Abstract citation ID: bvac150.1735

Thyroid

PSAT360

Thyrotoxicosis as a Delayed Response to Amiodarone

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Introduction: Amiodarone is often used in the treatment of cardiac dysrhythmia, but has been associated with toxicity of multiple organ systems including the thyroid. Patients treated with amiodarone can develop hypothyroidism or hyperthyroidism, also known as amiodarone induced thyrotoxicosis (AIT). AIT is further categorized into type I or type II depending on pathogenesis of the disease: type I resulting from increased hormone synthesis and type II resulting from glandular destruction. We report a case of a 68-year-old male patient who developed type II AIT approximately four years after cessation of Amiodarone therapy.

Case Presentation: A 64-year-old man initially presented to an outpatient endocrinology clinic for evaluation of incidental thyroid nodules. He had a normal thyroid function. His significant past medical history included atrial fibrillation/atrial flutter status post ablation. Thyroid ultrasound revealed bilateral subcentimeter nodules which remained stable over the subsequent two years. Approximately four years after establishing care, he presented to the endocrinologist again with weakness, decreased appetite, fatigue, and weight loss. Thyroid function testing revealed new onset hyperthyroidism with suppressed TSH, <0.01 mU/L (normal 0.5-5.0 mU/L), elevated FT4 4.5 ng/dL (normal 0.7-1.9 ng/dL) and elevated FT3, 7.4 pg/mL (normal 2.3-4.1 pg/mL). He had normal TSI, normal ESR, and no history of recent viral infection. Thyroid ultrasound showed stable subcentimeter nodules. Evaluation with radioactive iodine uptake (RAIU) scan was not possible due to recent contrast exposure during CT spine imaging.

The patient was started on methimazole therapy with an assumptive diagnosis of toxic multinodular goiter. The dose was rapidly increased, within weeks, to 60mg daily, due to persistent severe hyperthyroidism. He was hospitalized twice for exacerbation of atrial fibrillation with rapid
ventricular rate requiring cardioversion and augmentation of medical therapy. Further investigation revealed the patient had received amiodarone therapy for approximately three months, almost four years prior to his current presentation. A trial of 40mg prednisone daily was started in conjunction with methimazole for possible delayed type II AIT, resulting in a rapid improvement of symptoms and Free T4 levels. Once the patient’s urinary iodine levels returned to normal, he underwent RAIU scan which revealed low uptake supporting the diagnosis of type II AIT. Methimazole therapy was stopped, and prednisone therapy was tapered down slowly and then discontinued over the course of three months, with complete resolution of hyperthyroidism.

**Conclusion:** This report reiterates the difficulties clinicians are often faced with when diagnosing and treating AIT, which, in our case, occurred four years after the discontinuation of amiodarone therapy. It highlights the importance of obtaining a history of remote amiodarone therapy, and considering steroids treatment for type II AIT, especially in patients with no clear other etiology and no response to high dose methimazole.

**Presentation:** Saturday, June 11, 2022 1:00 p.m. - 3:00 p.m.