Thyroid dysfunction in a patient with morbid obesity

M. Usman Chaudhry, MD, FACE

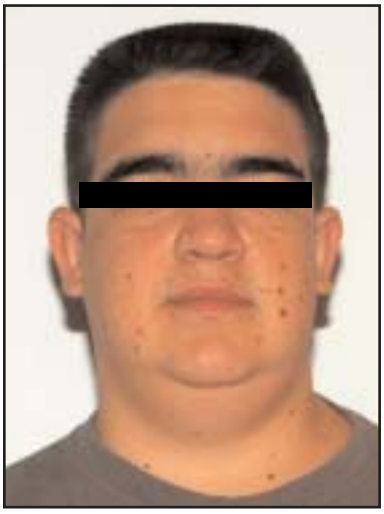

Figure 1. Hypothyroid patient with weight gain.

Tony's case

Tony, 22, was referred for an endocrine opinion on weight gain (Table 1). He started a dedicated dietary and intensive exercise program eight months before presentation for a sudden 75-pound weight gain from baseline of 240 pounds (Figure 1). He successfully lost 80 pounds by aggressive weight control measures, but noticed a sudden syncopal episode while on a treadmill six weeks before presentation. The emergency medical response team was called in, and found his sugar and blood pressure to be normal. He had no recurrence of such symptoms afterwards.

Retrospectively he noticed a new, progressive onset of moderate fatigue, muscle weakness, cold intolerance, and dry skin for the last six weeks. He denied any diplopia, headaches, galactorrhea, easy bruising, abnormal stretch marks, or sexual dysfunction.

He has a family history of hypothyroidism in his father and two of his paternal aunts. Tony doesn't drink alcohol or smoke cigarettes.

Table 1
A rundown on Tony

Upon presentation

- Morbidly obese (240 lbs on a 5'6" frame)
- Body mass index of 38.5-kg/m^2
- Blood pressure of 92/60 mmHg
- Heart rate of 45 beats per minute
- Was alert, awake, and ambulatory
- Pertinent exam was negative for nystagmus, diplopia, or visual fields defects by confrontation method
- No discrete nodules in thyroid (which was difficult to palpate due to obesity)
- Acanthosis nigricans on neck
- Heart and breath sounds normal
- Chest exam showed bilateral lipomastia without galactorrhea
- Dry skin on leg, absence of edema, and deep tendon reflexes at ankles had normal relaxation phase

Laboratory results

- Thyroid stimulating hormone: > 300 mIU/L (normal 0.4-5.50)
- Free thyroxine: 4.3 pmol/L (normal 11-23)
- Hemoglobin: 133 g/L (normal 124-164)

His past medical issues include laser eye surgery for severely myopic eyesight. He is allergic to sulpha drugs, and he was not on any medication at presentation.

Discussion

Primary hypothyroidism is caused by a decreased production of thyroid hormones by the thyroid gland. It is a relatively common disease in both iodine-deficient and iodine-sufficient populations. It usually arises from primary thyroid disease, but in rare cases it is due to hypothalamic-pituitary disease (secondary hypothyroidism) or generalized tissue resistance to thyroid hormone (Table 2).

Myxedema coma

Myxedema coma is severe hypothyroidism when complicated by trauma, infection, cold exposure, or inadvertent administration of hypnotics or narcotics. The diagnosis should be suspected in comatose patients who are hypothermic, hypercapnic, hyponatremic, and hypotensive.

What is the diagnosis?

The diagnosis of primary hypothyroidism is confirmed by a reduced free-thyroxine (T4) level and elevated thyroid stimulating hormone (TSH) level. Subclinical hypothyroidism is diagnosed by the demonstration of elevated TSH levels with normal free T4 levels and no clinical symptoms. The TSH level may be reduced, normal, or even slightly elevated in secondary hypothyroidism resulting from hypothalamic-pituitary dysfunction. Evaluation of other pituitary hormone levels (prolactin, growth hormone, cortisol, and gonadotropins) and imaging studies (computerized tomography or magnetic resonance imaging of the brain) may need to be considered. A positive antithyroid antibodies test may indicate the presence of Hashimoto's disease.

What is the treatment?

Complete discussion on this subject is beyond the scope of this case report. There is no disagreement in the treatment of primary clinical hypothyroidism with levothyroxine. The American Association of Clinical Endocrinologists recommends a standard

Dr. Chaudhry is a staff endocrinologist and internist, department of internal medicine, Moncton Hospital, and Dalhousie University affiliate, Moncton, New Brunswick.

Table 2

Clinical signs and symptoms of hypothyroidism

- Fatigue
- Lethargy
- Cold intolerance
- Sleepiness
- Mental impairment
- Arthralgia
- Depression
- Decreased appetite
- Hoarseness
- Paresthesia
- Decreased perspiration
- Dry, scaly skin
- Dry hair
- Hair loss
- Generalized edema
- Pitting edema of lower extremities
- Weight gain
- Constipation
- Menorrhagia
- Infertility

stable brand of levothyroxine preparation and to avoid desiccated animal thyroid products or triiodothyronine (T3). Mean replacement dosage should be approximately 0.75 mcg/lb/day. Initial starting dosage may vary from 12.5 mcg/day to full replacement dose depending on cardiac status. Therapy should be titrated after six weeks following any start or change in levothyroxine brand or dose. Thyroid hormone absorption may be affected by various factors (Table 3).

Subclinical hypothyroidism should be approached as follows: An elevated serum thyrotropin level should be confirmed. If the serum level of free thyroxine is low, then the patient has overt hypothyroidism and should be treated with thyroxine. Testing for antibodies against thyroperoxidase and obtaining a lipid profile are important in subsequent decision making. If the results of these tests are not abnormal, there are no symptoms or goiter, and the serum thyrotropin level is less than 10 mU/L, therapy is optional. Women who are pregnant or who have ovulatory dysfunction and infertility should be treated regardless of the presence or absence of symptoms, antibody status, or serum lipid levels.

Tony's case resolved

This patient had severe primary hypothyroidism resulting in massive weight gain over a short period of time along with typical clinical symptoms. He absolutely needed Synthroid® (levothyroxine sodium) replacement. To avoid any unlikely possibility of pre-existing adrenal decompensation due to hypoadrenalism, an immediate cosyntropin test was performed. It showed normal peak response of cortisol 799 nmol/L (normal > 500) after 60 minutes of cosyntropin injection. His thyroid peroxidase antibodies were high at 396 IU/mL (normal < 35), depicting primary thyroid failure due to Hashimoto's disease.

Table 3

Factors possibly affecting thyroid hormone absorption

- Cholestyramine
- Sucralfate
- Malabsorption
- Ferrous sulfate
- Calcium

He was started on Synthroid 175 mcg/day (0.75 mcg/lb/day). He was advised to avoid any intense exercise as dictated by his weight loss program for the next four weeks. His thyroid functions at four weeks showed biochemical and clinical euthyroidism; TSH 2.55 mU/L and free T4: 18.8 pmol/L.

Given his risk factors, including morbid obesity and presence of acanthosis nigricans, his fasting blood sugar at second followup was confirmed to be normal at 4.8 mmol/L. His thyroid functions remain stable at his seven-month followup on the aforementioned dosage of Synthroid. His fasting lipid profile was normal as was his liver function tests and serum creatinine. He doesn't have sleep apnea.

Aggressive lifestyle modification after normalization of his thyroid functions was advised. He declined weight loss therapy with sibutramine or orlistat and continues to do well. CME

References

1. Guha B, Krishnaswamy G, Peiris A: The Diagnosis and Management of Hypothyroidism. South Med J 2002; 95(5):475-80.
2. Mandel SJ, Brent GA, Larsen PR: Levothyroxine Therapy in Patients with Thyroid Disease. Ann Intern Med, Sep 1993; 119: 492-502.
3. Cooper DS: Subclinical Hypothyroidism. NEJM 2001; 345(4):260-5.
4. American Association of Clinical Endocrinologists: Medical guidelines for clinical practice for the evaluation and treatment of hyperthyroidism and hypothyroidism. Endocrine Practice 2002; 8(6):458-67.