THE PLANET

Original Article

Assessment of Body Mass Index Changes after Levothyroxine Therapy in Patients with Hypothyroidism

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ARTICLE INFO

Received: 19 July 2026
Accepted: 25 July 2026
Published Online: 28 July 2026

DOI: 10.5281/zenodo.21781904

Volume: 10, Number: 1, Page: 104-108

e-ISSN: 2789-5912
ISSN: 2617-0817

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ABSTRACT

Background: Hypothyroidism is a common endocrine disease that is marked by inadequate production of thyroid hormone, which is usually manifested by weight gain and increased body mass index (BMI). The study aimed to evaluate the BMI changes after levothyroxine treatment in patients with hypothyroidism. **Methods & Materials:** This prospective study was carried out at the Department of Pharmacology, Dhaka Medical College, between January and December 2022. Purposive sampling was used to enroll 30 adult patients (≥18 years) with newly diagnosed hypothyroidism. Every patient was put on levothyroxine therapy with a dose increase every 4-6 weeks until euthyroidism was attained. At baseline and 12 weeks, anthropometric and thyroid hormone parameters were measured. Data were entered and analyzed using SPSS version 26. **Results:** Most of the patients were women (96.7%) with an average age of 20-39 years. The baseline consisted of 43.3% overweight and 33.3% obese. Following 3 months of levothyroxine therapy, mean weight declined significantly from 65.84 ± 10.21 kg to 64.98 ± 10.10 kg ($p<0.001$), and mean BMI decreased from 27.93 ± 3.66 to 27.56 ± 3.56 kg/m² ($p<0.001$). FT4 increased significantly (8.06 to 16.40 pmol/L, $p<0.001$), while TSH normalized (32.55 to 2.52 mIU/L, $p<0.001$). Weakness, fatigue, constipation, weight gain, and menstrual irregularity were improved symptomatically. **Conclusion:** Levothyroxine treatment led to decreases in body weight and BMI and normalization of thyroid hormone levels and clinical symptoms. These results confirm the metabolic advantage of early LT4 treatment in hypothyroid patients.

Keywords: *Hypothyroidism, levothyroxine, body mass index, thyroid-stimulating hormone, Euthyroidism*

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INTRODUCTION

Hypothyroidism is a type of endocrine disorder that is experienced in clinical practice across the globe and is characterized by insufficient production of thyroid hormones by the thyroid gland. It is characterized by a continuum of clinical manifestations of subclinical hormonal imbalances to overt systemic illness with severe metabolic effects [1]. Epidemiological evidence worldwide has shown that women, especially those of reproductive age, are more prevalent and the disease has significant public health consequences in both developed and developing countries [2]. Hypothyroidism is a major percentage of endocrine outpatient visits in Bangladesh, but there is a lack of population-level data relative to Western cohorts [3]. The hypothyroidism metabolic load is well known. Thyroid hormones are key in the regulation of basal metabolic rate, lipid metabolism, thermogenesis, and body weight homeostasis [4]. The lack of these hormones causes decreased energy expenditure, lipolysis, and fluid retention, which together cause weight gain and elevated BMI, which are characteristic of overt hypothyroidism [5]. Already a major public health issue in South Asia, with the rapid nutritional transition, obesity and overweight are further exacerbated by the overlay of thyroid dysfunction, resulting in a dual metabolic burden in those affected [6]. The pharmacological management of hypothyroidism is based on levothyroxine (LT4) sodium, a synthetic form of thyroxine (T4). It replenishes the levels of circulating thyroid hormones, inhibits high levels of TSH, and is linked with the reversal of the metabolic sequelae of the disease [7]. Although biochemical euthyroidism is a well-defined therapeutic outcome, the degree to which LT4 treatment is associated with clinically significant changes in BMI has been controversial. Several studies have shown a small to large weight loss after treatment, and some studies indicate that full metabolic normalization, especially BMI recovery, might not be possible with pharmacotherapy alone [8]. This gap highlights the necessity of future, protocol-based research that methodically evaluates anthropometric changes and hormonal parameters. Inter-individual differences in dose requirements, patient compliance, dietary habits, and baseline metabolic profile further complicate the relationship between thyroid functioning and body composition [9]. In resource-constrained environments like Bangladesh, where dietary habits and comorbidities are not similar to those in high-income nations, there is a need to produce locally applicable evidence. The current South Asian literature, although expanding, still does not have sufficient prospective data on the short-term BMI response to LT4 treatment in newly diagnosed patients [10]. Moreover, the symptomatic relief that comes with levothyroxine therapy, such as fatigue, constipation, menstrual abnormalities, cold intolerance, etc., may indirectly lead to lifestyle change and also play a role in weight control [11]. The composite impact of LT4 on hormonal and anthropometric variables in a systematic follow-up model is thus of significant clinical and pharmacological interest. Therefore, the study aimed to evaluate the changes in BMI and anthropometric indices after 12 weeks of levothyroxine treatment in newly diagnosed hypothyroid patients in a tertiary teaching hospital in Dhaka, Bangladesh.

METHODS & MATERIALS

This prospective study was carried out in the Department of Pharmacology, Dhaka

Medical College, based on the data obtained in the Endocrinology Outpatient Department of Dhaka Medical College Hospital, Dhaka, Bangladesh. The research was conducted over 12 months, between January 2022 and December 2022. Purposive sampling technique was used to enroll thirty adult patients aged 18 years and above with newly diagnosed hypothyroidism, which was considered suitable due to the clinical and logistical limitations of the study environment. Patients with either overt hypothyroidism, which is low serum free thyroxine (FT4) levels in combination with high levels of thyroid-stimulating hormone (TSH), or subclinical hypothyroidism, which is high levels of TSH and normal FT4, were eligible to be included. Patients were not included in case of serious comorbid illness, secondary hypothyroidism, current pregnancy, or taking medications that are known to influence thyroid functioning or body weight, which guaranteed a homogeneous study population and reduced confounding. Baseline demographic, clinical, and anthropometric data were collected using a structured questionnaire and data collection form after written informed consent. The main variables measured were age, sex, family history of hypothyroidism, body weight, height, and body mass index (BMI), calculated as weight in kilograms/square of height in meters and categorized according to Asian BMI standards normal weight (18.524.9 kg/m 2), overweight (25.029.9 kg/m 2), and obese (30.0 and above Chemiluminescence immunoassay was used to measure serum FT4 and TSH at baseline. Every patient enrolled was put on levothyroxine therapy, with dose changes being done individually at four to six weeks until biochemical euthyroidism was attained. All patients were re-evaluated at the 12-week follow-up regarding the changes in body weight, BMI, thyroid hormone parameters, and clinical symptomatology. IBM SPSS Statistics version 26.0 was used to enter and analyze the data. Qualitative variables were presented in terms of frequency and percentage; quantitative variables were presented in terms of mean and standard deviation. Chi-square test or Fisher's exact test was used to compare between groups with categorical data, and paired t-test was used to compare pre- and post-treatment of continuous variables. A p-value of 0.05 or less was taken to be significant.

RESULTS

Table I represents the demographic distribution of the 30 study patients. Most of them were aged 20-29 years (36.7%), then 30-39 years (26.7%), which is a young adult female population. The 40-49 and 50-59 age groups accounted for 16.7% and 13.3%, respectively, while those aged ≤20 and 60-69 years each represented 3.3%. It was also significantly female-dominated, with 29 of 30 patients (96.7%) female and only 1 male patient (3.3%). This gender distribution is in line with the established increased predisposition of women to thyroid disorders, especially in the reproductive age group.

Table I
Demographic characteristics of the study patients with hypothyroidism, *n*=30.

Category	Variable	Frequency (n)	Percentage (%)
Age group (years)	≤20	1	3.3
	20-29	11	36.7
	30-39	8	26.7
	40-49	5	16.7
	50-59	4	13.3
	60-69	1	3.3
Sex	Male	1	3.3
	Female	29	96.7

Table II shows the baseline clinical and background features of the study population. In 6 patients (20%), a family history of hypothyroidism was reported, and 24 (80%) had no family history of hypothyroidism, indicating that most of the cases in this cohort were probably sporadic. In terms of baseline BMI status, the highest percentage of patients was overweight (43.3%), then obese (33.3%), and normal weight (23.3%).

Table II
Baseline clinical and background characteristics of the study patients, *n*=30.

Category	Variable	Frequency (n)	Percentage (%)
Family history of hypothyroidism	Present	6	20.0
	Absent	24	80.0
Baseline BMI status	Normal weight (18.5-24.9 kg/m²)	7	23.3
	Overweight (25.0-29.9 kg/m²)	13	43.3
	Obese (≥30.0 kg/m²)	10	33.3

Table III compares the clinical profiles of patients before and after 3 months of levothyroxine treatment. Weakness (73.3%), weight gain (56.7%), fatigue/depression (56.7%), and constipation (40%) were the most common baseline symptoms. Post-treatment, statistically significant reductions were observed in weakness (73.3% to 40.0%, p=0.010), weight gain (56.7% to 16.7%, p=0.001), constipation (40% to 6.7%, p=0.006), and fatigue/depression (56.7% to 26.7%, p=0.018). Menstrual status also improved significantly, with regular menstrual cycles rising by 20.7 to 48.3 (p=0.047) and irregular cycles falling by 51.7% to 24.1%. There was no statistically significant change in symptoms like alopecia, hoarseness, oedema, and goiter (p>0.05).

Table III
Clinical features of hypothyroidism before and after 3 months of levothyroxine therapy, *n*=30.

Clinical feature	Before treatment, n (%)	After treatment, n (%)	p-value
Weakness	22 (73.3)	12 (40.0)	0.010
Alopecia	3 (10.0)	1 (3.3)	0.605
Weight gain	17 (56.7)	5 (16.7)	0.001
Cold intolerance	8 (26.7)	2 (6.7)	0.083
Constipation	12 (40.0)	2 (6.7)	0.006
Hoarseness of voice	7 (23.3)	4 (13.3)	0.505
Fatigue/depression	17 (56.7)	8 (26.7)	0.018
Goiter	1 (3.3)	1 (3.3)	1.000
Oedema	4 (13.3)	2 (6.7)	0.667
Menstrual status	Before treatment, n (%)	After treatment, n (%)	p-value
Regular	6 (20.7)	14 (48.3)	0.047
Irregular	15 (51.7)	7 (24.1)	
Menopause	8 (27.6)	8 (27.6)	

Table IV shows the thyroid hormone profile at the start and end of 3 months of levothyroxine treatment. Mean serum FT4 was significantly lower at baseline at 8.06 ± 3.78 pmol/L, and mean TSH was significantly higher at 32.55 ± 37.31 mIU/L, which confirms the existence of overt hypothyroidism in most patients. After 3 months of LT4 treatment, the mean FT4 level rose to 16.40 ± 3.95 pmol/L ($p<0.001$), which is a good indication of thyroid hormone replacement. Meanwhile, mean TSH decreased significantly to 2.52 ± 2.71 mIU/L ($p<0.001$), which indicates successful biochemical euthyroidism.

Table IV
Thyroid hormone profile before and after 3 months of levothyroxine therapy, *n*=30.

Hormone profile	Before treatment	After 3 months of follow-up	p-value
FT4, Mean ± SD	8.06 ± 3.78	16.40 ± 3.95	<0.001
TSH, Mean ± SD	32.55 ± 37.31	2.52 ± 2.71	<0.001

Table V shows the comparison of anthropometric parameters prior to and after treatment. Height was held constant at 153.17 ± 6.06 cm. The mean body weight decreased significantly between 65.84 ± 10.21 kg at baseline and 64.98 ± 10.10 kg at 3 months of levothyroxine treatment ($p<0.001$), which is a significant decrease that can be attributed to metabolic restoration. Correspondingly, mean BMI decreased from 27.93 ± 3.66 kg/m² to 27.56 ± 3.56 kg/m² ($p<0.001$).

Table V
Comparison of anthropometric measurements before and after 3 months of levothyroxine therapy, *n*=30.

Measurement	Before treatment, Mean ± SD	After 3 months of follow-up, Mean ± SD	p-value
Height (cm)	153.17 ± 6.06	153.17 ± 6.06	-
Weight (kg)	65.84 ± 10.21	64.98 ± 10.10	<0.001
BMI (kg/m²)	27.93 ± 3.66	27.56 ± 3.56	<0.001

Table VI demonstrated a combined overview of treatment response in major metabolic and anthropometric variables. All parameters showed statistically significant changes ($p<0.001$ or $p=0.001$) in the anticipated directions: weight and BMI reduced, FT4 rose, TSH fell, and the percentage of patients who reported subjective weight gain dropped significantly (56.7% to 16.7%). This synthesized summary validates that levothyroxine treatment results in coordinated hormonal normalization and anthropometric enhancement, which creates a definite connection between biochemical euthyroidism and a quantifiable decrease in body weight and BMI in a 3-month follow-up.

Table VI
Overall treatment response in relation to BMI.

Variable	Baseline	After treatment	Direction of change	p-value
Weight, kg	65.84 ± 10.21	64.98 ± 10.10	Decreased	<0.001
BMI, kg/m²	27.93 ± 3.66	27.56 ± 3.56	Decreased	<0.001
FT4	8.06 ± 3.78	16.40 ± 3.95	Increased	<0.001
TSH	32.55 ± 37.31	2.52 ± 2.71	Decreased	<0.001
Patients with weight gain symptoms, n (%)	17 (56.7)	5 (16.7)	Decreased	0.001

DISCUSSION

This prospective study demonstrated that levothyroxine treatment results in statistically significant decreases in body weight and BMI in patients with newly diagnosed hypothyroidism during 12 weeks, which is accompanied by the achievement of biochemical restoration of euthyroidism. These findings are consistent with Pearce et al., who reported the metabolic effects of thyroid hormone deficiency and its recovery through pharmacological treatment [12]. The female majority (96.7%) and the young adult age group seen in this study are in line with Vanderpump (2011), who reported that women are more susceptible to thyroid diseases, especially in the reproductive age group [13]. Over three-quarters of patients at baseline had a BMI above the normal range (43.3% overweight, 33.3% obese), which

highlights the close connection between hypothyroidism and impaired body weight regulation. The metabolic effects of thyroid hormones are mediated by regulation of basal metabolic rate, fatty acid oxidation, activity of the sympathetic nervous system, and adipogenesis; their deficiency is predictably associated with decreased thermogenesis and weight gain [14]. Our results have shown a high baseline prevalence of overweight and obesity, which is consistent with Malik et al., who have repeatedly associated untreated hypothyroidism with high BMI [15]. Following 3 months of levothyroxine therapy and achievement of biochemical euthyroidism, mean body weight decreased from 65.84 ± 10.21 kg to 64.98 ± 10.10 kg ($p<0.001$), and mean BMI declined from 27.93 ± 3.66 to 27.56 ± 3.56 kg/m² ($p<0.001$). These were moderate changes, but statistically significant and indicate the importance of thyroid hormone replacement in reversing at least some of the metabolic derangements. The same results were reported by Wang et al., who found that BMI decreased significantly after levothyroxine therapy in hypothyroid patients, and by a study by Abdi et al., who found that TSH normalization was linked to a small but significant weight loss [16,17]. The moderate level of BMI change was probably due to the relatively short follow-up period of 12 weeks, which implies that further therapy can produce more significant anthropometric effects in the long run. The simultaneous normalization of thyroid hormone levels means FT4 increasing to 16.40 ± 3.95 pmol/L, and TSH decreasing to 2.52 ± 2.71 mIU/L, is a strong indication of effective pharmacological euthyroidism and a strong biochemical reference point to interpret the anthropometric improvements [18]. The sharp reduction in TSH levels is a sign of the sufficiency of levothyroxine dosing, and the dose-adjustment regimen at 4-to-6-week intervals guaranteed personalized titration in line with Bekkering et al. [19]. This methodical hormonal normalization is perhaps the basis of the metabolic gains. The efficacy of the treatment was further supported by clinical symptom resolution. Weakness ($p=0.010$), weight gain complaint ($p=0.001$), constipation ($p=0.006$), fatigue/depression ($p=0.018$), and menstrual irregularity ($p=0.047$) were statistically significantly improved. The enhancement of menstrual activity is especially remarkable: hypothyroidism is a known cause of menstrual dysfunction due to its impact on sex hormone-binding globulin, prolactin, and gonadotropin-releasing hormone pulsatility [20]. The increase in regular menstrual cycles (20.7% to 48.3%) after treatment is indicative of the systemic hormonal benefits of LT4, not just thyroid activity [21]. Other symptoms like alopecia, hoarseness, oedema, and goiter did not exhibit statistically significant changes at 12 weeks, probably due to the longer time needed to resolve structural or chronic manifestations. One of the most notable results of this study was the fact that overweight or obese BMI persisted in most patients despite the restoration of euthyroidism, which is in line with Safiri et al. [22]. The residual weight burden is caused by factors such as dietary habits, physical activity levels, socioeconomic determinants, and individual metabolic variability. This observation supports the significance of a multidisciplinary approach to the treatment of hypothyroid patients beyond hormonal correction.

LIMITATIONS

The small sample size (n=30), single-centre design, and relatively short follow-up period of 12 weeks. These limitations of this study might have limited the ability to detect more significant anthropometric changes and limited the extrapolation of the results to larger hypothyroid groups. Also, the lack of a control group and data on the level of dietary intake and physical activity did not allow a thorough evaluation of confounders affecting the results of BMI.

CONCLUSION

This study demonstrated that levothyroxine treatment leads to changes in body weight, BMI, and thyroid hormone levels in newly diagnosed hypothyroid patients during a 12-week treatment. Mean BMI decreased to 27.56 kg/m^2 and mean weight to 64.98 kg, both statistically significant ($p<0.001$), and FT4 and TSH levels were normalized. The treatment showed significant improvements in weakness, weight gain, complaints, fatigue, constipation, and menstrual irregularity. Nevertheless, the fact that overweight and obesity persist in a significant percentage of patients despite the restoration of euthyroidism suggests that pharmacotherapy is not enough to fully normalize anthropometry. These results support the need to combine levothyroxine treatment with lifestyle modification interventions, such as dietary counselling and exercise, to obtain the best metabolic results. Early diagnosis and timely pharmacological treatment of hypothyroidism are essential in reducing its metabolic load, particularly in a young female population that is predominantly young.

RECOMMENDATIONS

Future studies must use larger, multi-centre prospective studies with longer follow-up of at least 6-12 months, including dietary and physical activity measures, to better assess the long-term anthropometric and metabolic outcomes of levothyroxine treatment in hypothyroid patients in a wide range of populations. Higher-level evidence would be offered by randomized controlled trials that would compare levothyroxine therapy alone with combined pharmacological and lifestyle interventions to inform clinical management strategies.

FUNDING

No funding sources.

CONFLICT OF INTEREST

None declared.

REFERENCES

1. Wojtachnio D, Osiejewska A, Bartoszewicz J, Grądzik A, Nowakowska I, Kudan M, Gorajek A, Mikut K. Hypothyroidism: clinical presentation, diagnosis, treatment. Journal of Education, Health and Sport. 2022 Aug 20;12(8):650-60.
2. Garber JR, Cobin RH, Gharib H, Hennessey JV, Klein I, Mechanick JI, Pessah-Pollack R, Singer PA, Woeber for the American Association of Clinical Endocrinologists and American Thyroid Association Taskforce on Hypothyroidism in Adults KA. Clinical practice guidelines for hypothyroidism in adults: cosponsored by the American Association of Clinical Endocrinologists and the American Thyroid Association. Thyroid. 2012 Dec;22(12):1200-35.
3. Akter S, Rahman MM, Abe SK, Sultana P. Prevalence of diabetes and prediabetes and their risk factors among Bangladeshi adults: a nationwide survey. Bulletin of the World Health Organization. 2014 Jan 10;92:204-13A.
4. Mullur R, Liu YY, Brent GA. Thyroid hormone regulation of metabolism. Physiological reviews. 2014 Apr 1.
5. Sanyal D, Raychaudhuri M. Hypothyroidism and obesity: An intriguing link. Indian journal of endocrinology and metabolism. 2016 Jul 1;20(4):554-7.
6. Misra A, Chowbey P, Makkar BM, Vikram NK, Wasir JS, Chadha D, Joshi SR, Sadikot S, Gupta R, Gulati S, Munjal YP. Consensus statement for diagnosis of obesity, abdominal obesity and the metabolic syndrome for Asian Indians and recommendations for physical activity, medical and surgical management. Japi. 2009 Feb;57(2):163-70.
7. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, Cooper DS, Kim BW, Peeters RP, Rosenthal MS, Sawka AM. Guidelines for the treatment of hypothyroidism: prepared by the American Thyroid Association task force on thyroid hormone replacement. thyroid. 2014 Dec;24(12):1670-751.
8. Knudsen N, Laurberg P, Rasmussen LB, Bulow I, Perrild H, Ovesen L, Jørgensen T. Small differences in thyroid function may be important for body mass index and the occurrence of obesity in the population. The Journal of Clinical Endocrinology & Metabolism. 2005 Jul 1;90(7):4019-24.
9. Biondi B, Wartofsky L. Treatment with thyroid hormone. Endocrine reviews. 2014 Jun;35(3):433-512.
10. Alam Khan V, Khan MA, Akhtar S. Thyroid disorders, etiology and prevalence. J Med Sci. 2002 Mar;2(2):89-94.

11. Iqbal A, Jorde R, Figenschau Y. Serum lipid levels in relation to serum thyroid-stimulating hormone and the effect of thyroxine treatment on serum lipid levels in subjects with subclinical hypothyroidism: the Tromsø Study. Journal of internal medicine. 2006 Jul;260(1):53-61.
12. Pearce SH, Brabant G, Duntas LH, Monzani F, Peeters RP, Razvi S, Wemeau JL. 2013 ETA guideline: management of subclinical hypothyroidism. European thyroid journal. 2013 Dec 1;2(4):215-28.
13. Vanderpump MP. The epidemiology of thyroid disease. British medical bulletin. 2011 Sep 1;99(1).
14. McLeod DS, Cooper DS. The incidence and prevalence of thyroid autoimmunity. Endocrine. 2012 Oct;42(2):252-65.
15. Malik S, Rehman S, Naz F, Rehman N, Syed Z. Exploring the link between obesity and hypothyroidism. Journal of Ayub Medical College Abbottabad. 2023 Dec 22;35(4):608-11.
16. Wang X, Liu H, Chen J, Huang Y, Li L, Rampersad S, Qu S. Metabolic characteristics in obese patients complicated by mild thyroid hormone deficiency. Hormone and Metabolic Research. 2016 May;48(05):331-7.
17. Abdi H, Kazemian E, Gharibzadeh S, Amouzegar A, Mehran L, Tohidi M, Rashvandi Z, Azizi F. Association between thyroid function and body mass index: a 10-year follow-up. Annals of Nutrition and Metabolism. 2017 Jun 16;70(4):338-45.
18. Brenta G, Vaisman M, Sgarbi JA, Bergoglio LM, Andrada NC, Bravo PP, Orlandi AM, Graf H. Clinical practice guidelines for the management of hypothyroidism. Arquivos Brasileiros de Endocrinologia & Metabologia. 2013;57:265-91.
19. Bekkering GE, Agoritsas T, Lytvyn L, Heen AF, Feller M, Moutzouri E, Abdulazeem H, Aertgeerts B, Beecher D, Brito JP, Farhoumand PD. Thyroid hormones treatment for subclinical hypothyroidism: a clinical practice guideline. Bmj. 2019 May 14;365.
20. Krassas GE, Poppe K, Glinoer D. Thyroid function and human reproductive health. Endocrine reviews. 2010 Oct 1;31(5):702-55.
21. Vargas-Uricoechea H, Wartofsky L. LT4/LT3 combination therapy vs. monotherapy with LT4 for persistent symptoms of hypothyroidism: A systematic review. International Journal of Molecular Sciences. 2024 Aug 25;25(17):9218.
22. Safiri S, Sorkhabi AD, Aletaha R, Hamidi S, Asghari KM, Sarkesh A, Alamdary SJ, Jolfayi AG, Nejadghaderi SA, Fazlollahi A, Mohammadinasab R. Obesity: global epidemiology, trends, risk factors, and clinical aspects. International Journal of Aging. 2024 Dec 1;2(1):e19-e19.