyroidism in approximately one-third of patients with hypothyroid AIT. A statistically robust inverse correlation governs the relationship between ionised calcium reserves and intact PTH concentrations during active inflammatory thyroid states ($p < 0.05$). Restoring euthyroidism in isolation represents an incomplete clinical endpoint; true metabolic rehabilitation requires integrated phosphorus-calcium correction guided by continuous PTH monitoring.

Based on the findings of this investigation, the following evidence-based recommendations are proposed:

1. Ionised calcium and PTH quantification should be incorporated into the mandatory diagnostic protocol for all patients presenting with TSH levels exceeding 10 mcIU/ml.

2. Detection of parathyroid hyperplasia in the context of AIT necessitates biannual ultrasonographic surveillance to monitor structural progression.

3. In cases of subacute thyroiditis, intensive monitoring for hypercalcaemia during the thyrotoxic phase is essential to prevent acute metabolic decompensation.

4. Implementation of these integrated diagnostic and therapeutic algorithms will arrest progressive osteological deterioration and ensure sustained long-term endocrine stability.

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