

ORIGINAL ARTICLE

Frequency of thyroid disorders in children with obesity.

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ABSTRACT... **Objective:** To determine the frequency of thyroid disorders among children with obesity attending public sector hospital of Karachi, Pakistan. **Study Design:** Descriptive, Cross-sectional. **Setting:** Departments of Emergency and Outpatient, National Institute of Child Health (NICH), Karachi, Pakistan. **Period:** March 2024 to August 2024. **Methods:** A total of 193 children aged 5–16 years of either gender with obesity (BMI-for-age ≥ 95 th percentile by WHO standards) were enrolled using non-probability consecutive sampling. Sociodemographic and clinical data were recorded. Fasting blood samples were analyzed for TSH, T3, and T4. Thyroid dysfunction was classified using standard TSH and FT3 and FT4 thresholds. Data were analyzed with SPSS using t-tests and Chi-square, considering $p < 0.05$ significant. **Results:** Of the 193 obese children, the mean age was 11.1 ± 2.8 years, and 105 (54.4%) were boys. There were 135 (69.9%) children who were resided in urban areas, while 124 (64.2%) children had class 1 obesity, and 69 (35.8%) had severe obesity. The mean BMI z-score was 2.56 ± 0.46 . Thyroid dysfunction was detected in 35 children (18.1%). Severe obesity was significantly more common among those with thyroid dysfunction (45.7% vs 33.5%, $p = 0.011$). **Conclusion:** Thyroid dysfunction was frequently observed in obese children, with the highest risk among those with severe obesity.

Key words: Children, Hyperthyroidism, Hypothyroidism, Obesity, Thyroid Disorders.

INTRODUCTION

Childhood obesity is now seen as a serious, ongoing health problem worldwide. In Pakistan, around 6% of pediatric population is estimated to have obesity.^{1,2} Important interaction exist between thyroid function, weight control, and obesity.³ This could be because one of the most recognized targets of thyroid hormone action is energy metabolism.⁴ Isolated hyperthyrotropinemia, or subclinical hypothyroidism (SH), is the most frequent aberration of thyroid function in obese children. A study from Turkey documented 5.2% of obese children and adolescents to have subclinical hypothyroidism.⁵ It is proposed that there exists an adipose tissue-hypothalamus-pituitary-thyroid axis, and leptin is produced by adipocytes influences communication between adipose tissue and the hypothalamus by stimulating the secretion of thyrotropin-releasing hormone, TSH, and the conversion of T4 to T3 in peripheral tissues.^{6,7} A local study noted thyroid function alterations in children with obesity, highlighting the importance of careful

consideration when diagnosing these patients.⁸

The childhood obesity has become a pressing global health issue, with Pakistan witnessing a concerning rise in its prevalence. The interplay between childhood obesity and thyroid disorders remains understudied in Pakistan. Thyroid hormones intricately regulate metabolism, any thyroid dysfunction in obese children could have substantial health implications.⁹ Understanding the prevalence and patterns of thyroid disorders in this specific population is critical for targeted healthcare interventions and management strategies. By understanding the relationship between thyroid dysfunction and childhood obesity in Karachi's public sector hospital, this research seeks to bridge an existing knowledge gap, inform local healthcare practices, and contribute to the broader global understanding of this multifaceted health concern, ultimately improving the health and well-being of obese children in Pakistan and similar settings worldwide.

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This study was planned to determine the frequency of thyroid disorders among children with obesity attending public sector hospital of Karachi, Pakistan.

METHODS

This descriptive, cross-sectional study was conducted at the emergency and outpatient departments of “National Institute of Child Health (NICH), Karachi”, Pakistan during March 2024 to August 2024 following approval from the Institutional Ethical Review Board (IERB-68/2023, dated: 21-02-2024). Inclusion criteria were children of either gender, aged 5–16 years, and who were obese. Children taking steroids or other medications that could induce obesity, and those with diseases known to affect weight such as chronic infections (e.g., tuberculosis), malabsorption syndromes, other endocrine disorders, or congenital anomalies were excluded. Obesity was defined as BMI-for-age at or above the 95th percentile based on WHO reference standards. Severe obesity was defined as a BMI $\geq 120\%$ of the 95th percentile or ≥ 35 kg/m², in accordance with American Academy of Pediatrics guidelines. Class 2 obesity was defined as BMI $\geq 120\%$ to $<140\%$ of the 95th percentile or BMI ≥ 35 to <40 kg/m², while class 3 obesity was defined as BMI $\geq 140\%$ of the 95th percentile or BMI ≥ 40 kg/m². The sample size was calculated using the Epilnfo online calculator, with a confidence level of 95%, margin of error 5%, and an anticipated prevalence of thyroid disorders among obese children as 14.7%¹⁰, resulting in a required sample of 193 participants. Non-probability consecutive sampling was adopted.

After obtaining written informed consent from parents or guardians, detailed sociodemographic and clinical data were collected. Laboratory investigations were performed by collecting fasting blood samples in the morning after 12 hours of fasting. Serum was separated and analyzed for TSH, total triiodothyronine (T3), and total thyroxine (T4) using chemiluminescence immunoassay. The reference ranges used were: TSH 0.5–4.0 mIU/L, T4 4.5–11.5 µg/dL, and T3 0.8–2.0 ng/mL. Thyroid disorder was defined as an elevation of TSH (>4.0 mIU/L) with or without

altered total T3 or T4 concentrations beyond these reference limits. Thyroid dysfunction was further defined as subclinical hypothyroidism if TSH was elevated (>4.0 mIU/L) with normal FT4 (0.8–2.0 ng/dL), while overt hypothyroidism required both elevated TSH and low FT4 (<0.8 ng/dL). Subclinical hyperthyroidism was defined as suppressed TSH (<0.5 mIU/L) with normal FT4, and overt hyperthyroidism as suppressed TSH with elevated FT4 (>2.0 ng/dL). All values were interpreted according to laboratory reference ranges.

Data were analyzed using “IBM-SPSS version 26.0”. Quantitative variables such as age, weight, height, BMI z-score, waist circumference, and hormone levels were presented as mean and standard deviation. Categorical variables including gender, residence, and the presence of thyroid disorder were expressed as frequencies and percentages. The independent sample t-test was used to compare means, while Chi-square or Fisher’s exact test was applied to compare categorical data, taking $p < 0.05$ as significant.

RESULTS

A total of 193 obese children aged 5–16 years were included in the study. The mean age of participants was 11.1 ± 2.8 years, and 105 (54.4%) were boys. There were 135 (69.9%) children who were from urban areas. With respect to obesity classification, 124 (64.2%) children were classified as having class 1 obesity, while 69 (35.8%) were identified as having severe obesity (class II or III). The mean BMI z-score, and waist circumference were 2.56 ± 0.46 , and 86.1 ± 10.7 cm, respectively. Table-1 is showing characteristics of participants studied.

The mean TSH, FT4, and FT3 values were 3.11 ± 2.04 mIU/L, 1.12 ± 0.18 ng/dL, and 3.28 ± 0.60 pg/mL, respectively. Thyroid dysfunction was identified in 35 (18.1%) children. Among these 35 children, 23 (65.7%) had subclinical hypothyroidism, 8 (22.9%) followed by overt hypothyroidism, 3 (8.6%) subclinical hyperthyroidism, whereas 1 (2.9%) child had overt hyperthyroidism (Figure-1).

Characteristics		Number (%)
Gender	Boys	105 (54.4%)
	Girls	99 (45.6%)
Age (years)	5-12	121 (62.7%)
	> 12 to 16	72 (37.3%)
Residence	Urban	135 (69.9%)
	Rural	58 (30.1%)
Obesity classification	Class-1	124 (64.2%)
	Severe obesity (class II or III)	69 (35.8%)

Table-I. Characteristics of obese children (n=193)

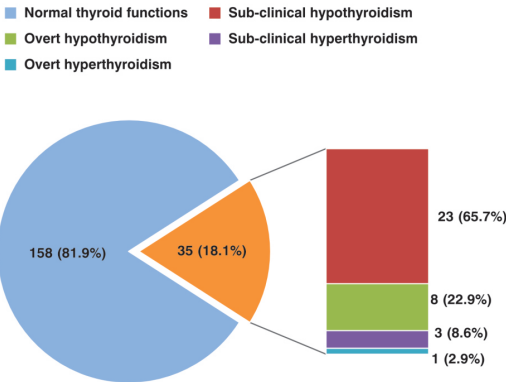


Figure-1. Frequency and distribution of thyroid disorders among obese children

Comparison of baseline characteristics by thyroid status indicated no significant difference between children with and without thyroid dysfunction in terms of gender ($p=0.696$), age ($p=0.701$), and area of residence ($p=0.536$). Severe obesity was significantly more common among children with thyroid dysfunction than those without (45.7% vs 33.5%, $p=0.011$), and the details are shown in Table-II.

DISCUSSION

The frequency of thyroid dysfunction in obese children was 18.1% in this study. Ghergherehchi and Hazhir¹⁰, in an Iranian cohort, reported a subclinical hypothyroidism prevalence of 14.7% among obese children, which parallels the proportion found in the present study. The Turkish cross-sectional analysis by Dundar and Akinci⁵, observed subclinical hypothyroidism in 5.2% of obese children, a somewhat lower rate, potentially attributable to different population characteristics and stricter TSH thresholds. In a US-based study,

Shalitin et al.¹¹, identified hyperthyrotropinemia in 22.2% of obese participants, a prevalence slightly higher than observed in this study.

Characteristics		Thyroid Disorders (n=35)	No Thyroid Disorders (n=158)	p-Value
Gender	Boys	18 (51.4%)	87 (55.1%)	0.696
	Girls	17 (48.6%)	71 (44.9%)	
Age (years)		11.3±2.7	11.1±2.8	0.701
Residence	Urban	26 (74.3%)	109 (69.0%)	0.536
	Rural	9 (25.7%)	49 (31.0%)	
Obesity classification	Class-1	19 (54.3%)	105 (66.5%)	0.011
	Severe obesity (class II or III)	16 (45.7%)	53 (33.5%)	

Table-II. Association of thyroid disorders with characteristics of obese children (N=193)

The predominance of subclinical hypothyroidism is significant, given ongoing debate regarding its etiology in pediatric obesity. Kartal et al.¹², and Ghergherehchi and Hazhir¹⁰, observed a positive correlation between BMI z-score and TSH concentration, suggesting a mechanistic interplay. Some researchers have indicated that even high-normal TSH levels in overweight and obese children were associated with adverse metabolic parameters, reinforcing the notion that thyroid axis alterations may reflect a compensatory response to increased adiposity rather than primary thyroid pathology.¹²

Hyperthyroidism, including both subclinical and overt forms, was found in less proportion in this study. This finding is in accordance with large regional pediatric data, where hyperthyroidism was identified in less than 5% of cases, most commonly attributed to Graves' disease or other rare etiologies.¹³ The present study's exclusion of children with known endocrine or congenital disorders may have further limited the detection of such conditions. These results support the notion that the principal thyroid abnormality in pediatric obesity is hypothyroidism, usually subclinical and mild in degree.^{14,15}

This study documented a significant association between severe obesity and thyroid dysfunction (45.7% among those with thyroid disorders compared to 33.5% among euthyroid children, $p=0.011$). The clustering of thyroid dysfunction in the severely obese subgroup corroborates with contemporary observations that TSH concentrations were directly linked to anthropometric indices, including waist circumference.¹¹ Data from developed world also demonstrates that mild TSH elevations are not uncommon in the absence of autoimmune thyroid disease, with no special clinical characteristics differentiating such children from those with normal TSH levels.¹⁶ These convergent findings raise the possibility that elevated TSH in childhood obesity is an adaptive, potentially reversible phenomenon, a view supported by the lack of increased thyroid antibody positivity.¹⁰

The clinical relevance of subclinical hypothyroidism in obese children remains a matter of debate. Dundar and Akinçi,⁵ reported that children with subclinical hypothyroidism demonstrated higher serum insulin, HOMA-IR, triglyceride, and atherogenic indices, along with lower HDL cholesterol, compared to euthyroid obese peers. The current study did not assess these metabolic parameters directly, but the observed high prevalence of subclinical hypothyroidism in severe obesity reinforces the imperative to monitor cardiometabolic risk factors in this group.^{17,18} Significant positive correlations between TSH, fasting blood glucose, and cholesterol levels, have been found in the past, indicating that thyroid dysfunction in obesity may exacerbate metabolic risk profiles.¹⁹ Mahdavi et al.²⁰, examining adult populations, found that obesity was associated with increased odds of overt hypothyroidism and thyroid peroxidase antibody positivity, but not with subclinical hypothyroidism.

Clinical implications of this study are substantial for pediatric practice. The high prevalence of thyroid dysfunction among obese children, particularly those with severe obesity, underscores the importance of thyroid function assessment in this population, not only for the

identification of primary thyroid disease but also as part of a broader cardiometabolic risk evaluation.^{21,22} The demonstrated association between severe obesity and thyroid dysfunction suggests suspicion for hormonal abnormalities in this group, particularly when weight loss is unexpectedly slow or when other metabolic derangements are present.²³ Given the evidence for reversibility with weight loss, clinicians should prioritize non-pharmacological management and reinforce the message that most mild thyroid abnormalities in obesity do not require lifelong treatment.

There are several limitations to this study that merit consideration. The cross-sectional design precludes any inference regarding causality or the temporal relationship between obesity and thyroid dysfunction. Future research should incorporate assessment of thyroid autoimmunity to clarify the underlying mechanisms. The lack of comprehensive metabolic profiling, including insulin resistance and lipid panels, restricts the capacity to explore the full spectrum of obesity-related cardiometabolic risk associated with thyroid dysfunction; inclusion of these variables in future studies would enhance understanding of the clinical significance of mild thyroid hormone disturbances. The single-center, hospital-based recruitment strategy may introduce referral bias, potentially inflating the observed prevalence of thyroid dysfunction compared with community samples, multicenter, population-based investigations are warranted to confirm the generalizability of these findings. Additionally, pubertal status, a known confounder in both thyroid physiology and body composition, was not systematically assessed, which may have influenced hormonal measurements and risk stratification. Future studies should adjust for pubertal staging to enhance interpretability.

CONCLUSION

Thyroid dysfunction, predominantly subclinical hypothyroidism, was a common finding among obese children attending a tertiary care hospital in Karachi, Pakistan. The highest risk of thyroid dysfunction was observed among children with severe obesity.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1	Wafa Nisar: Data collection, responsible for data.
2	Mohsina Noor Ibrahim: Study concept, proof reading.
3	Maira Riaz: Critical revisions, literature review.
4	Versha Rani Rai: Methodology, critical revisions, discussion.
5	Zubair Ahmed Khoso: Literature review, discussion.