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Subclinical Hypothyroidism Post-Bariatric Surgery: A Reversible Case of Iodine Deficiency Without Autoimmune Thyroid Disease

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ABSTRACT: Subclinical hypothyroidism is a common finding in clinical practice, often prompting initiation of levothyroxine therapy. However, not all cases of elevated thyroid-stimulating hormone (TSH) reflect permanent thyroid dysfunction. This report discusses a 39-year-old woman with a history of Roux-en-Y gastric bypass surgery who presented with elevated TSH in the absence of thyroid autoantibodies or classic hypothyroid symptoms. Laboratory and clinical findings suggested iodine deficiency as the underlying cause. Her TSH normalized following potassium iodide supplementation. This case underscores the need for a nuanced approach to managing elevated TSH, particularly in post-bariatric surgery patients where iodine deficiency may be the etiology of subclinical hypothyroidism.

KEYWORDS: Bariatric Surgery, Iodine deficiency, Subclinical hypothyroidism, TSH.

INTRODUCTION

Subclinical hypothyroidism, characterized by elevated serum thyroid-stimulating hormone (TSH) with normal free thyroxine (T4), is frequently encountered in endocrine practice [1]. While autoimmune (Hashimoto's) thyroiditis is the most common etiology in iodine-sufficient populations, alternative causes such as iodine deficiency must be considered [2]. Patients who have undergone bariatric surgery are at increased risk for micronutrient deficiencies due to reduced dietary intake and altered absorption especially if supplements are not taken [3]. Iodine deficiency is not particularly likely after bariatric surgery as it is usually sufficiently absorbed by the other parts of the gastrointestinal tract [4]. Misinterpretation of elevated TSH in after bariatric surgery may lead to unnecessary lifelong thyroid hormone replacement. This case highlights the importance of evaluating iodine status before diagnosing hypothyroidism, especially when thyroid peroxidase antibodies (TPO-Ab) are absent and symptoms are nonspecific or lacking.

CASE PRESENTATION

A 39-year-old woman with a history of Roux-en-Y gastric bypass surgery 12 years ago, presented for a follow-up of an elevated TSH discovered during routine testing. Her past medical history was notable for iron deficiency anemia, recurrent migraines, vitamin D deficiency, and persistent tinnitus. She also reported chronic fatigue, loss of lateral eyebrows, and blue discoloration of the toes. Of note, she denied hallmark symptoms of hypothyroidism such as dry skin, weight gain, cold intolerance, or significant constipation prior to treatment. On physical examination, vital signs were normal and there was no goiter.

Laboratory testing showed an elevated TSH (6.1 mIU/L) with a normal free T4 (0.84 ng/dL), normal free T3 (3.6 pmol/L) and negative TPO-Ab. There was no personal or significant family history of autoimmune thyroid disease. Given her surgical history and the absence of autoimmune markers, iodine deficiency was suspected as the etiology of the mildly elevated TSH.

She was started empirically on potassium iodide 130 mg daily. After three months of treatment, her TSH improved significantly, decreasing to 3.06 mIU/L with a normal free T4 (0.89 ng/dL) and free T3 (3.96 pmol/L). Laboratory investigations revealed a persistent iron deficiency (ferritin 8.3 ng/mL), stable hemoglobin (13.2 g/dL), and mildly low vitamin D (24 ng/mL). Vitamin B12 and folic acid levels were within normal ranges. Neurologic workup of her migraines showed improvement following Botox therapy, and her persistent tinnitus had been deemed non-neurologic in origin. Throughout treatment, she experienced new-onset

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constipation managed with glycerin suppositories, but did not report any worsening fatigue, weight gain, or depression. Despite multiple overlapping medical concerns and frustration with fragmented care, the patient remained actively engaged in treatment and follow-up schedules.

DISCUSSION

Subclinical hypothyroidism affects 4.3% of the populations in the USA and is more common in females than males [1]. The majority of cases are caused by autoimmune thyroiditis. Patients with biochemical evidence of subclinical hypothyroidism may experience one of the three outcomes: reversion to a euthyroid state with normalization of TSH levels, persistent subclinical hypothyroidism, or progression to overt hypothyroidism if free T4 levels decrease below the normal range [5]. The risk of progression to overt hypothyroidism is around 2.6-4.3% per year. Certain factors increase this risk including a positive TPO-Ab, TSH higher than 10 mIU/L, presence of goiter, history of autoimmune diseases and prior radiation exposure [6]. Our patient with no goiter on examination, a negative TPO-Ab, and TSH less than 10 mIU/L, is at lower risk to progression to overt hypothyroidism thus prompting us to consider causes of subclinical hypothyroidism other than autoimmune thyroiditis. Roux-en-Y gastric bypass significantly alters gastrointestinal anatomy and physiology, impairing the absorption of multiple micronutrients including iodine. While iodine is primarily absorbed in the stomach and upper small intestine, reduced gastric acid and bypassed segments limit bioavailability. Furthermore, post-operative dietary restrictions and avoidance of iodized salt compound the problem [7]. The American Association of Clinical Endocrinology and other expert bodies recommend routine screening and supplementation of micronutrients following bariatric surgery, but iodine is often overlooked [8]. In fact, most studies in this context suggest that dietary absorption of iodine is not significantly altered by bariatric surgery and prevalence of subclinical hypothyroidism reduces after Roux-en-Y gastric bypass surgery [4, 9]. This in turn has led to the suggestion of not including iodine in the supplements recommended after bariatric surgery [10]. Data from recent literature supports the use levothyroxine to treat subclinical hypothyroidism in symptomatic patients, presence of goiter, TSH more than 10 mIU/L, positive TPO-Ab, and in pregnant ladies [11]. Our case did not fall under those categories and thus was presumed to have iodine deficiency from bariatric surgery. In support of this, the significant response to potassium iodide therapy eliminates the need for levothyroxine therapy with associated potential cost and complications. This case emphasizes the importance of context in interpreting thyroid function tests. Elevated TSH should not be viewed in isolation, and clinicians must assess potential contributing factors, including iodine deficiency. This is particularly important in populations at risk for micronutrient deficiencies, such as patients with a history of bariatric surgery. Empirical iodine supplementation in such cases may serve both diagnostic and therapeutic purposes.

CONCLUSIONS

In patients presenting with elevated TSH, especially those without TPO-Ab or overt symptoms, clinicians must evaluate for reversible causes such as iodine deficiency. This is particularly relevant in individuals with altered gastrointestinal anatomy, such as those with a history of bariatric surgery. Empirical iodine supplementation can normalize TSH and prevent unnecessary levothyroxine therapy.

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