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# Amiodarone-Induced Thyroiditis, The Importance Of Differentiating AIT-1 And AIT-2 - A Case Report.

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***Abstract:***
*A middle-aged male patient with a medical history encompassing Cardiac Sarcoidosis, Rheumatoid arthritis, Non-ischemic cardiomyopathy, and hyperlipidemia was referred to our endocrinology clinic following a diagnosis of hyperthyroidism. Upon careful review of the patient's medication history, it was ascertained that he had recently ceased taking Amiodarone. Suspicions arose regarding Amiodarone-Induced Thyrotoxicosis Type 2 (AIT-2). Consequently, the patient commenced a regimen of 20mg prednisone and was scheduled for a follow-up appointment after one week. Subsequent laboratory results exhibited marked improvement, prompting the decision to persist with prednisone therapy and establish a schedule for regular monitoring of the patient's laboratory findings. Additionally, owing to the patient's symptomatic improvement, it was determined that prednisone administration should continue for an additional two weeks, subsequent to which a gradual tapering schedule over the ensuing month was recommended, contingent upon the patient's clinical progress. This case underscores the significance of not only diagnosing Amiodarone-induced thyroiditis but also comprehending the underlying pathophysiology and attaining a precise diagnosis of the specific subtype of Amiodarone- induced thyroiditis, all of which are imperative for the provision of the most focused treatment for the patient.*

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## I. Introduction

Amiodarone is a class III anti-arrhythmic drug used in the management of ventricular and atrial arrhythmias. It is used both in life-threatening emergencies such as ventricular fibrillation/pulseless ventricular tachycardia unresponsive to defibrillation (1) and for maintenance therapy in patients who have had sustained ventricular tachyarrhythmias, particularly those with left ventricular dysfunction(2) The incidence of amiodarone- induced thyroid disorders is estimated to vary from 2% to 24% (3, 4). Typically, hypothyroidism will manifest within a short time (< 3 months) after Amiodarone initiation in 10% to 20% of cases, but can present later (> 1 year) in 5% to 10% of cases.(5) Thyrotoxicosis occurs less often than hypothyroidism with an incidence of 5% to 10%. (3,5,) Women taking amiodarone are more predisposed to develop AIH at a female:male ratio of 1.5:1(6). AIT instead appears to be more common in men (~3.9%-8.5% males vs 0.4% females) (7,8).The risk of developing thyroid or liver dysfunction appears similar in those on low-dose Amiodarone compared with those on high-dose Amiodarone and suggests that Amiodarone-related thyroid toxicity is not dose-dependent (9,10,11).Although thyrotoxicosis is less frequent than hypothyroidism in patients taking Amiodarone, the potential clinical consequences are more severe. The latter patients often have already compromising cardiac conditions, and a rise in thyroid hormone levels further increase the risk of atrial and ventricular arrhythmias. Understanding the underlying pathophysiology and attaining a specific diagnosis of the type of AIT is crucial to delivery of the most appropriate treatment(12).There are 2 main mechanisms by which Amiodarone causes thyrotoxicosis: excessive thyroid hormone production and thyroid destruction (13). Type 1 AIT occurs when the thyroid gland increases the production of thyroid hormones in response to

exposure to an iodine overload (14, 15). Type 2 is marked by the destruction of thyroid tissue, releasing thyroid hormone. It can occur years after initiation of Amiodarone therapy (27-32 months) or after the drug is discontinued, with 23% developing thyrotoxicosis after Amiodarone withdrawal (16). One clue to making the distinction between type 1 or type 2 thyrotoxicosis is that patients with type 1 frequently have underlying thyroid pathology such as diffuse or nodular goiter, or Graves' disease (with elevated TSH receptor antibodies). However, presence of positive tests for antithyroglobulin and/or antithyroperoxidase antibodies does not rule out a diagnosis of type 2 AIT (17). Thyroid ultrasound can also be used to distinguish type 1 from type 2. Before initiation of amiodarone therapy, the thyroid in the subject with type 2 AIT is more likely to be normal in size and structure. Moreover, the thyroid gland in type 1 AIT should demonstrate normal or increased vascularization on Echo color Doppler, whereas the type 2 gland will be marked by distorted architecture due to the inflammatory thyroiditis and will demonstrate normal/ reduced vascularity (18).

## II. Case Presentation

A middle-aged male patient with a medical history notable for Cardiac Sarcoidosis, Rheumatoid arthritis, Non- ischemic cardiomyopathy, and hyperlipidemia was referred to our endocrinology clinic due to a diagnosis of hyperthyroidism. The patient presented with symptoms of fatigue, weight loss, and occasional palpitations. The patient's BMI was 24.4 kg, and his blood pressure and heart rate were within normal limits. Physical examination revealed no acute stress, tremors, or exophthalmos. There was no tenderness, enlargement, or palpable nodules upon thyroid palpation. Laboratory results showed TSH levels below 0.015, FT4 levels at 5.7, FT3 at 6.3, and TSH receptor antibodies below 1.0 at presentation. Upon reviewing the patient's medications, it was noted that he had been taking amiodarone for cardiac sarcoidosis-related ventricular tachyarrhythmias until less than 1 week prior. The patient was advised to discontinue Amiodarone by his primary care physician. It was observed that the onset of thyrotoxicosis occurred almost a year after the initiation of Amiodarone. Due to the absence of prior thyroid disorders and negative thyroid antibodies, Amiodarone-Induced Thyrotoxicosis Type 2 (AIT-2) was suspected. The patient was initiated on 20mg prednisone and scheduled for a follow-up appointment after one week. A subsequent thyroid ultrasound revealed normal results, further confirming the diagnosis of AIT-2. Laboratory results at the one- week follow-up showed TSH levels below 0.015 and FT4 levels at 4.7.The notable increase in FT4 levels justified the decision to proceed with prednisone treatment and establish a schedule for regular monitoring of the patient's laboratory results. Concurrently, the patient exhibited symptomatic improvement. Given the observed progress, it was determined to sustain the administration of prednisone for an additional two weeks, after which a gradual tapering process over the subsequent month will be initiated in accordance with the patient's clinical advancement.

## III. Conclusion

Patients undergoing treatment with Amiodarone should be meticulously monitored due to the potential occurrence of amiodarone-induced thyroid disorders at any given point during the course of treatment. Accurate diagnosis and management of both hypothyroidism and thyrotoxicosis are imperative. Hypothyroidism necessitates treatment with levothyroxine, while the management of thyrotoxicosis depends on its subtype. Type 1 typically requires treatment with Methimazole, whereas type 2 is commonly treated with prednisone. Given the presence of underlying cardiac conditions, it is crucial to maintain vigilant monitoring throughout the duration of treatment .

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