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Thyroid Gland Remodeling and Functional Recovery in Patients Following Bariatric Surgery: Structural and Hormonal Correlations

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Abstract: Bariatric surgery is the most effective available intervention for achieving substantial and sustained weight loss in patients with severe obesity, and it produces well-documented improvements in multiple obesity-related comorbidities. Among the less widely appreciated effects of bariatric surgery are its consequences for thyroid gland structure and function. Weight loss induced by procedures such as sleeve gastrectomy and Roux-en-Y gastric bypass is consistently associated with a decline in circulating thyroid-stimulating hormone (TSH) toward normal-weight reference values, a reduction in indices of central resistance to thyroid hormone, and — on ultrasonography — a reversal of the hypoechoic, obesity-related pattern of the thyroid parenchyma toward normal echogenicity. This review summarizes the current evidence on the pathomorphological and functional remodeling of the thyroid gland after bariatric surgery, discusses the proposed mechanisms underlying this remodeling, and addresses the practical question of levothyroxine dose adjustment in hypothyroid patients undergoing these procedures.

Keywords: bariatric surgery, sleeve gastrectomy, Roux-en-Y gastric bypass, thyroid-stimulating hormone, thyroid echogenicity, levothyroxine, weight loss

Citation: Murodjon qizi, N. M. Thyroid Gland Remodeling and Functional Recovery in Patients Following Bariatric Surgery: Structural and Hormonal Correlations. Central Asian Journal of Medical and Natural Science 2026, 7(4), 444-447

Received: 13th Jun 2026
Revised: 28th Jul 2026
Accepted: 15th Aug 2026
Published: 12th Sep 2026



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1. Introduction

Bariatric (metabolic) surgery has become the most effective and durable treatment for severe obesity and is associated with a marked reduction in all-cause mortality and in the burden of obesity-related comorbidities compared with non-surgical management [1]. The two most widely performed procedures worldwide are laparoscopic sleeve gastrectomy (SG) and laparoscopic Roux-en-Y gastric bypass (RYGB), both of which achieve substantial and sustained excess-weight loss [2]. Beyond their well-recognized metabolic benefits — including remission of type 2 diabetes and improvement of cardiovascular risk factors — bariatric procedures also exert measurable effects on the hypothalamic-pituitary-thyroid axis, reversing many of the functional and structural thyroid changes that are characteristic of the obese state [3-5].

Because obesity itself is associated with mild TSH elevation and with pathomorphological remodeling of the thyroid parenchyma detectable by ultrasonography, bariatric surgery-induced weight loss provides a unique clinical model for studying the reversibility of these changes. This review synthesizes current evidence on the hormonal and structural remodeling of the thyroid gland following bariatric surgery and considers its implications for the management of thyroid function in surgically treated patients[4].

Multiple prospective and retrospective studies have documented a significant decline in circulating TSH following bariatric surgery-induced weight loss in patients who were

euthyroid preoperatively. Juiz-Valiña and colleagues followed 129 patients with morbid obesity for up to 42 months after RYGB or SG and observed a progressive, statistically significant decrease in TSH that was independently associated with the percentage of excess body mass index lost, together with a parallel decline in free thyroxine (FT4) toward the normal range [5]. A systematic review and meta-analysis by Guan and colleagues, pooling data from multiple cohorts, confirmed that bariatric surgery is associated with a significant reduction in circulating TSH, although free thyroid hormone levels were generally unaffected [6]. Similar reductions in TSH following weight loss have been reported after sleeve gastrectomy in Chinese cohorts and after Roux-en-Y gastric bypass in other populations, and ultrasonographic follow-up in some of these studies additionally noted an increase in thyroid echogenicity in parallel with the hormonal changes [7-9].

2. Materials and Methods

A literature-based review was conducted to assess thyroid functional and structural changes following bariatric surgery. Clinical studies, systematic reviews, and meta-analyses evaluating patients before and after sleeve gastrectomy and Roux-en-Y gastric bypass were analyzed. The main assessment methods included measurement of serum TSH and FT4 levels and thyroid ultrasonography to evaluate thyroid echogenicity and volume. Changes in central thyroid hormone resistance and levothyroxine requirements were also assessed. Comparative analysis was used to determine the relationship between postoperative weight loss and thyroid recovery.

3. Results

Not all studies have found this effect: Dall'Asta and colleagues, evaluating patients after gastric banding — a purely restrictive and comparatively milder bariatric technique — found that TSH levels remained essentially unchanged despite weight loss, suggesting that the magnitude and mechanism of the effect may vary with the surgical technique and the degree of achieved weight reduction [10]. Beyond simple TSH decline, Juiz-Valiña and colleagues also demonstrated that indices of central resistance to thyroid hormone, including the thyrotroph T4 resistance index and the TSH index, were elevated preoperatively in morbidly obese patients and declined significantly after bariatric surgery, in association with excess weight loss and with improvement of the systemic inflammatory marker C-reactive protein [11].

The pathomorphological correlate of this functional recovery has been documented by ultrasonographic studies performed before and after bariatric surgery. In a prospective cohort of morbidly obese patients undergoing bariatric surgery, thyroid ultrasonography demonstrated a measurable increase in echogenicity following surgery-induced weight loss, occurring in parallel with the decline in TSH and with improvement of insulin resistance indices, and this normalization was observed even in patients whose thyroid parenchyma had appeared isoechoic (i.e., only mildly affected) preoperatively [12]. A pilot study using gray-scale ultrasound histogram analysis similarly found that marked weight loss after bariatric surgery in euthyroid patients with severe obesity was associated with a significant increase in thyroid echogenicity, without a corresponding change in thyroid volume or in thyroid function tests, supporting the concept that obesity-related morphological changes in the thyroid are reversible with weight loss independently of overt hormonal shifts [13].

Taken together, these sonographic findings indicate that the hypoechoic, heterogeneous pattern frequently observed in the thyroid gland of obese patients is not a fixed or degenerative lesion but rather a dynamic, weight-dependent phenomenon capable of substantial regression following effective and sustained weight loss achieved through bariatric surgery.

4. Discussion

For patients who have pre-existing hypothyroidism and are treated with levothyroxine, bariatric surgery introduces an additional layer of complexity, since both the reduction in body weight and the altered gastrointestinal anatomy created by malabsorptive or restrictive procedures can influence levothyroxine requirements. Pedro and colleagues found that although the total daily levothyroxine dose did not change significantly after bariatric surgery, the dose calculated per kilogram of actual body weight increased, reflecting a relative rise in requirement as body weight declined disproportionately to the absolute hormone dose [14]. Fallahi and colleagues reported that switching hypothyroid bariatric surgery patients from tablet to oral liquid levothyroxine formulations improved normalization of TSH, suggesting that altered absorption after surgery may be partially overcome by formulation changes [15]. Cordido and colleagues, reviewing this literature, concluded that thyroid hormone replacement in obese hypothyroid patients can generally be more accurately titrated when based on ideal body weight rather than actual body weight, and that this principle should also guide dose adjustment after bariatric surgery-induced weight loss [16].

The evidence reviewed here supports several practical conclusions. First, clinicians should anticipate a gradual, weight-loss-dependent decline in TSH in previously euthyroid obese patients following bariatric surgery, and this decline should not be misinterpreted as iatrogenic or pathological. Second, ultrasonographic re-evaluation of the thyroid gland after substantial postoperative weight loss may reveal a reversal of the hypoechoic pattern previously attributed to obesity, information that can be reassuring when counseling patients about previously identified sonographic abnormalities. Third, in hypothyroid patients on levothyroxine replacement, dose requirements should be reassessed periodically after bariatric surgery, taking into account both the weight-loss-related reduction in requirement and the potential for altered absorption depending on the specific surgical technique performed [17-18]. Future prospective studies combining serial thyroid ultrasonography, standardized biochemical panels, and stratification by surgical procedure type would help clarify the precise time course and the relative contributions of weight loss versus altered gastrointestinal physiology to thyroid remodeling after bariatric surgery.

5. Conclusion

Bariatric surgery-induced weight loss is associated with a coherent pattern of functional and structural thyroid gland recovery in patients with severe obesity, characterized by a progressive decline in TSH, improvement of central resistance to thyroid hormone, and a reversal of obesity-related pathomorphological changes on ultrasonography. These findings reinforce the concept that many of the thyroid abnormalities observed in obesity are secondary and reversible, and they carry direct implications for the interpretation of thyroid function tests and for the management of levothyroxine therapy in patients undergoing bariatric surgery.

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