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Hypothyroidism

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Hypothyroidism

INTRODUCTION

Hypothyroidism, resulting primarily from autoimmune destruction of the thyroid gland, is among the most common of endocrine diseases, affecting 1% to 1.5% of all women and up to 0.5% of men.¹ Several community-based surveys show an increasing prevalence with age in both sexes, reaching over 12% among women over the age of 70.² Many more individuals of both sexes develop subclinical disease, and some evidence of autoimmune thyroiditis can be found at autopsy in 45% of women and 20% of men in the United States.³

The diagnosis of hypothyroidism generally is straightforward. Treatment with oral thyroid hormone replacement is simple and well tolerated. The widespread availability of sensitive thyroid-stimulating hormone (TSH) assays and the broad range of available strengths of levothyroxine have made accurate dose titration possible. However, there remains controversy about the dose of thyroid hormone required for replacement therapy, the use of synthetic thyroxine (T_4) alone or in combination with triiodothyronine (T_3), the need for dose adjustment during pregnancy or intercurrent illness, and the treatment of patients with subclinical hypothyroidism.³

Hypothyroidism most often is caused by decreased production of thyroid hormone resulting from disease of the thyroid gland (ie, *primary hypothyroidism*). Far less common is *secondary hypothyroidism*, or *central hypothyroidism*, caused by reduced pituitary TSH secretion. *Transient hypothyroidism*, seen most often as postpartum thyroiditis, is increasingly recognized and may be associated with adverse maternal and fetal outcomes, including postpartum depression.⁴

PRIMARY HYPOTHYROIDISM

Primary hypothyroidism may be caused by damage to or destruction of thyroid tissue or by interference with thyroid hormone synthesis (**Table 1**). By far the most common cause is autoimmune thyroiditis, commonly known as *Hashimoto's disease*.

CASE PRESENTATION

Initial Presentation

A 48-year-old woman is referred to an endocrinologist for evaluation of a goiter.

History

The patient was first noted to have a swelling in her neck 5 years ago during a routine physical examination. At that time, the patient had no compressive symptoms and no clinical features suggesting thyroid disease. The goiter has gradually enlarged and is now noticeable to the patient. The right side is more prominent than the left, and a nodule was felt on the right side by the patient's primary physician. The patient has noticed progressive fatigue, mild depression, dry skin, weight gain of 10 lb over 4 years, and cold intolerance. She admits to worsening of her lifelong tendency to constipation. Her menstrual periods have become heavy and prolonged, and she thinks she may be entering menopause. Family history is significant for hypothyroidism in the patient's mother and a maternal aunt and for Graves' disease in a cousin.

Physical Examination

The patient is overweight, with some coarsening of her facial features. Her skin is sallow, cool, and dry, with fissuring on the fingers. A small, firm goiter is noted, which is approximately twice normal size and irregular in outline. A 1.5-cm nodule is palpable in the left lobe of the thyroid, which is mobile on swallowing.

Laboratory Evaluation

Thyroid function testing produces the following results:

- TSH, 42.8 $\mu\text{U}/\text{mL}$ (normal, 0.3 to 5.0 $\mu\text{U}/\text{mL}$)
- Free T_4 (FT_4), 0.6 ng/dL (normal, 0.8 to 1.8 ng/dL)
- Thyroid peroxidase antibody (TPOAb), 358 U/L (normal, less than 10 U/L)

- **Based on this patient's clinical presentation and laboratory findings, what is the most likely diagnosis?**

CLINICAL FEATURES OF HASHIMOTO'S DISEASE

This patient exhibits classic symptoms and signs of hypothyroidism (**Table 2**). The finding of a goiter argues against a central cause of hypothyroidism, and the elevated TSH largely excludes hypothalamic or pituitary causes. Although a positive TPOAb finding is common among women in this patient's age-group, the high titer along with the presence of a goiter and a family history of autoimmune thyroid disease argue strongly for Hashimoto's disease in this patient.