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AMIODARONE-INDUCED THYROID DYSFUNCTION: A RARE ADVERSE EFFECT

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ABSTRACT

Background: Amiodarone is a widely prescribed class III antiarrhythmic drug used for the management of atrial and ventricular arrhythmias. Despite its effectiveness, long-term therapy may lead to thyroid dysfunction because of its high iodine content and direct effects on thyroid tissue. Amiodarone-induced thyrotoxicosis is an uncommon but clinically important adverse effect that requires early recognition and management. **Case Presentation:** A 68-year-old male with persistent atrial fibrillation receiving amiodarone 200 mg daily for three years presented with fatigue, palpitations, excessive sweating, heat intolerance, tremors, and significant weight loss. Laboratory investigations revealed suppressed thyroid-stimulating hormone levels with elevated free thyroxine and free triiodothyronine levels. Thyroid autoantibodies were negative, and Doppler ultrasonography demonstrated reduced thyroid vascularity, suggestive of Type 2 amiodarone-induced thyrotoxicosis. Amiodarone was discontinued, and treatment with oral prednisolone and beta-blocker therapy was initiated. The patient showed progressive clinical and biochemical improvement, achieving complete recovery during follow-up. **Conclusion:** This case emphasizes the importance of regular thyroid monitoring in patients receiving long-term amiodarone therapy to facilitate early diagnosis and prevent serious complications.

KEYWORDS: Amiodarone, Thyrotoxicosis, Thyroid Dysfunction, Adverse Drug Reaction, Atrial Fibrillation, Corticosteroids.

INTRODUCTION

Amiodarone is an antiarrhythmic of class III which is widely used in the therapy of supraventricular and ventricular arrhythmias such as atrial fibrillation and ventricular tachycardia. Because of its efficiency in controlling cardiac rhythm and minimizing arrhythmia complications, amiodarone continues to be among the most widely prescribed antiarrhythmic drugs. However, amiodarone has a broad spectrum of side effects targeting various organs and systems, namely, the respiratory system, liver, eyes, skin, nervous system, and thyroid gland. The drug demonstrates the prolonged effect due to a slow elimination rate and accumulation in different tissues. As for the side effects, one of the most clinically important ones is a thyroid dysfunction as it can greatly impact the cardiovascular system condition.[1,2]

The mechanism through which amiodarone causes thyroid problems is due to the high iodine concentration and direct actions of the drug on the metabolism of the thyroid hormones. Amiodarone tablets each contain about 75 milligrams of iodine out of which 10% is secreted as free iodine on a daily basis. The iodine content is much higher than the required daily value and it interferes with the physiology of the thyroid gland. Moreover, the drug blocks the peripheral conversion of T4 to T3 and impacts on the synthesis and release of thyroid hormones, and hence causing hypothyroidism or thyrotoxicosis depending on the condition of the patients' thyroid gland.[3,4]

Amiodarone-induced thyroid problems are typically grouped as either amiodarone-induced hypothyroidism or amiodarone-induced thyrotoxicosis (AIT). Amiodarone-induced hypothyroidism is relatively more common in iodine-rich environments than amiodarone-induced thyrotoxicosis, which is more prevalent in iodine-poor settings. However, AIT can be sub-classified as either Type 1 or Type 2. Type 1 AIT arises owing to excess production of thyroid hormones in individuals suffering from underlying thyroid conditions, for instance, multinodular goiter or latent Graves' disease. In contrast, Type 2 AIT arises owing to the destruction of thyroid follicular cells and consequent release of preformed thyroid hormones into the bloodstream. Differentiation between these types is necessary since their treatment and outcomes vary.[5,6]

Clinical signs and symptoms of amiodarone induced thyrotoxicosis are diverse and could mimic those found in individuals suffering from some sort of cardiovascular disorder. Individuals might exhibit symptoms like weight loss, weakness, heart palpitations, tremors, excessive sweating, anxiety, muscular weakness, or worsening of heart failure. The elderly population might have atypical symptoms which will not be easily recognized resulting in delayed treatment and diagnosis. The diagnosis is based upon clinical presentation, laboratory tests that include thyroid hormone levels, thyroid antibodies test, ultrasonography and color-flow Doppler examination. Early recognition of thyroid abnormalities is crucial as thyrotoxicosis if left untreated could worsen heart arrhythmia, increase hospitalization and lead to high mortality and morbidity in patients on chronic amiodarone medication.[7,8]

It is important to monitor thyroid function prior to the administration of amiodarone therapy and during the course of treatment to be able to detect thyroid dysfunction at an early stage. The current clinical recommendations are aimed at the initial examination of the thyroid-stimulating hormone, free thyroxine, and presence of antibodies of the thyroid gland, along with the follow-up examinations on a regular basis. This may help to prevent complications. The following case study involves the unusual case of Type 2 amiodarone-induced thyrotoxicosis of an older patient who has been taking amiodarone therapy for persistent atrial fibrillation.[9,10]

CASE PRESENTATION

Patient Demographics

A 68-year-old male presented to the cardiology outpatient department of a tertiary care teaching hospital with complaints of progressive fatigue, palpitations, excessive sweating, tremors, and unintentional weight loss over the preceding two months. The patient was a known case of persistent atrial fibrillation and had been receiving long-term amiodarone therapy for rhythm control. He was conscious, oriented, and cooperative at the time of presentation.

Chief Complaints

The patient presented with the following complaints:

- Progressive fatigue and generalized weakness for 2 months
- Palpitations for 6 weeks
- Excessive sweating for 2 months
- Heat intolerance for 2 months
- Fine tremors of both hands for 1 month
- Unintentional weight loss of approximately 6 kg over 8 weeks
- Reduced exercise tolerance and easy fatigability

History of Present Illness

The patient was apparently well until two months prior to presentation when he began experiencing generalized weakness and fatigue. Initially, the symptoms were mild and intermittent but gradually worsened, affecting his routine daily activities. Over the following weeks, he developed excessive sweating, heat intolerance, and noticeable tremors of both hands. He also reported episodes of palpitations associated with mild shortness of breath on exertion.

Despite maintaining a normal appetite, he experienced an unintentional weight loss of approximately 6 kg over two months. The patient denied any fever, neck pain, diarrhea, recent viral illness, chest pain, syncope, or changes in bowel habits. There was no history suggestive of thyroid disease before the initiation of amiodarone therapy.

The patient had been receiving amiodarone 200 mg once daily for approximately three years for persistent atrial fibrillation. Considering the temporal relationship between prolonged amiodarone exposure and onset of symptoms, drug-induced thyroid dysfunction was suspected.

Past Medical History

The patient had a history of:

- Persistent atrial fibrillation diagnosed 3 years earlier
- Hypertension for 10 years
- Stable ischemic heart disease for 5 years
- Dyslipidemia for 6 years

Medication History

The patient had been receiving the following medications regularly:

Drug	Dose	Frequency	Indication
Amiodarone	200 mg	Once daily	Atrial fibrillation
Metoprolol	50 mg	Twice daily	Rate control, hypertension
Aspirin	75 mg	Once daily	Ischemic heart disease
Atorvastatin	20 mg	Once daily	Dyslipidemia
Telmisartan	40 mg	Once daily	Hypertension

Family History

His parents had a history of hypertension, but no thyroid abnormalities were documented among first-degree relatives.

Social History

The patient lived with his spouse and was independent in activities of daily living. No smoking and alcohol habit.

Occupational History

The patient was a retired government employee who had worked in an administrative position for over 35 years.

Allergic History

The patient had no known history of any allergies.

Vital Signs

Parameter	Value
Blood Pressure	136/84 mmHg
Heart Rate	112 beats/minute
Respiratory Rate	18 breaths/minute
Temperature	98.4°F
Oxygen Saturation	98% on room air

General Examination Findings

- Conscious, alert, and oriented
- Mild weight loss evident
- Warm and moist skin
- Fine tremors of both hands
- No pallor, icterus, cyanosis, clubbing, or edema
- No exophthalmos
- No pretibial myxedema
- No palpable thyroid enlargement

Systemic Examination

Cardiovascular Examination

- Pulse irregularly irregular
- Tachycardia present
- Normal heart sounds
- No audible murmurs
- Findings consistent with atrial fibrillation

Respiratory Examination

- Bilateral air entry equal
- No wheeze or crackles
- Normal vesicular breath sounds

Abdominal Examination

- Soft and non-tender
- No organomegaly
- Normal bowel sounds

Neurological Examination

- Higher mental functions intact
- Fine postural tremors present
- No focal neurological deficits
- Muscle power preserved

Laboratory Investigations

Complete Blood Count

Parameter	Result	Reference Range
Hemoglobin	13.4 g/dL	13–17 g/dL
Total Leukocyte Count	7,800 cells/mm³	4,000–11,000
Neutrophils	65%	40–75%
Lymphocytes	28%	20–45%
Platelet Count	245,000/mm³	150,000–450,000

Renal Function Tests

Parameter	Result	Reference Range
Blood Urea Nitrogen	28 mg/dL	10–50 mg/dL
Serum Creatinine	0.9 mg/dL	0.6–1.2 mg/dL
Serum Sodium	138 mmol/L	135–145 mmol/L
Serum Potassium	4.3 mmol/L	3.5–5.0 mmol/L

Liver Function Tests

Parameter	Result	Reference Range
AST	32 U/L	<40 U/L
ALT	30 U/L	<40 U/L
ALP	92 U/L	44–147 U/L
Total Bilirubin	0.8 mg/dL	0.2–1.2 mg/dL
Serum Albumin	4.0 g/dL	3.5–5.0 g/dL

Thyroid Function Tests

Parameter	Result	Reference Range
TSH	0.02 µIU/mL	0.4–4.5 µIU/mL
Free T4	3.8 ng/dL	0.8–1.8 ng/dL
Free T3	6.4 pg/mL	2.3–4.2 pg/mL

Thyroid Autoimmune Profile

Parameter	Result
Anti-Thyroid Peroxidase Antibody	Negative
Anti-Thyroglobulin Antibody	Negative
TSH Receptor Antibody	Negative

Radiological and Special Investigations

Thyroid Ultrasonography

- Mild diffuse heterogeneity of thyroid parenchyma
- No thyroid nodules
- No evidence of multinodular goiter
- No thyroid enlargement

Color Flow Doppler Ultrasonography

- Reduced thyroid vascularity
- Findings suggestive of destructive thyroiditis (Type 2 AIT)

Electrocardiography (ECG)

- Atrial fibrillation with rapid ventricular response
- Heart rate approximately 110–120 beats/minute

Echocardiography

Parameter	Finding
Left Ventricular Ejection Fraction	50%
Chamber Size	Mild left atrial enlargement
Valvular Function	No significant abnormalities
Pericardial Effusion	Absent

DIAGNOSIS

Based on the patient's clinical presentation of progressive fatigue, weight loss, palpitations, heat intolerance, excessive sweating, and fine tremors, along with a history of prolonged amiodarone therapy for persistent atrial fibrillation, drug-induced thyroid dysfunction was strongly suspected. Laboratory investigations revealed markedly suppressed thyroid-stimulating hormone (TSH) levels with elevated free thyroxine (FT4) and free triiodothyronine (FT3) levels, confirming thyrotoxicosis. Thyroid autoantibodies were negative, and color Doppler ultrasonography demonstrated reduced thyroid vascularity, suggestive of destructive thyroiditis. Considering the temporal relationship with amiodarone exposure and exclusion of other causes, a final diagnosis of Amiodarone-Induced Type 2 Thyrotoxicosis (AIT) was established.

TREATMENT

Following the diagnosis of amiodarone-induced Type 2 thyrotoxicosis, a multidisciplinary team consisting of cardiologists and endocrinologists was involved in the patient's management. Considering the strong temporal association between the onset of thyrotoxic symptoms and long-term amiodarone therapy, amiodarone 200 mg orally once daily was discontinued after careful assessment of the patient's cardiovascular status and arrhythmia risk.

Alternative rate-control strategies were considered to maintain cardiac stability while minimizing further thyroid injury.

The patient was closely monitored for recurrence of arrhythmias and worsening cardiac symptoms during the withdrawal period.

To control the inflammatory process associated with destructive thyroiditis, the patient was initiated on oral prednisolone 40 mg once daily (approximately 0.5–0.7 mg/kg/day). Corticosteroid therapy was selected because Type 2 amiodarone-induced thyrotoxicosis is primarily caused by inflammation and destruction of thyroid follicular cells rather than increased hormone synthesis. Prednisolone was continued at the initial dose for four weeks, resulting in a gradual reduction in thyrotoxic symptoms and improvement in thyroid hormone levels. Following clinical stabilization, the dose was tapered gradually by 5–10 mg every two weeks over a period of approximately three months to prevent relapse and minimize corticosteroid-related adverse effects.

For symptomatic control of thyrotoxicosis and cardiovascular manifestations, metoprolol tartrate 50 mg orally twice daily was continued. Beta-blocker therapy helped reduce palpitations, tachycardia, tremors, anxiety, and excessive sweating by antagonizing the peripheral effects of excess thyroid hormones. Heart rate and blood pressure were monitored regularly throughout hospitalization and follow-up visits. The patient demonstrated significant symptomatic improvement within the first few weeks of treatment, with a reduction in resting heart rate and improved exercise tolerance.

The patient's pre-existing cardiovascular medications were continued to optimize cardiac function and prevent thromboembolic complications. These included aspirin 75 mg orally once daily as antiplatelet therapy for ischemic heart disease, atorvastatin 20 mg orally at bedtime for dyslipidemia management, and telmisartan 40 mg orally once daily for blood pressure control. Routine monitoring of liver function, renal function, electrolyte levels, and cardiovascular parameters was performed during treatment to ensure medication safety and overall clinical stability.

Regular thyroid function monitoring formed an essential component of management. Serum TSH, free T4, and free T3 levels were assessed every four to six weeks to evaluate treatment response. At the first follow-up visit after one month, the patient reported marked improvement in fatigue, sweating, palpitations, and tremors. Serial laboratory investigations demonstrated progressive reductions in free thyroid hormone levels with gradual recovery of TSH concentrations. By the third month of therapy, substantial biochemical improvement was observed, allowing further tapering of corticosteroid treatment.

At six months of follow-up, the patient achieved complete clinical and biochemical remission. Thyroid function tests had returned to normal ranges, with TSH 2.1 µIU/mL, free T4 1.3 ng/dL, and free T3 3.4 pg/mL. No recurrence of thyrotoxic symptoms was noted, and the patient remained hemodynamically stable without further thyroid-related complications. The overall management was considered successful, with complete resolution of amiodarone-induced Type 2 thyrotoxicosis following withdrawal of the offending drug, corticosteroid therapy, symptomatic treatment with beta-blockers, and close endocrinological and cardiological follow-up.

DISCUSSION

Amiodarone remains one of the most effective antiarrhythmic drugs for the management of atrial and ventricular arrhythmias, but thyroid dysfunction is a well-recognized adverse effect because of its high iodine content and direct cytotoxic effects on thyroid follicular cells. The present patient developed classical manifestations of thyrotoxicosis, including weight loss, palpitations, tremors, and excessive sweating following long-term amiodarone exposure. The temporal relationship between symptom onset and prolonged therapy strongly supports amiodarone-induced thyroid dysfunction. Similar findings were reported by Martino et al., who described the thyroid gland as one of the major

target organs affected by amiodarone toxicity. Likewise, Harjai and Licata observed that both hyperthyroidism and hypothyroidism can occur during chronic amiodarone treatment, particularly among elderly cardiac patients receiving prolonged therapy.[11,12]

The diagnosis of Type 2 amiodarone-induced thyrotoxicosis in this patient was established through clinical findings, biochemical evidence, and thyroid imaging. Suppressed TSH with elevated FT3 and FT4 levels confirmed thyrotoxicosis, while negative thyroid autoantibodies and reduced vascularity on Doppler ultrasonography favored destructive thyroiditis. Similar diagnostic features were reported by Tsang and Houlden, who emphasized the value of Doppler ultrasonography in distinguishing Type 2 AIT from iodine-induced hyperthyroidism. In another review, Eskes and Wiersinga highlighted that Type 2 AIT frequently develops in patients without pre-existing thyroid disease and is characterized by inflammatory destruction of thyroid tissue resulting in release of stored thyroid hormones.[13,14]

The patient responded favorably to discontinuation of amiodarone and initiation of corticosteroid therapy. Prednisolone was selected because Type 2 AIT is predominantly an inflammatory process involving destructive thyroiditis. Significant symptomatic improvement and normalization of thyroid hormone levels were observed during follow-up. Bogazzi et al. demonstrated that glucocorticoids remain the treatment of choice for Type 2 AIT and are associated with rapid biochemical improvement. Similarly, Bartalena and colleagues, in the European Thyroid Association guidelines, recommended oral glucocorticoids as first-line therapy for patients with confirmed Type 2 disease, particularly when imaging findings support destructive thyroiditis.[15,16]

This case highlights the importance of routine thyroid surveillance in patients receiving long-term amiodarone therapy.

Thyroid dysfunction may remain undetected because symptoms often overlap with manifestations of underlying cardiovascular disease. Delayed diagnosis can worsen arrhythmias, precipitate heart failure, and increase morbidity.

Basaria and Cooper emphasized that baseline thyroid evaluation and periodic thyroid function testing are essential before and during amiodarone treatment. Furthermore, Batcher et al. reported that regular biochemical monitoring facilitates early detection of thyroid abnormalities and improves patient outcomes by allowing prompt intervention. The complete recovery observed in this patient further reinforces the benefits of vigilant monitoring and multidisciplinary collaboration between cardiologists and endocrinologists.[17,18]

CONCLUSION

Amiodarone-induced thyroid dysfunction is an important and potentially serious adverse effect that may occur during long-term therapy, particularly in elderly patients with underlying cardiac disorders. The present case highlights the development of Type 2 amiodarone-induced thyrotoxicosis characterized by fatigue, weight loss, palpitations, tremors, and biochemical evidence of hyperthyroidism following prolonged amiodarone use. Early recognition of clinical symptoms, timely thyroid function testing, and appropriate imaging studies were crucial in establishing the diagnosis.

Prompt discontinuation of the suspected drug and initiation of corticosteroid therapy resulted in significant clinical and biochemical improvement. This case emphasizes the importance of baseline and periodic thyroid monitoring in all patients receiving amiodarone therapy. Increased awareness among clinicians regarding this adverse drug reaction can facilitate early diagnosis, prevent serious cardiovascular complications, and improve overall patient outcomes through timely multidisciplinary management.

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