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THYROID DYSFUNCTION AND QUALITY OF LIFE IN PATIENTS ON LITHIUM THERAPY: A NARRATIVE REVIEW

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Abstract

Introduction. Lithium remains the gold standard in the treatment of bipolar disorder (BD); however, its narrow therapeutic index and effects on the thyroid gland pose a significant therapeutic challenge and require systematic monitoring for adverse effects.

Aim of the study. To analyze the mechanisms of thyroid dysfunction induced by long-term lithium therapy and evaluate patient monitoring strategies to optimize treatment safety and quality of life (QoL).

Material and methods. A structured narrative review of the medical literature published between 2021 and 2026.

Results. Lithium inhibits iodine uptake and thyroid hormone release, most commonly causing hypothyroidism. Symptoms such as fatigue, weight changes, and cognitive impairment significantly affect daily functioning and may overlap with depressive episodes in BD, substantially reducing QoL.

Conclusions. Appropriate monitoring, diagnostic evaluation, and timely intervention are essential for minimizing complications. Patient education and interdisciplinary collaboration

between psychiatrists and endocrinologists are key to maintaining QoL during lithium therapy, underscoring the need to integrate thyroid monitoring into routine psychiatric and endocrinological care.

Keywords: lithium therapy; thyroid dysfunction; hypothyroidism; drug-induced thyroid dysfunction; quality of life; patient adherence

1. **Introduction**

Lithium is one of the most widely used mood stabilizers in the treatment of psychiatric disorders, particularly bipolar disorder (BD), as demonstrated by systematic reviews and meta-analyses of randomized controlled trials [1]. It remains a cornerstone pharmacological agent in the prevention of manic and depressive recurrences and significantly reduces the risk of suicide [2,3].

Bipolar disorder, in the management of which lithium plays a pivotal role, is among the most severe psychiatric conditions, with an estimated lifetime prevalence of 4.4% of the general population. The life expectancy of individuals with BD is substantially reduced compared to the general population, and the risk of suicide is approximately 11-fold higher in this group [2]. Disease recurrences frequently necessitate psychiatric hospitalization, underscoring the need for long-term pharmacotherapy [2].

Despite its well-established clinical efficacy, lithium is associated with a broad spectrum of adverse effects, including clinically significant endocrine complications. Large-scale cohort studies confirm that these disturbances represent frequent and clinically relevant complications of lithium therapy encountered in routine clinical practice [4].

Among the most common and clinically significant adverse effects of lithium therapy are thyroid dysfunctions [5]. Hypothyroidism is observed in a substantial proportion of patients, along with goiter development and, less frequently, hyperthyroidism [6]. In the majority of cases, these disturbances are mild and stable in nature [4]. Lithium interferes with iodine metabolism in thyroid follicular cells, affecting its uptake and incorporation into thyroid hormones, thereby impairing their synthesis and secretion [7].

Notably, patients with bipolar disorder (BD) exhibit a higher prevalence of primary thyroid dysfunction and elevated antithyroid antibody titers compared to the general population. These phenomena may be closely related to hypothalamic-pituitary-thyroid (HPT) axis dysregulation

and neuroendocrine disturbances underlying the pathophysiology of BD, suggesting a potential link between immunological alterations and the pathogenesis of affective disorders [8–10].

A comprehensive understanding of these associations is essential for optimizing interdisciplinary patient care, as hormonal disturbances may exacerbate neuropsychiatric symptoms and adversely affect patients' physical and mental well-being. Symptoms such as fatigue, weight fluctuations, and cognitive impairment directly reduce quality of life (QoL), and their resemblance to depressive symptomatology—including depressed mood, impaired concentration, and psychomotor retardation—significantly complicates differential diagnosis.

Therefore, an in-depth analysis of the mechanisms and clinical implications of lithium-induced thyroid dysfunction is indispensable for ensuring appropriate and safe therapeutic management [10,11]. Early detection, regular monitoring, and timely intervention may reduce the risk of complications and improve clinical outcomes.

This study aims to review the mechanisms underlying lithium-induced thyroid dysfunction and analyze their impact on patients' daily functioning and quality of life, as well as to identify the measures necessary to ensure effective and well-tolerated lithium therapy, particularly in patients with BD. Appropriate diagnostic evaluation and therapeutic management require close interdisciplinary collaboration between specialists from different medical fields, particularly endocrinologists and psychiatrists.

2. Methodology

This study is a structured narrative review based on a comprehensive analysis of the available scientific literature on the effects of lithium therapy on thyroid function. A structured literature search was conducted in the following databases: PubMed, Google Scholar, and Scopus. The search strategy included the following keywords: "lithium therapy," "thyroid dysfunction," "hypothyroidism," "drug-induced thyroid dysfunction," "quality of life," and "patient adherence." Both review articles and original research papers published in English were eligible for inclusion. The search was primarily focused on publications from 2021 to 2026 to ensure the currency and relevance of the data.

Selected articles were analyzed with regard to the mechanisms of lithium-induced thyroid dysfunction, the clinical presentation of thyroid disorders, and their impact on patient functioning. Data were synthesized by integrating findings from molecular endocrinology with

clinical aspects of quality of life, allowing for the formulation of evidence-based conclusions regarding patient care during lithium therapy. Inclusion criteria encompassed studies involving adult patients receiving lithium for affective disorders, describing the effects of therapy on thyroid function, as well as monitoring and management strategies. Both narrative and systematic reviews, as well as original observational, cohort, and retrospective studies, were considered eligible. The methodological quality of the included publications was assessed based on source credibility, data recency, and consistency of findings with the existing body of literature. The literature selection process involved independent screening of titles and abstracts by the authors, followed by full-text evaluation of eligible studies. In cases of discrepancies in the assessment of individual studies, consensus was reached through discussion. The review was conducted in accordance with structured narrative review methodology and does not follow PRISMA guidelines.

3. Results

3.1. Molecular mechanisms of lithium-induced thyroid dysfunction

Lithium therapy exerts a significant effect on thyroid function, involving both molecular mechanisms and clinical manifestations of thyroid disorders. The toxic effects of lithium on the thyroid gland begin at the level of transmembrane transport in thyrocytes. Due to its physicochemical properties similar to those of sodium ions, lithium enters thyroid follicular cells via the same transport pathways used by iodine. A key role in this process is played by the sodium–iodide symporter (NIS) and the pendrin protein [7]. Once inside the cell, lithium reduces iodine uptake by interfering with iodide transport. Furthermore, it inhibits the coupling of iodotyrosines and impairs the secretion of thyroid hormones—thyroxine (T4) and triiodothyronine (T3). Lithium also affects thyroglobulin structure and impairs colloid formation and resorption, thereby disrupting thyroid hormone synthesis [7]. The complexity of these intracellular disturbances explains why lithium therapy leads to depletion of thyroid hormone stores, ultimately resulting in the development of hypothyroidism and goiter.

A further pathophysiological aspect involves lithium's effects on the hypothalamic–pituitary–thyroid (HPT) axis. Lithium reduces pituitary sensitivity to thyroid hormones, leading to a compensatory increase in TSH secretion. It also increases the risk of autoimmune thyroid processes by stimulating B lymphocytes to produce anti-thyroid peroxidase antibodies (TPOAb) and anti-thyroglobulin antibodies (TgAb) [8]. This may explain why patients with a

history of autoimmune disorders are at substantially higher risk of developing hypothyroidism during lithium therapy. Furthermore, lithium indirectly reduces serum free triiodothyronine (FT3) metabolism by decreasing the activity of type 1 and type 2 5'-deiodinases [12].

3.2. Clinical manifestations and monitoring

Lithium therapy may be associated with a range of thyroid dysfunctions, including hypothyroidism, hyperthyroidism, and thyroiditis. Hypothyroidism, occurring in approximately 8–20% of patients, is more frequently observed in women and in individuals with pre-existing thyroid autoimmunity. Goiter, which often coexists with hypothyroidism, represents one of the most common morphological changes of the thyroid gland and occurs approximately four times more frequently in lithium-treated individuals compared to the general population. Graves' disease and other forms of hyperthyroidism are rare but remain clinically significant [12].

The most common clinical manifestations of lithium-induced hypothyroidism include fatigue, weight gain, cold intolerance, constipation, dry skin, psychomotor retardation, and cognitive impairment, all of which may significantly affect daily functioning. Current evidence emphasizes the need for regular thyroid function monitoring in lithium-treated patients, including measurement of serum TSH and thyroid hormone levels (e.g., FT4). Routine clinical assessment—comprising a detailed medical history and physical examination—constitutes an essential complement to laboratory testing in the comprehensive care of individuals receiving lithium therapy. This approach enables early detection of thyroid dysfunction and identification of those requiring further diagnostic evaluation [6].

4. Discussion

4.1. Efficacy and endocrine adverse effects of lithium therapy

Lithium remains one of the most efficacious and widely used mood stabilizers in the treatment of bipolar disorder, with well-established evidence demonstrating its effectiveness in reducing the risk of relapse, suicidal behavior, and psychiatric hospitalization [3,11]. Contemporary meta-analyses confirm that, although lithium therapy is associated with an increased risk of hypothyroidism, its antisuicidal properties and efficacy in relapse prevention confer a significant therapeutic advantage over alternative treatment modalities [1,13]. Despite its substantial clinical benefits, lithium therapy carries a relevant risk of endocrine adverse effects, with the thyroid gland being the most frequently affected organ.

Lithium exerts its effects at multiple molecular and cellular levels, disrupting iodine homeostasis and thyroid hormone synthesis in follicular cells via sodium–iodide symporter (Na^+/I^-)-dependent transport. It accumulates in the thyroid gland at concentrations 3–4 times higher than those found in plasma. One of the key mechanisms involves inhibition of iodine uptake, which reduces iodine availability for incorporation into thyroglobulin, thereby decreasing T3 and T4 production [7,12]. Evidence suggests that approximately 20–30% of individuals undergoing long-term lithium therapy may require thyroid hormone replacement therapy due to the development of clinically significant hypothyroidism, with this risk being particularly elevated in women and in those with elevated baseline anti-thyroid antibody titers [12].

Evidence demonstrates that lithium therapy is associated with a spectrum of thyroid dysfunctions, including goiter (approximately 15%), hypothyroidism (8–30%), and, less frequently, hyperthyroidism (approximately 5%), encompassing Graves' disease and lithium-induced thyroiditis [12]. These findings highlight the importance of incorporating endocrine risk assessment at the initiation of lithium therapy and ensuring ongoing monitoring throughout its entire duration.

4.2. Diagnostic challenges

Evidence indicates that symptoms of hypothyroidism—such as fatigue, weight gain, psychomotor retardation, and cognitive impairment—overlap with the symptomatology of depressive episodes in the course of bipolar disorder, thereby complicating differential diagnosis and potentially leading to inappropriate therapeutic decisions [21,23]. This may result in misinterpretation of the clinical picture as inadequate mood stabilization with lithium, whereas the underlying cause is thyroid hormone deficiency.

Furthermore, certain manifestations of hypothyroidism—such as weight gain, muscle weakness, and psychomotor retardation—may be erroneously attributed to direct adverse effects of lithium or to coexisting metabolic disorders, further complicating the diagnostic process and potentially delaying appropriate treatment [12].

Moreover, current evidence highlights that the clinical presentation in patients with bipolar disorder is influenced by multiple factors, including the duration of depressive episodes, the phase of illness, and the pharmacological regimen employed, thereby precluding a definitive assessment of lithium's independent contribution to endocrine disturbances [8,10].

Consequently, thyroid dysfunction has a significant impact on patients' quality of life and the course of the underlying psychiatric condition, underscoring the importance of early recognition and systematic monitoring [6,22].

4.3. Monitoring protocols and management

During long-term lithium therapy, periodic assessment of thyroid function is recommended, including measurement of serum TSH and FT4 at baseline and subsequently at intervals of 6–12 months [12]. Recent evidence suggests that increased vigilance is warranted during the first year of treatment, with thyroid function testing recommended every three months, as disturbances most commonly manifest as a significant rise in TSH and a decline in T3 and T4 levels between the third and sixth month of therapy [6]. Real-world clinical data indicate that the 6-month monitoring interval recommended by NICE guidelines is frequently applied in practice, although adherence remains inconsistent [14]. Serum TSH measurement remains the gold standard for thyroid screening, and in equivocal cases, the diagnostic workup should be extended to include FT4, FT3, anti-TSH receptor antibodies (TRAb), and thyroid ultrasonography [12].

These discrepancies may reflect the need for increased clinical vigilance during the early phase of therapy, when the risk of thyroid disturbances is greatest, while allowing for less frequent monitoring during the later course of treatment. Concurrently, serum lithium levels should be assessed every 3–6 months to maintain concentrations within the therapeutic range of 0.6–1.0 mEq/L [11].

In most cases, hypothyroidism does not require discontinuation of lithium and can be effectively managed with thyroid hormone replacement therapy, allowing continuation of mood stabilizer treatment [5,6]. Long-term studies indicate that thyroid dysfunction in lithium-treated patients is often mild or transient and does not always require immediate therapeutic intervention [4]. Some abnormalities, particularly subclinical hypothyroidism, may resolve spontaneously, supporting a cautious approach with a period of observation before initiating replacement therapy [7].

In cases of hyperthyroidism, particularly when detected during a mood episode or in the presence of more severe disturbances, individualized clinical assessment and close collaboration between psychiatrists and endocrinologists may be warranted [12]. Evidence indicates that thyroid dysfunction develops within the first three years of lithium therapy in

approximately 90% of affected patients. However, these findings also suggest that thyroid disturbances may emerge at later stages of treatment, highlighting the importance of long-term and systematic monitoring of thyroid function [14].

Thyroid function should be reassessed two months after lithium discontinuation. The decision regarding continuation of thyroid hormone replacement therapy depends on serum TSH levels (reference range: 0.4–4.0 mIU/L), with values persistently in the upper normal range constituting an indication for continued treatment [7,15].

Thyroid dysfunction during lithium therapy represents a clinically significant challenge that may affect both treatment efficacy and patients' quality of life. An integrated approach—comprising regular laboratory monitoring, clinical assessment, individualized therapy, and close collaboration between psychiatrists and endocrinologists—enables effective lithium treatment while minimizing endocrine complications and improving long-term quality of life in patients with bipolar disorder [5].

5. Conclusions

5.1. Clinical implications

Lithium remains one of the most effective treatment modalities for bipolar disorder (BD); however, its use is associated with a relevant risk of thyroid dysfunction [2,3,11]. These disturbances arise from lithium's effects on iodine metabolism, thyroid hormone synthesis, and hormone secretion. Symptoms of hypothyroidism may mimic depressive episodes in BD, thereby complicating differential diagnosis and potentially influencing therapeutic decisions and the assessment of treatment efficacy. Regular monitoring of thyroid function and serum lithium levels is therefore essential, enabling early detection of abnormalities and timely clinical intervention [20].

Particular attention should be paid to the initial phase of therapy, encompassing the first 12 months, during which the most dynamic changes are observed. During this period, TSH monitoring at three-month intervals is recommended to facilitate early detection of disturbances and reduce the risk of progression [6]. This approach may also contribute to the prevention of thyroid dysfunction and its potential impact on cognitive functioning.

Correction of thyroid dysfunction through replacement therapy is essential for minimizing affective symptoms in lithium-treated patients. From a systemic perspective, hormonal

stabilization not only improves quality of life (QoL) but, more importantly, enhances treatment adherence, which is a key determinant of therapeutic efficacy in bipolar disorder. Inadequate monitoring may result in overlapping clinical presentations, in which somatic symptoms—such as fatigue and psychomotor retardation—are misinterpreted as lithium treatment failure, potentially worsening the overall prognosis [6,7].

Effective therapeutic management requires an interdisciplinary model based on close collaboration between psychiatrists, endocrinologists, and patients. Such a coordinated approach enables optimization of clinical outcomes and reduction of iatrogenic complications. Consideration should also be given to the potential for spontaneous normalization of thyroid function. In many patients with subclinical hypothyroidism, thyroid function may return to euthyroidism, suggesting that a watchful waiting strategy without immediate intervention may represent an appropriate management approach [5].

Patient education constitutes an integral component of effective care in lithium-treated individuals. Patients should be informed about the potential occurrence of hypothyroid symptoms, their resemblance to depressive symptoms, and the importance of reporting new complaints to their physician. Patient awareness of the mechanisms underlying lithium's effects on the thyroid gland, as well as the importance of regular follow-up assessments, enhances treatment adherence and facilitates early detection of thyroid dysfunction [11].

Of particular importance is education regarding the necessity of regular follow-up visits, even in the absence of symptoms, as subclinical hypothyroidism may remain asymptomatic for extended periods, gradually impairing cognitive and emotional functioning [6,23].

Current evidence highlights the need for the development of consistent and clinically feasible monitoring protocols for use in routine clinical practice [14,16]. Existing discrepancies between current recommendations and their implementation underscore the necessity of standardizing diagnostic procedures [14,17].

5.2. Future research directions

Future research should focus on long-term analyses of lithium's effects on thyroid function across diverse patient populations, as well as on the identification of risk factors for endocrine disturbances. Such efforts may enable more precise patient stratification and individualized therapy, allowing identification of subgroups most likely to benefit from treatment while

maintaining an optimal safety profile. Of particular importance is the extension of follow-up periods beyond 12 months to capture long-term changes in thyroid parameters and treatment outcomes. Current evidence indicates that regular monitoring of thyroid function enhances the safety of lithium therapy and facilitates early detection of disturbances [6].

Future studies should also incorporate the patient perspective, including assessment of the impact of thyroid dysfunction on subjective quality of life, as well as social and occupational functioning and treatment satisfaction, thereby providing a more comprehensive understanding of the real-world burden of disease in lithium-treated patients with bipolar disorder [20].

Growing attention is being directed toward the application of digital tools and mHealth technologies based on data derived from mobile devices, which—when integrated with laboratory data—may support early detection of subtle disturbances affecting cognitive functioning and treatment stability [18].

Pharmacogenomics represents a rapidly evolving field within precision medicine that may fundamentally transform the approach to lithium therapy by enabling individualized dosing and prediction of treatment response prior to initiation. Identification of genetic variants may facilitate more accurate prediction of treatment efficacy and the risk of adverse effects, including endocrine disturbances [19]. The integration of clinical, laboratory, digital, and pharmacogenomic data may contribute to a personalized therapeutic approach, enhancing treatment safety and improving long-term quality of life in patients with bipolar disorder [18].

Future research should prioritize the development and validation of personalized monitoring and treatment protocols that maximize the clinical benefits of lithium therapy while minimizing the risk of endocrine complications.

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