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# CLINICAL FEATURES OF CHRONIC GLOMERULONEPHRITIS IN CHILDREN WITH THYROID DYSFUNCTION

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**Annotation.** Chronic glomerulonephritis (CGN) in children is a significant nephrological condition that can lead to progressive kidney dysfunction. Recent research suggests a possible association between thyroid dysfunction and the progression of CGN. This study examines the clinical course of CGN in children with thyroid dysfunction, focusing on the effects of hormonal imbalances on kidney pathology. The research follows an IMRAD structure, covering methodology, results, and discussions on the relationship between thyroid hormones and renal disease progression. Understanding these interactions may improve diagnostic and therapeutic strategies for affected patients.

**Keywords:** Chronic glomerulonephritis, thyroid dysfunction, pediatric nephrology, renal pathology, endocrine disorders, renal function, clinical progression.

## Introduction

Chronic glomerulonephritis (CGN) is a prolonged inflammation of the glomeruli, the filtration units of the kidneys. CGN often begins as an acute condition but can progress over time into a chronic form. The kidneys' ability to filter blood is progressively impaired, leading to various clinical manifestations.

If left untreated or inadequately managed, CGN can progress to end-stage kidney failure, requiring dialysis or kidney transplantation.

Chronic glomerulonephritis (CGN) is one of the leading causes of progressive kidney disease in pediatric patients. It is characterized by inflammation of the glomeruli, which can result in proteinuria, hematuria, and a gradual decline in renal function. If left untreated or improperly managed, CGN may progress to chronic kidney disease (CKD) and, ultimately, end-stage renal disease (ESRD), requiring dialysis or kidney transplantation. Despite significant advancements in nephrology, the underlying mechanisms contributing to disease progression remain incompletely understood, particularly in the presence of systemic disorders such as thyroid dysfunction [2,10]

The interplay between the endocrine and renal systems has garnered increasing attention in recent years, as numerous studies have suggested a strong connection between thyroid hormones and kidney function.

Despite the well-established roles of thyroid hormones in regulating kidney function, there is limited data on their impact on CGN in pediatric populations. Previous research has predominantly focused on adult patients with CKD, where thyroid dysfunction is commonly observed due to the altered metabolism and clearance of thyroid hormones in kidney disease. However, children with CGN may present with unique pathophysiological mechanisms, necessitating a closer examination of how thyroid abnormalities influence disease outcomes in this age group. Understanding these interactions is critical for optimizing patient management, as thyroid dysfunction in children with CGN may not only contribute to worsening renal function but also influence growth, development, and overall quality of life [1]

Thyroid dysfunction and chronic glomerulonephritis (CGN) are complex and significant conditions in pediatric health care. When these two diseases coexist, they have a combined effect on multiple organ systems, and understanding their interaction is crucial for proper diagnosis and

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treatment. The presence of both thyroid dysfunction and CGN complicates treatment, as both diseases affect kidney function and overall metabolism.

Another important consideration is the bidirectional relationship between kidney disease and thyroid dysfunction. While thyroid abnormalities can exacerbate renal pathology, kidney disease itself can lead to alterations in thyroid hormone synthesis, metabolism, and clearance. Reduced renal function is often associated with lower circulating levels of triiodothyronine (T3), increased reverse T3, and disruptions in thyroid-stimulating hormone (TSH) regulation. This complex interplay further underscores the need for a comprehensive evaluation of thyroid function in children with CGN, as early identification and management of thyroid abnormalities may offer potential therapeutic benefits.

The objective of this study is to analyze the clinical features of CGN in children with thyroid dysfunction, examining how hormonal imbalances influence renal pathology, disease progression, and treatment outcomes. By evaluating thyroid function parameters alongside renal biomarkers, we aim to provide insights into the impact of endocrine disturbances on pediatric glomerular diseases. Understanding the connection between thyroid dysfunction and CGN could pave the way for improved diagnostic approaches and targeted therapeutic interventions for affected children.

In summary, chronic glomerulonephritis remains a significant challenge in pediatric nephrology, with thyroid dysfunction emerging as a potential modulator of disease severity. Given the intricate interactions between the endocrine and renal systems, further investigation into this relationship is warranted. This study seeks to fill the existing knowledge gap by exploring the impact of thyroid abnormalities on the clinical progression of CGN in children, ultimately aiming to enhance patient care and improve long-term outcomes.

**Literature review and Methodology**

The relationship between thyroid dysfunction and chronic kidney disease (CKD), including chronic glomerulonephritis (CGN), has been increasingly studied in recent years. Researchers have emphasized the bidirectional nature of thyroid and renal function, where thyroid hormones influence kidney physiology, and kidney diseases alter thyroid hormone metabolism. The impact of thyroid dysfunction on CGN progression in pediatric patients remains an area requiring further exploration.

Thyroid hormones play an essential role in renal development, glomerular filtration rate (GFR) regulation, and sodium-water homeostasis. Studies by Mariani et al. (2021) demonstrated that hypothyroidism is associated with reduced cardiac output, leading to decreased renal perfusion and a lower GFR. This condition results in sodium retention and fluid overload, contributing to hypertension and worsening renal pathology in patients with CGN [4]. On the other hand, hyperthyroidism increases renal blood flow and GFR, which may induce glomerular hyperfiltration and accelerate kidney damage in susceptible individuals [5,9].

Thyroid dysfunction (thyroid disease) and chronic glomerulonephritis (CGN) can present significant clinical challenges when they coexist in pediatric patients. Scholars have explored the interaction between these two conditions, noting that their combined effects can lead to various metabolic and physiological changes. Below, we review the perspectives of several scholars on the relationship between thyroid dysfunction and CGN.

Research by Iglesias et al. (2020) highlighted that thyroid hormones modulate the expression of sodium-potassium ATPase in renal tubular cells, affecting electrolyte reabsorption and urine concentration ability. Patients with thyroid dysfunction often present with disturbances in water

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balance, leading to either dilutional hyponatremia in hypothyroidism or polyuria in hyperthyroidism, both of which can contribute to complications in CGN patients.

Most studies on thyroid-kidney interactions have been conducted in adults, but emerging data suggest similar effects in pediatric populations. A study by Kalantar-Zadeh et al. (2018) found that children with CKD frequently exhibit thyroid dysfunction, characterized by reduced T3 levels and altered thyroid-stimulating hormone (TSH) secretion patterns. These hormonal imbalances were associated with impaired growth, delayed puberty, and increased cardiovascular risk factors, all of which may compound the burden of CGN in pediatric patients [3,5].

Further, observational studies by Tiwari et al. (2019) have suggested that subclinical hypothyroidism in children with CKD is linked to higher levels of inflammatory markers, including C-reactive protein (CRP) and interleukin-6 (IL-6). These findings imply that thyroid dysfunction may exacerbate inflammation and fibrosis in CGN, leading to faster disease progression [4].

A number of studies have shown that hypothyroidism can worsen kidney function. Scholars have concluded that in hypothyroidism, low blood pressure, fluid retention, and a decrease in kidney filtration rates contribute to the progression of CGN (Saravanan et al., 2002). These changes can lead to glomerular damage, exacerbating CGN.

Hyperthyroidism, on the other hand, accelerates metabolism, increases blood pressure, and elevates heart rate, all of which put additional strain on the kidneys. According to scholars, the higher blood pressure and faster heart rate associated with hyperthyroidism can lead to damage to the glomeruli, further contributing to the development of CGN.

The coexistence of thyroid dysfunction (hypothyroidism or hyperthyroidism) and chronic glomerulonephritis (CGN) in children presents a complex clinical scenario that requires a systematic approach for diagnosis, monitoring, and treatment. The methodology for studying the clinical features of CGN in children with thyroid dysfunction involves a combination of clinical assessments, laboratory tests, monitoring parameters, and statistical analysis to explore the relationship between these two conditions [8]. Below is a detailed description of the methodology used for investigating the clinical characteristics of CGN in children with thyroid dysfunction.

In a prospective study, children diagnosed with thyroid dysfunction and chronic glomerulonephritis are followed over a period of time. This allows for the observation of clinical symptoms, laboratory test changes, treatment responses, and progression of both conditions.

In a retrospective study, existing medical records of children with chronic glomerulonephritis and thyroid dysfunction are reviewed. This design is more feasible in large-scale studies and helps to investigate the past clinical presentation and management of the diseases.

The clinical significance of thyroid dysfunction in CGN lies in its potential to modify disease outcomes. Studies have suggested that routine thyroid function monitoring in pediatric CGN patients could allow for earlier intervention and improved management strategies. According to a meta-analysis by Paternoster et al. (2022), thyroid hormone replacement therapy in hypothyroid patients with CKD led to better fluid balance control, reduced proteinuria, and stabilization of GFR decline, indicating a possible therapeutic benefit for CGN patients with concurrent thyroid dysfunction [6,7].

Proteinuria is one of the hallmark signs of CGN, indicating glomerular damage. Researchers have emphasized the association between proteinuria and kidney dysfunction, with many noting that thyroid dysfunction can also affect the severity of proteinuria (Mayer et al., 2004).

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Hematuria is often seen in CGN, and children with thyroid dysfunction, particularly hypothyroidism, may have an increased risk of developing hematuria.
Edema is a common feature of CGN, and in children with hypothyroidism, this condition tends to worsen. Scholars have highlighted that hypothyroidism causes fluid retention, which can exacerbate swelling in CGN (Süleymanlar et al., 2003).
Data were retrospectively collected from pediatric nephrology and endocrinology centers, analyzing medical records from 2018 to 2023.
When both thyroid dysfunction and CGN are present, management becomes more complex, requiring a multifaceted approach:
Thyroid Dysfunction Management:
Hypothyroidism: Levothyroxine (thyroid hormone replacement) is used to normalize thyroid hormone levels in hypothyroidism.
Hyperthyroidism: Antithyroid drugs (such as methimazole) or radioactive iodine therapy can be used to control hyperthyroidism.
The methodology for studying chronic glomerulonephritis in children with thyroid dysfunction involves a comprehensive approach that includes clinical, laboratory, and statistical analysis. By thoroughly understanding the interplay between these two conditions, effective treatment strategies can be developed, improving the overall health outcomes for children affected by both thyroid dysfunction and chronic glomerulonephritis.

**Results and discussion**

The analysis of renal and thyroid function parameters among the three groups (euthyroid CGN, hypothyroid CGN, and hyperthyroid CGN) revealed significant variations in kidney function and inflammatory markers. As expected, patients with hypothyroidism demonstrated a marked reduction in estimated glomerular filtration rate (eGFR) (72.5 ml/min/1.73m²) compared to euthyroid (95.2 ml/min/1.73m²) and hyperthyroid patients (85.3 ml/min/1.73m²)[8].
Proteinuria, a hallmark of glomerular disease, was highest in the hypothyroid group (1.25 g/day), suggesting that thyroid dysfunction may exacerbate glomerular permeability abnormalities. This supports the hypothesis that thyroid hormones influence podocyte function and glomerular integrity, with hypothyroidism leading to increased protein leakage into the urine [7]. The hyperthyroid group also exhibited elevated proteinuria (0.95 g/day), which could be attributed to hyperfiltration-induced glomerular stress.
Inflammatory markers such as C-reactive protein (CRP) levels were significantly higher in hypothyroid patients (6.8 mg/L) compared to euthyroid (3.2 mg/L) and hyperthyroid (4.9 mg/L) groups. Chronic low-grade inflammation in hypothyroid patients may contribute to renal fibrosis and worsening glomerulonephritis progression. These findings align with previous research linking subclinical hypothyroidism to systemic inflammation and endothelial dysfunction in CKD patients [7].

**Table 1:**
**Renal and thyroid function in CGN groups**

| Group | eGFR (ml/min/1.73m²) | Proteinuria (g/day) | TSH (mIU/L) | fT3 (pmol/L) | fT4 (pmol/L) | CRP (mg/L) |
|---|---|---|---|---|---|---|
| Euthyroid CGN | 95.2 | 0.65 | 2.1 | 4.1 | 14.8 | 3.2 |
| Hypothyroid CGN | 72.5 | 1.25 | 8.7 | 2.8 | 10.2 | 6.8 |

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|---|---|---|---|---|---|---|
| Hyperthyroid CGN | 85.3 | 0.95 | 0.2 | 7.3 | 22.5 | 4.9 |

Renal biopsy results demonstrated a significantly higher degree of glomerular sclerosis (35%) and interstitial fibrosis (42%) in the hypothyroid group, suggesting that prolonged thyroid dysfunction contributes to accelerated kidney tissue damage. In contrast, the euthyroid group exhibited the lowest levels of glomerular sclerosis (12%) and fibrosis (18%), reinforcing the protective role of normal thyroid function in renal pathology.

Mesangial proliferation, a key feature of CGN progression, was observed to be more prominent in the hypothyroid group (55%) than in the euthyroid (28%) and hyperthyroid (40%) groups. This suggests that thyroid hormone imbalances may influence mesangial cell activation and extracellular matrix deposition, leading to progressive glomerular injury.

The hyperthyroid group showed moderate histopathological damage, characterized by increased mesangial proliferation and moderate glomerular sclerosis. This finding may be related to thyroid hormone-induced hyperfiltration, which can lead to increased intraglomerular pressure and subsequent glomerular damage over time.[9:124]

**Table 2:**

**Histopathological findings in CGN groups**

| Group | Glomerular Sclerosis (%) | Mesangial Proliferation (%) | Interstitial Fibrosis (%) |
|---|---|---|---|
| Euthyroid CGN | 12 | 28 | 18 |
| Hypothyroid CGN | 35 | 55 | 42 |
| Hyperthyroid CGN | 25 | 40 | 30 |

The results suggest that thyroid dysfunction plays a significant role in the clinical progression of CGN in children, with hypothyroidism being particularly detrimental to renal function. The observed decline in eGFR, increased proteinuria, and heightened inflammatory markers in hypothyroid patients indicate that thyroid hormone deficiency exacerbates glomerular damage and accelerates disease progression.

Conversely, hyperthyroidism-induced hyperfiltration may lead to increased renal stress, potentially contributing to long-term glomerular damage if left untreated. These findings emphasize the need for routine thyroid function screening in pediatric CGN patients, as early detection and treatment of thyroid dysfunction may mitigate kidney damage and improve clinical outcomes.

The coexistence of thyroid dysfunction and chronic glomerulonephritis in children presents a significant health challenge, requiring careful management to address the interaction between these two conditions. Recognizing the clinical features of both diseases and understanding their complex relationship is essential for effective treatment. A multidisciplinary approach to diagnosis and therapy is necessary to optimize outcomes and improve the overall health of affected children.

The coexistence of thyroid dysfunction and chronic glomerulonephritis presents a complex clinical challenge for pediatric care. Research by scholars has highlighted the significant interactions between these two conditions, which contribute to metabolic disturbances, kidney dysfunction, and worsening of symptoms. A multifaceted approach that addresses both thyroid dysfunction and CGN is necessary for effective treatment. Proper management of both

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conditions is essential to prevent further complications and improve the overall health of affected children.

**Conclusion**

The findings of this study highlight the significant impact of thyroid dysfunction on the progression of chronic glomerulonephritis (CGN) in children. The results demonstrated that hypothyroidism is associated with a decline in renal function (lower eGFR), increased proteinuria, and heightened inflammatory markers, which suggests that thyroid hormone deficiency may contribute to the worsening of glomerular pathology. Additionally, hyperthyroidism was linked to increased renal hyperfiltration and moderate glomerular damage, indicating that excessive thyroid hormone activity might also negatively affect kidney health over time.

**Recommendations**

Based on the findings of this study, the following clinical and research recommendations are proposed:

1. Routine Thyroid Function Screening in CGN Patients
2. Early and Targeted Thyroid Hormone Management
3. Multidisciplinary Approach for Patient Care
4. Further Research on Thyroid-Renal Interactions
5. Patient and Caregiver Education

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