

Thyroiditis: Review Questions

Jennifer Pedersen-White, DO

QUESTIONS

Choose the single best answer for each question.

Questions 1 to 5 refer to the following case.

A 32-year-old woman presents to her primary care physician with a 1-month history of heat intolerance, fatigue, and palpitations. She delivered a healthy baby girl by uncomplicated vaginal delivery 12 weeks prior to this visit. She is no longer breastfeeding. The patient's past medical history is significant for type 1 diabetes diagnosed at age 7 years, which has been well controlled. She has no personal or family history of thyroid disorder. She denies the use of over-the-counter herbal supplements or exogenous thyroid hormone replacement or having received iodinated contrast in the past year. On physical examination, the patient is afebrile with a heart rate of 100 bpm and a blood pressure of 146/88 mm Hg. The patient appears clinically euthyroid. There is no proptosis or lid lag. The thyroid is normal in size, nontender to palpation, and without appreciable masses. Cardiac examination reveals tachycardia and a regular rhythm. The skin is diffusely warm to touch and is nondiaphoretic. Deep tendon reflexes are 3+/4 with assessment of biceps and patellar tendon reflexes. Laboratory testing reveals a suppressed thyroid-stimulating hormone (TSH) level of 0.19 μ IU/mL (normal, 0.35–5.5 μ IU/mL) and an elevated free thyroxine (FT_4) level of 1.98 ng/dL (normal, 0.61–1.76 ng/dL). Medical records show that the patient's TSH has been within normal limits with routine laboratory evaluation over the past 3 years.

1. Which of the following tests would be most useful in determining a diagnosis in this patient?

- (A) Repeat TSH and FT_4
- (B) Thyroid scan and radioactive iodine uptake (RAIU)

- (C) Thyroid ultrasound
- (D) TSH receptor antibody assay

2. An ^{123}I thyroid scan and uptake reveals a grossly normal thyroid contour without evidence of hot or cold nodules. RAIU at 4 hours is less than 1% (normal, 4%–15%). TSH receptor antibody assay is negative, but thyroid peroxidase (TPO) antibody assay returns markedly elevated. What is the patient's most likely diagnosis?

- (A) Graves' disease
- (B) Postpartum thyroiditis
- (C) Toxic multinodular goiter (TMG)
- (D) Transient gestational hyperthyroidism

3. What is the next step in the management of this patient?

- (A) ^{131}I thyroid ablation
- (B) Initiate β -blocker therapy
- (C) Initiate levothyroxine therapy
- (D) Initiate thionamide therapy

4. What is the relationship of type 1 diabetes to the patient's current thyroid dysfunction?

- (A) There is no relationship between type 1 diabetes and thyroid dysfunction
- (B) Type 1 diabetes has a protective effect against thyroid dysfunction
- (C) Type 1 diabetics have an increased prevalence of thyroid dysfunction
- (D) Uncontrolled type 1 diabetes contributes to abnormal thyroid function tests

5. Which of the following is most likely to occur in this patient in the future?

- (A) No recurrence of thyroid dysfunction
- (B) Low likelihood of recurrence after each subsequent pregnancy
- (C) Permanent hyperthyroidism is likely to develop
- (D) Permanent hypothyroidism is likely to develop

Dr. Pedersen-White is an assistant professor, Section of Endocrinology, Department of Medicine, Medical College of Georgia, Augusta, GA.

6. A 74-year-old man with a past medical history of recurrent atrial fibrillation presents to his primary care physician with a 3-month history of fatigue, weakness, occasional palpitations, hand tremors, and a 10-lb weight loss. Atrial fibrillation has been managed with daily amiodarone therapy for 3 years. He has no personal or family history of thyroid disorder. On physical examination, the patient is afebrile with a heart rate of 104 bpm and blood pressure of 132/78 mm Hg. On examination, the thyroid is normal in size and nontender to palpation, with no thyroid nodules. The heart rate is tachycardic with a regular rhythm. An electrocardiogram reveals sinus tachycardia. Deep tendon reflexes are within normal limits. Laboratory tests reveal a TSH level less than 0.01 $\mu\text{IU/mL}$ and an FT_4 level of 3.21 ng/dL, and assays for TSH receptor and TPO antibodies are negative. Thyroid scan and RAIU reveal patchy tracer uptake within the thyroid and an uptake of less than 1% at 6 hours. Thyroid ultrasound reveals normal thyroid size with normal tissue echogenicity and blood flow without nodules or masses. What is this patient's most likely diagnosis?

- (A) Acute suppurative thyroiditis
- (B) Amiodarone-induced thyrotoxicosis (AIT)
- (C) Graves' disease
- (D) TMG

ANSWERS AND EXPLANATIONS

1. (B) **Thyroid scan and RAIU.** If the patient is not breastfeeding, thyroid scan and RAIU test can safely be performed to help elucidate the diagnosis. RAIU is elevated with hyperthyroidism due to Graves' disease, solitary autonomous nodule, or TMG. RAIU is low with hyperthyroidism due to thyroiditis.¹ If the patient has been exposed to exogenous iodine (eg, radiocontrast dye injection), RAIU will also be low, even if the patient has Graves' disease; it can take up to 3 months to obtain an accurate RAIU result in such cases. Although a thyroid ultrasound would identify possible gland enlargement and/or thyroid nodules, it would not provide information about glandular activity/function. In addition, the presence of TSH receptor antibodies may suggest Graves' disease; however, this provides no information about glandular activity/function. Retesting of TSH and FT_4 levels would confirm abnormal results but would not provide information regarding the etiology of thyrotoxicosis in this patient.

2. (B) **Postpartum thyroiditis.** Both Graves' disease and

postpartum thyroiditis can cause hyperthyroidism in the postpartum period. A suppressed TSH with elevated total T_4 , FT_4 , and free triiodothyronine levels may be seen in both disorders. The presence of TPO antibodies with a low 4-hour RAIU is consistent with thyroiditis in this patient. The lack of exophthalmos, significant thyroid enlargement, and TSH receptor antibodies makes Graves' disease unlikely in this case. Up to 50% of women who develop postpartum thyroiditis have high serum antibodies to TPO.² Postpartum thyroiditis is an autoimmune-mediated inflammation of the thyroid gland that occurs in the first year after delivery. The mean prevalence of postpartum thyroiditis in iodine-sufficient areas is 5% to 7%.³ Considered to be a transient form of Hashimoto's thyroiditis, postpartum thyroiditis occurs as the relatively immune-tolerant state of pregnancy normalizes after delivery. It occurs as either transient hyperthyroidism alone, transient hypothyroidism alone, or transient hyperthyroidism followed by transient hypothyroidism. Most women are euthyroid at 1-year postpartum. The most common presentation of postpartum thyroiditis is hypothyroidism without preceding hyperthyroidism, occurring in approximately 40% of patients. Approximately 20% to 30% of women with postpartum thyroiditis have hyperthyroidism alone. The remaining cases (25%) present with characteristic hyperthyroidism followed by hypothyroidism.^{2,4} The hyperthyroid phase of postpartum thyroiditis most commonly presents within 1 to 4 months of delivery and lasts from 2 to 8 weeks,² whereas the hypothyroid phase occurs between 2 and 12 months postpartum and lasts from 2 weeks to several months.^{2,4} In Graves' disease and TMG, RAIU would be elevated, not suppressed. Additionally, thyroid scan would reveal diffuse thyroid uptake with Graves' disease and focal uptake with TMG. Infrequently, some women with postpartum thyroiditis will be TSH receptor antibody-positive. However, the prevalence of postpartum thyroiditis is 20 times that of Graves' disease, making postpartum thyroiditis the more likely cause of hyperthyroidism in this population.⁵ Transient gestational hyperthyroidism can be seen in early pregnancy and occurs when high serum concentrations of human chorionic gonadotropin act as a weak stimulator of the TSH receptor.

3. (B) **Initiate β -blocker therapy.** The combination of low serum TSH, elevated FT_4 , positive TPO antibodies, and low RAIU is consistent with the hyperthyroid phase of postpartum thyroiditis. Most women with

postpartum thyroiditis do not require treatment. Because the hyperthyroid phase of postpartum thyroiditis is caused by the release of preformed thyroid hormone from a destructive inflammation of the thyroid, thionamides, which block thyroid hormone synthesis, would not be an effective treatment. ¹³¹I ablation would also be ineffective because radioiodine would not be incorporated into the thyroid gland because of low uptake. However, β -blockers can be given to alleviate symptoms of hyperthyroidism that are caused by increased β -adrenergic tone (eg, palpitations, anxiety, heat intolerance, tachycardia).⁶ If there are no contraindications to their use, β -blockers can be started in most patients at the time hyperthyroidism is diagnosed, even prior to RAIU. As thyroid inflammation resolves, a transient period of hypothyroidism can be seen as thyroid follicles regenerate. If this condition occurs, symptomatic hypothyroidism can be treated with daily levothyroxine.

4. **(C) Type 1 diabetics have an increased prevalence of thyroid dysfunction.** Individuals with type 1 diabetes have a three- to fourfold increased prevalence of postpartum thyroiditis.^{7,8} Other autoimmune diseases are thought to increase the risk of thyroid dysfunction as well. There is no consensus as to the value of screening for postpartum thyroiditis; however, some physicians screen patients with autoimmune diseases, such as lupus, type 1 diabetes, and rheumatoid arthritis. There is no evidence to suggest that there is an association between uncontrolled diabetes and abnormal postpartum thyroid function tests.
5. **(D) Permanent hypothyroidism is likely to develop.** Seventy percent of women with a previous episode of postpartum thyroiditis develop recurrence with subsequent pregnancies.⁹ By convention, hypothyroidism that persists for more than 1 year postpartum is not considered postpartum thyroiditis. Although most women with postpartum thyroiditis become euthyroid within 1 year, women who present with higher titers of TPO antibodies are at increased risk of permanent hypothyroidism.¹⁰ It has been suggested that women with a history of postpartum thyroiditis should have a serum TSH level checked annually.¹ Additionally, the presence of TPO antibodies is associated with an increased risk of the development of hypothyroidism in patients with type 1 diabetes.¹¹

6. **(B) AIT.** Amiodarone is an iodine-rich antiarrhythmic drug that has been associated with thyroid dysfunction in up to 15% of long-term users.^{12,13} AIT can develop suddenly, even after many years of therapy. There are 2 forms of AIT, type 1 and type 2.^{12,13} Type 1 AIT is a result of increased thyroid hormone synthesis and secretion and usually occurs in patients with underlying thyroid abnormalities (eg, diffuse or nodular goiter). In type 1 AIT, iodine activates thyroid hormone synthesis in autonomous areas of the thyroid gland. Type 2 AIT occurs due to release of preformed thyroid hormone and usually occurs in patients with seemingly normal thyroid glands. In type 2 AIT, amiodarone is directly toxic to the thyroid tissue and results in an inflammatory destruction of the gland and subsequent leakage of thyroid hormone into the circulation.^{12,13} Given the extremely high iodine content of amiodarone, a low RAIU can be seen in type 1 and type 2 AIT. RAIU is low in all patients with type 2 AIT; a normal or high RAIU excludes type 2 AIT. Thyroid function tests with Graves' disease and TMG could be similar to those in this case. However, thyroid ultrasound in Graves' disease usually reveals a diffusely enlarged gland with generalized hyperemia and in TMG would most likely demonstrate several thyroid nodules. Acute suppurative thyroiditis is a rare infection of the thyroid gland associated with fever, dysphagia, and thyroidal erythema and pain.

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