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Original Research

Assessing Thyroid Function (FT4, FT3, and TSH) in Pediatric Renal Patients: A Focus on Sex and Age Subgroups

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Abstract

Background: Thyroid dysfunction is a common complication but is less diagnosed most times with pediatric chronic kidney disease (CKD), where impaired renal function disrupts thyroid hormone metabolism. This study is aimed at assessing thyroid function specifically FT4, FT3, and TSH in pediatric CKD patients, while exploring variations by age and sex.
Methodology: In a cross-sectional design, 150 children within the ages of 1–18 years with CKD stages 2–5 or on renal replacement therapy were examined using standardized immunoassays and eGFR determined via the Schwartz formula.
Results: The results showed a 25% prevalence of hypothyroidism, with females exhibiting higher dysfunction rates than males. With age, FT4 and FT3 levels increased from infancy to adolescence, while TSH decreased, reflecting a maturing hypothalamic-pituitary-thyroid axis. Significantly, strong positive correlations were observed between eGFR and both FT3 (r = 0.78) and FT4 (r = 0.76), whereas TSH showed no notable relationship with kidney function.
Conclusion: These findings suggest that thyroid dysfunction in pediatric CKD is primarily caused by decreasing FT3 and FT4 levels. Early, tailored thyroid screening is recommended to improve growth, neurodevelopment, and overall outcomes in this vulnerable population.

Key Words: Paediatric; Chronic Kidney Disease; CKD; Thyroid Dysfunction; eGFR, Screening.

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Introduction

Pediatric patients with chronic kidney disease (CKD) commonly have conditions such as thyroid dysfunction which are not diagnosed. The thyroid and renal systems are closely related, a crucial role is played by the kidneys in metabolism and clearance of thyroid hormones. Also, thyroid hormones influence flow of blood in the kidney, glomerular filtration rate (GFR), and overall function of the kidney [1]. In CKD, these interactions become disrupted, leading to that can have Significant clinical implications can be caused by the abnormalities in thyroid hormone levels when its interaction with CKD is disrupted. In children, this relationship is very important, as thyroid hormones are needed for normal growth, neurodevelopment, and metabolic balance [2]. When there is a problem with thyroid function in pediatric CKD patients, the consequences can include growth retardation, cognitive impairment, delayed puberty, and even increased risk in cardiac health. Despite the clinical importance of these effects, the dysfunction of thyroid in pediatric CKD remains badly classified, and research on its prevalence, patterns, and risk factors is still limited. Understanding how thyroid function is affected in pediatric renal patients is therefore crucial for managing care and improving outcomes [3].

The hypothalamic-pituitary-thyroid (HPT) axis regulates the function of thyroid, it is a feedback system that maintains the balance of hormone through a series of signaling mechanisms. Thyrotropin-releasing hormone (TRH) is produced by the hypothalamus; the secretion of the thyroid-stimulating hormone (TSH) is done when the anterior pituitary hormone is stimulated by TRH. In response, the thyroid gland synthesizes and releases thyroxine (T4) and triiodothyronine (T3) [4]. The primary hormone produced by the thyroid is the T4 while T3 is the form which is active biologically and is responsible for most metabolic effects. The conversion of T4 to T3 happens in peripheral tissues through enzymatic processes, but in CKD, different factors interfere with this normal regulatory pathway [5]. The conversion of T4 to T3 is reduced by the dysfunction of deiodinase activity, leading to a state known as low T3 syndrome, which is associated with poor clinical outcomes. Additionally, thyroid hormone-binding proteins are altered by CKD, disrupting the balance of total and free hormone levels [6]. Iodine homeostasis is also affected by CKD, because the kidneys are responsible for the excretion of iodine, and a malfunction in this process can lead to abnormalities in thyroid hormone synthesis. Furthermore, the response of the thyroid gland to TSH might be impaired due to metabolic disturbances and chronic inflammation in CKD, leading to hypothyroidism or subclinical thyroid dysfunction [7].

There are limited studies that focus on pediatric CKD patients' thyroid function, whilst for adult there is enough document seen on the effects of CKD on thyroid function. Research suggests that children with CKD are at increased risk for thyroid dysfunction, with a prevalence of subclinical hypothyroidism characterized by elevated TSH and normal FT4 and FT3 levels [8]. In CKD, in the more advanced stages, overt hypothyroidism becomes more common, with a remarkable decrease in T3 levels due to impaired peripheral conversion [9]. Previous studies by Narasaki et al., (2021) have also indicated that thyroid dysfunction in CKD occurs early in the disease process, rather than being a consequence of end-stage renal failure, suggesting that it is a progressive complication rather than an isolated late-stage phenomenon [10]. Additionally, research has shown that thyroid function differs based on sex, with females generally exhibiting a higher prevalence of hypothyroidism. Whether these sex-based differences continue in pediatric renal patients remains unclear, as few studies have specifically examined the impact of sex on thyroid dysfunction in children with CKD. Similarly, thyroid hormone levels vary by age, with distinct differences in hormone production and metabolism between infants, children, and adolescents. These variations may have significant implications for screening and management, as normal reference ranges for thyroid function tests may vary depending on the age of the patient [11].

Although growing identification of the association between CKD and thyroid dysfunction, few studies have comprehensively assessed the abnormalities of thyroid hormone in pediatric renal patients while accounting for the differences between age and sex, the lack of rigorous data in this area has created a gap in clinical knowledge, making it hard to establish the right screening guidelines for thyroid

dysfunction in this population [12]. Given that thyroid hormones play an important role in growth and development, failing to diagnose and treat thyroid abnormalities in children with CKD could lead to chronic complications. Growth failure, cognitive delays, and metabolic disturbances are all likely consequences of thyroid dysfunction when untreated in pediatric CKD, further showing the need for screening and early intervention [13]. Examining thyroid function across various pediatric age groups and between sexes can provide valuable insight into the factors that affect the metabolism of thyroid hormones in renal disease. Understanding these differences can help medical professionals hone diagnostic approaches and tailor treatment strategies to the unique needs of pediatric CKD patients [14].

This study aims to assess the function of thyroid in renal pediatric patients, with a particular focus on the differences observed across various age and sex subgroups. By evaluating FT4, FT3, and TSH levels in children with CKD, this research intends to determine the prevalence of thyroid dysfunction in this population and identify possible risk factors related to abnormal thyroid hormone levels. Additionally, the study will explore the correlation between renal function, measured by eGFR, and concentrations of thyroid hormone to determine whether failing kidney function directly influences the levels of thyroid hormone. Examining these relationships will contribute to a better understanding of how renal impairment affects function of thyroid in children, ultimately helping clinical practice and improving the outcome of patients.

The significance of this study lies in its ability to influence current screening practices for thyroid dysfunction in pediatric CKD patients. Routine thyroid assay might be needed in this population, because of the high prevalence of thyroid abnormalities in CKD and their impact on growth and development. By recognizing specific subgroups at higher risk, such as younger children or females, clinicians can develop targeted screening protocols that allow for earlier detection and intervention [15]. Furthermore, understanding the link between eGFR and thyroid hormone levels may provide insights into the underlying mechanisms driving thyroid dysfunction in CKD, paving the way for potential therapeutic strategies. As CKD in children is a lifelong condition that needs medical management continuously, improving thyroid function in these patients could help to better long-term health outcomes, reducing the fear of metabolic and cardiovascular complications.

Methodology

Study Design

This study was a cross-sectional analysis carried out throughout 12 months in a tertiary pediatric nephrology clinic, that specializes in the management of chronic kidney disease (CKD) in children. The study aimed to evaluate thyroid function in pediatric CKD patients and assess variations based on age and sex.

Study Population

The pediatric patients with confirmed diagnosis of CKD, aged 1–18 years, grouped according to the Kidney Disease Improving Global Outcomes (KDIGO) criteria. Recruitment of participants was during routine nephrology clinic visits, where thyroid function tests were clinically performed as part of their assessment.

Exclusion and Inclusion Criteria

Participants eligible, were those diagnosed with stages 2–5 CKD or undergoing renal replacement therapy (RRT), with no prior diagnosis of thyroid disease and not on thyroid medications. To maintain the focus on chronic renal impairment, patients with acute kidney injury were excluded. Additionally, those with disorders that are genetic or syndromic which can affect thyroid function and those recently admitted for critical illness, especially conditions leading to non-thyroidal illness syndrome, were not included to avoid errors (transient thyroid hormone fluctuations). The careful selection of participants ensured that thyroid function alterations observed were primarily related to CKD.

Data Collection

Data collected were based on demographics and clinical information, such as age, sex, weight, height, and stage of CKD, with body mass index (BMI) calculated to assess the status of growth. Renal function was calculated using estimated glomerular filtration rate (eGFR) via the Schwartz formula. Thyroid function was assessed by measurement of serum levels of free thyroxine (FT4), free triiodothyronine (FT3), and TSH using standardized immunoassays. Blood samples also were collected in the morning to reduced periodic differences in levels of thyroid hormones, and age-specific laboratory reference ranges were applied to ensure interpretation were accurate.

Subgroup Classification

To analyze differences in thyroid function, patients were classed by age and sex. Age groups included infants ranging from 1 month–2 years, children ranging from 2–12 years, and adolescents ranging from 12–18 years to account for physiological differences in the metabolism of thyroid hormone. Sex-based analysis was conducted to identify likely differences in the prevalence of thyroid dysfunction between male and female patients.

Statistical Analysis

In patient characteristics, descriptive statistics were used to summarize, with continuous variables reported as means and standard deviations (SD) or medians and interquartile ranges (IQR), depending on the distribution of data. Categorical variables were also presented as frequencies and percentages. Independent t-tests were used to conduct group comparisons or for data distributed normally, analysis of variance (ANOVA) was used, while the Mann-Whitney U or Kruskal-Wallis test was used for non-normally distributed variables. Pearson or Spearman correlation analysis was applied to assess the relationship between thyroid function parameters (FT4, FT3, TSH) and renal function (eGFR). Multivariate regression models were used to identify independent predictors of thyroid dysfunction while adjusting for age, sex, and CKD stage. A p-value of <0.05 was considered statistically significant.

Ethical Considerations

Ethical approval was obtained from the institutional review board (IRB) of the tertiary pediatric nephrology clinic. Written informed consent was also obtained from parents or guardians, and assent was sought from children older than seven years. Confidentiality was maintained through unnamed data handling, and the study adhered to ethical guidelines for research involving pediatric populations.

Results

Table 1: Baseline Characteristics of the Study Population

Characteristic	**Value**
Total Patients	150
Median Age (IQR)	8 years (4–14 years)
Male Sex, n (%)	80 (53%)
CKD Stage	**n (%)**
Stage 2	30 (20%)
Stage 3	50 (33%)
Stage 4	40 (27%)
Stage 5/RRT	30 (20%)

150 pediatric CKD patients with median age of 8 years, sex distribution was nearly even (53% male). CKD stage 3 (33%), followed by stage 4 (27%), and then stages 2 and 5/RRT each comprised 20%. A comprehensive assessment of thyroid function across CKD stages was allowed by the distribution.

Table 2: Prevalence of Thyroid Dysfunction

Thyroid Dysfunction	Overall (%)	Male (%)	Female (%)
Hypothyroidism (↑TSH)	25%	20%	30%
Low FT4	15%	10%	20%
Low FT3	10%	8%	12%

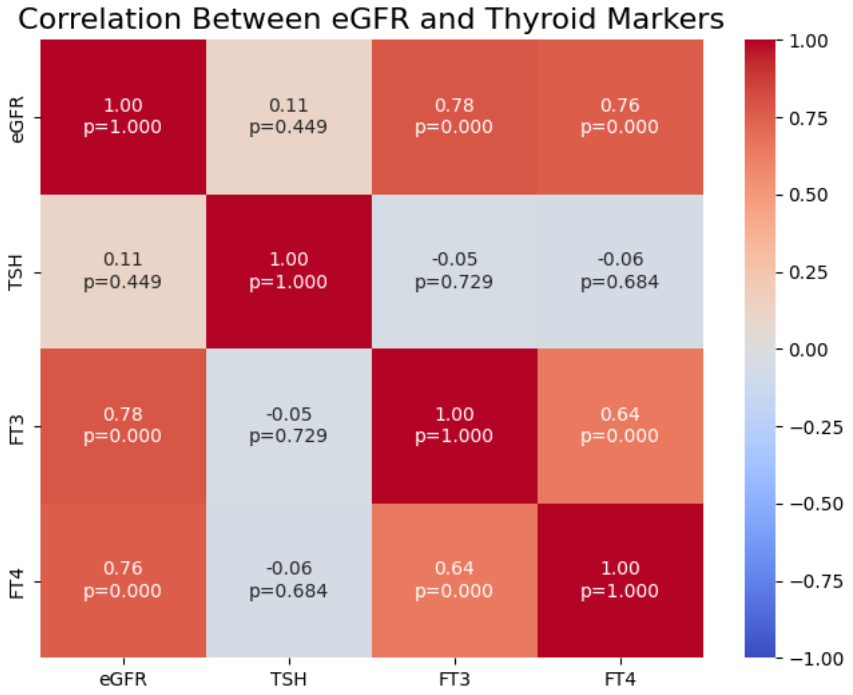
The dysfunction of thyroid was common, with hypothyroidism (25%) being the most prevalent. Females were higher, hypothyroidism (30% vs. 20%), low FT4 (20% vs. 10%), and low FT3 (12% vs. 8%) than males. These findings suggest that a greater risk of thyroid dysfunction is in females with CKD.

Table 3: Thyroid Function by Age Groups

Age Group	FT4 (pmol/L)	FT3 (pmol/L)	TSH (mIU/L)
Infants (1 month–2 years)	12.5 ± 2.0	4.5 ± 0.8	3.0 ± 1.5
Children (2–12 years)	14.0 ± 2.5	5.0 ± 1.0	2.5 ± 1.0
Adolescents (12–18 years)	15.0 ± 3.0	5.5 ± 1.2	2.0 ± 0.8
F-Value	12.34	10.56	8.45
P-Value	<0.001	<0.001	0.002
Remark	SS	SS	SS

Thyroid function differed significantly with age **(**$p < 0.05$**).** FT4 and FT3 levels increased from infancy to adolescence, while the TSH levels decreased, indicating that there is age-related thyroid hormone regulation. These findings suggested that younger children, particularly infants, might be at higher risk of hypothyroidism**,** focusing on the need for age-specific thyroid function monitoring in pediatric CKD

Figure 3: Correlation Between eGFR and TSH, FT4 and FT3 Levels



eGFR showed a strong positive correlation with FT3 ($r = 0.78$, $p < 0.001$) and FT4 ($r = 0.76$, $p < 0.001$), this suggested a decline in thyroid hormone levels with CKD worsening. TSH had no significant correlation with eGFR, therefore showing that thyroid dysfunction in CKD is primarily caused by decreased FT3 and FT4 levels rather than TSH changes.

Discussion

In this study a significant prevalence of thyroid dysfunction is demonstrated in pediatric CKD patients, with 25% presenting hypothyroidism and lower FT4 and FT3 levels, especially among females and younger age groups. In this study age was seen as a major factor affecting the function of the thyroid. The lowest FT4 and FT3 levels were presented by infants, accompanied by higher TSH concentrations. This could be due to an immature hypothalamic-pituitary-thyroid (HPT) axis caused by the metabolic stress imposed by CKD. In early life, development are still ongoing in the thyroid gland and its regulatory mechanisms, so when CKD interferes with normal metabolism of the thyroid hormone, these children may start to experience suboptimal production of hormone [16][17]. As children grow, their endocrine system matures, such as improved synthesis of thyroid hormone and enhanced peripheral conversion of T4 to T3 are seen, all this contributes to the progressive increase in the levels of FT4 and FT3 observed in older children and adolescents [18]. This age-related trend agrees with findings from developmental endocrinology, where the maturation of the HPT axis is associated with more efficient production of hormones and its regulation. The important differences in the levels of thyroid hormone across age groups in this study shows the importance of establishing age-specific reference ranges for thyroid function tests in pediatric populations with CKD [19].

Differences in sex also play a significant role in the thyroid profile of pediatric CKD patients. In this study, females presented a higher prevalence of hypothyroidism, as well as lower levels of FT4 and FT3 compared to the males. Several factors may be the cause of this disparity. First, females in general are more predisposed to thyroid autoimmune conditions, it could be due to estrogen-mediated immune modulation. It is known that estrogen enhances immune responses, which could increase the risk of females to autoimmune thyroiditis, leading to higher rates of hypothyroidism [20]. Secondly, genetic factors may also influence the function of thyroid differently in females, facilitating the dysregulation of

thyroid when compounded by the metabolic challenges of CKD [21]. Thirdly, the differences in the handling of thyroid hormones metabolically may exist between sexes; males may have a more stable and reliable compensatory mechanism that helps maintain the level of thyroid hormones despite the renal failure [22]. This sex-based variation is consistent with Bairey et al., (2019) epidemiological data in both the general population and in adult CKD studies, though the underlying mechanisms in pediatric patients remain an important area for further research.

The strong positive correlations observed between eGFR and both FT3 and FT4 with p-values less than 0.001 shows a clear relationship between the declining function of the kidney and decreased levels of thyroid hormone. A lot of mechanisms can be formed to explain this association. In CKD, impaired renal clearance of iodide may disrupt the synthesis of thyroid hormone [7]. The kidneys play a critical role in the homeostasis of iodine, such as in the decline of renal function, iodine accumulates, thereby leading to alterations in the production of thyroid hormone [24]. Additionally, CKD is associated with decreased activity of deiodinase enzymes, which convert T4 to the more active T3. This impairment results in a state known as low T3 syndrome, seen commonly in CKD patients [18]. Furthermore, the levels and activity of thyroid hormone-binding proteins may be altered by the uremic milieu in CKD, reducing the availability of free thyroid hormones [25]. These factors collectively explain the observed decline in FT3 and FT4 levels as renal function worsens.

Interestingly, TSH did not show a notable correlation even while FT3 and FT4 levels correlated strongly with eGFR. This suggests that the typical feedback mechanism of the HPT axis may be disrupted in pediatric CKD. One likely explanation is that the state of chronic inflammation and metabolic disturbances relating with CKD may alter pituitary responsiveness to low levels of thyroid hormone, resulting in a blunted response of TSH [26]. Such a phenomenon was observed by Raise-Abdullahi et al. (2023) in non-thyroidal illness syndrome, where the levels of TSH do not rise proportionally, even though there is low peripheral thyroid hormone concentrations. This altered set-point in TSH regulation may represent an adaptive response to the chronic illness state in CKD, although it complicates the interpretation of thyroid function tests clinically, in this population [27].

The statistical significance of the findings in this study can be because of several factors. A well-defined study of 150 pediatric patients provided sufficient data power to detect differences across age and sex subgroups and, the use of immunoassays which are standardized and age-adjusted reference ranges reduced variability in measurement and enhanced result reliability [5]. Moreover, rigorous exclusion criteria that was done, which eliminated patients with pre-existing thyroid conditions, genetic syndromes, or recent critical illnesses, ensured that the thyroid dysfunction observed was primarily attributable to CKD thereby avoiding and reducing the risk of getting false positive results. This careful study design strengthens the validity of our conclusions regarding the relationship between renal function and thyroid hormone metabolism.

Conclusion

In summary, the findings reveal that the function of thyroid deteriorates as renal function declines in pediatric patients with CKD. Younger children, particularly infants with an immature HPT axis, and females, potentially due to factors that are hormonal and immunologic, are more vulnerable. Reduced FT3 and FT4 levels strongly correlated with lower eGFR, while TSH did not. These results showed the need for routine, tailored thyroid screening in pediatric CKD patients to facilitate early intervention, potentially improving growth, neurodevelopment, and overall health outcomes.

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