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Case Report

Amiodarone-Induced Thyrotoxicosis Presenting as Congestive Heart Failure Exacerbation and Thyroid Storm: Role of Plasmapheresis in Management and Prevention of Complications

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ABSTRACT

Amiodarone-induced thyrotoxicosis (AIT) is a subtype of thyrotoxicosis caused by the long-term administration of the antiarrhythmic drug amiodarone. It's classified into type 1 AIT, which is defined as hypersecretion of the thyroid hormone, while type 2 AIT is destructive thyroiditis leading to increased release of thyroid hormone. Each type has its special management and diagnostic features. We report a case of a 72-year-old male with a history of heart failure with reduced ejection fraction (HFrEF) and type 2 diabetes mellitus (DM) who presented with congestive heart failure (CHF) exacerbation and symptoms of an impending thyroid storm. After the diagnosis of type 2 AIT, medical treatment, as well as plasmapheresis, was initiated. In further admissions, the patient returned with ventricular tachycardia and right subclavian deep venous thrombosis due to recurrent catheterization and a hypercoagulable state. The diagnostic workup revealed elevated free T4, suppressed TSH, and initially elevated AST and ALT, which normalized subsequently. Imaging showed decreased thyroid vascularity. This case report highlights the importance of distinguishing between AIT types, tailoring optimal treatment decisions, closely monitoring and following up on such cases, and adhering to treatment to prevent catastrophic complications. Further research is necessary to identify early markers of amiodarone toxicity to prompt early diagnosis and better prognosis.

1. Introduction

Amiodarone, a benzofuranic iodine-enriched agent, contains approximately 37.5% iodine by weight [1]. It is a commonly used antiarrhythmic medication for both ventricular arrhythmias and paroxysmal atrial tachycardia [2, 1]. It is particularly used in patients with complicated and uncontrolled conditions who require standard treatment regimens [1]. However, it is well known for its life-threatening adverse events (AEs) [1]. These AEs are related to amiodarone because its chemical structure resembles thyroxine (T4), enabling it to bind to thyroid receptors [3]. As a result, it leads to either thyrotoxicosis [amiodarone-induced thyrotoxicosis] (AIT) or hypothyroidism, which doesn't pose significant strains [3].

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However, AIT is a highly fatal adverse event, especially for cardiac patients [3]. AIT is commonly mistaken for spontaneous hyperthyroidism, as not all individuals exhibit thyrotoxicosis symptoms and signs [3]. An exacerbation of an underlying cardiac disease could also mask it. These manifestations may include worsening of cardiac arrhythmia, recurrence of atrial fibrillation, palpitations, or the development of angina [3].

AIT is subclassified into AIT types 1 and 2, which have different underlying pathophysiology, as shown in **(Table 1)** [3]. AIT 1 commonly occurs in conjunction with thyroid disease, such as latent Graves' disease or multinodular goiter, leading to increased thyroid hormone production due to a sudden increase in iodine intake; herein, AIT type 1 is more common in patients with low dietary iodine intake [4]. However, AIT type 2 is more frequent than AIT type 1, resulting in amiodarone toxicity, which can damage the thyroid gland, leading to the release of pre-formed hormones without an increase in hormone biosynthesis [5].

It's essential to distinguish between them, as their treatment regimens differ. AIT 1 is commonly associated with an enlarged thyroid gland, typically characterized by a low T4/T3 ratio (<4), elevated thyroid blood flow on Doppler ultrasound, and high iodine uptake levels on a thyroid scan [6]. Thus, the treatment regimen could be antithyroid drugs such as methimazole or propylthiouracil. While AIT 2 develops in healthy patients without underlying thyroid diseases and is presented with a normal or atrophic thyroid gland, it

Table 1: Comparison of Amiodarone-Induced Thyrotoxicosis (AIT) Type 1 and Type 2

Feature	AIT Type 1	AIT Type 2
Cause	Underlying thyroid disease (e.g., multinodular goiter, Graves' disease)	Direct thyroid destruction by amiodarone toxicity
Pathophysiology	Increased thyroid hormone synthesis	Thyroid follicular cell damage and release of thyroid hormones
Pre-existing Thyroid Condition	Often in patients with pre-existing thyroid disease	Usually occurs in a previously normal thyroid
Onset	Gradual	Sudden
Radioiodine Uptake (RAIU)	Normal or increased	Low or absent
Doppler Ultrasound	Increased vascularity (hypervascularity)	Decreased vascularity (hypovascularity)
Serum IL-6 Levels	Normal or mildly elevated	Markedly elevated
Response to Therapy	Poor response to corticosteroids; responds to thionamides (methimazole, propylthiouracil)	Responds well to corticosteroids; poor response to thionamides
Treatment Options	- Thionamides (Methimazole, PTU) - Potassium perchlorate (to reduce iodine uptake) - Sometimes, plasmapheresis or surgery is required in severe cases	- High-dose corticosteroids (prednisone) - Supportive treatment until resolution
Prognosis	May require long-term treatment or thyroidectomy	Usually self-limited, resolves with corticosteroids

AIT, Amiodarone-Induced Thyrotoxicosis; RAIU, Radioiodine Uptake; IL-6, Interleukin-6; PTU, Propylthiouracil

usually has a high T4/T3 ratio (>4) and low iodine uptake. As AIT 2, or destructive thyroiditis, has an ongoing inflammatory process, its effective treatment is glucocorticoids [6].

In this case report, we describe the challenging journey of an old man with heart failure and other comorbidities who developed AIT. So, he required urgent efforts to reduce serum thyroxine to prevent cardiac decompensation. With persistent ambiguity around the AIT subtype, we successfully restored the euthyroid state with a combination of methimazole and prednisone.

2. Case Presentation

A 72-year-old male with no prior history of thyroid disease presented to the Emergency Department (ED) with generalized weakness, shortness of breath, and increased confusion. He was admitted to acute on top of chronic congestive heart failure (CHF) and was diagnosed with severe hyperthyroidism based on abnormal thyroid function tests (TFTs). His past medical history includes heart failure with reduced ejection fraction (EF 10-20%) 1 year ago, coronary artery disease with a prior non-ST-elevation myocardial infarction (NSTEMI) 2 years ago, ischemic cardiomyopathy, type 2 diabetes mellitus, atrial fibrillation, and a Factor V Leiden mutation. The patient had been on amiodarone for one year but stopped the medication two months prior due to insurance issues.

The patient presented to the ED with generalized weakness, shortness of breath, confusion, and vomiting. He had stopped taking amiodarone two months prior due to insurance issues. His vital signs upon arrival were a temperature of 98.1°F, a heart rate of 94 beats per minute (bpm), a blood pressure of 121/60 mmHg, and an SpO2 of 91% on room air. Physical examination showed no signs of a goiter, clear lung fields, a regular heart rhythm, a non-tender abdomen, no conjunctival injection, no scleral icterus, and moist oral mucosal membranes. As seen in **(Table 2)**, pertinent laboratory tests upon admission revealed significantly elevated free T4 (7.7 ng/dL), suppressed TSH (<0.1 μIU/mL), and markedly elevated liver enzymes (AST/ALT in the 300s), consistent with amiodarone-induced thyrotoxicosis and impending thyroid storm. Thyroid antibodies were pending to further differentiate the type of AIT. The Burch-Wartofsky Point Scale (BWPS) indicated an impending thyroid storm. Final diagnoses included Type II amiodarone-induced thyrotoxicosis, impending thyroid storm, and acute-on-chronic CHF. Management included cholestyramine (4 g QID), beta-blockers (titrated to HR 60–90 bpm), hydrocortisone (100 mg q8 h), and insulin for steroid-induced hyperglycemia. Plasmapheresis was initiated with a central line, and the first session on day 2 after admission reduced Free T4 from 7.7 to 5.32. LFTs improved, and the patient was discharged with weekly TFT/LFT monitoring and continued medications. The family confirmed that they would be available to help the patient with insulin injections if needed. The patient is also learning how to give himself insulin, as he may need to be on a higher dose of insulin for some time since he is on high-dose steroids for AIT. The patient continued cholestyramine (4 g) four times daily, prednisone 60 mg daily, and a beta-blocker, titrating the dose to maintain a heart rate of less than 90 beats per minute.

Two months later, the patient was readmitted with a recurrent exacerbation of CHF and ventricular tachycardia (VT). Vital signs included a temperature of 97.8 °F, heart rate of 82 bpm, blood pressure of 110/83 mmHg, and SpO2 of 100%. The physical examination findings were consistent with previous admissions and showed no significant changes. As seen in **(Table 2)**, laboratory tests during the second admission showed persistently elevated free T4 (7.38 ng/dL), suppressed TSH (<0.1 μIU/mL), and mildly elevated liver enzymes (AST 49 U/L, ALT 90 U/L), indicating ongoing thyrotoxicosis and the need for continued aggressive management. Recurrent V. tach episodes and persistently elevated Free T4 (>7.77) prompted the initiation of methimazole (60 mg daily, increased to 90 mg on 10/24) and the continuation of p.o. Prednisone (60 mg daily). Cholestyramine was discontinued due to non-compliance. The patient underwent his first session of plasmapheresis on October 18. After the first plasmapheresis session, his thyroid hormone levels improved but gradually increased to approximately 6.75 (free T4). We conducted another plasmapheresis session on October 24, resulting in an immediate improvement in free T4 levels. We discontinued cholestyramine on 10/24 since the patient was not getting the medications as recommended. Post-TPE, LFTs normalized, and the patient was discharged on methimazole

Table 2: Laboratory Investigations

Test	Result	Normal range
First admission		
Free T4	7.7	0.8–1.8 ng/dL
TSH	0.01L	0.4–4.0 μIU/mL
Second admission		
Free T4	7.38	0.8–1.8 ng/dL
TSH	<0.01	0.4–4.0 μIU/mL
AST	49 U/L	10–40 U/L
ALT	90 U/L	7–56 U/L
Third admission		
Free T4	5,14	0.8–1.8 ng/dL
Free T3	2.9	2.3–4.2 pg/mL
TSH	<0.01	0.4–4.0 μIU/mL

Free T4, Free Thyroxine; Free T3, Free Triiodothyronine; TSH, Thyroid-Stimulating Hormone; AST, Aspartate Aminotransferase; ALT, Alanine Aminotransferase;

(90 mg daily), prednisone (60 mg daily), and beta-blockers, with plans for outpatient TPE as needed.

After a month, the patient was admitted with right shoulder pain due to deep vein thrombosis (DVT), requiring intravenous heparin. As seen in (**Table 2**), Thyroid function tests revealed a Free T4 level greater than 5.14 ng/dL, suppressed TSH, and normal Free T3 at 2.9 pg/mL. A neck ultrasound with Doppler imaging showed decreased thyroid vascularity, which supported a diagnosis of Type II AIT. Management included continued methimazole (90 mg daily), prednisone (60 mg daily), and IV heparin for DVT. Goals of care discussions reaffirmed the family's refusal of thyroidectomy. Outpatient TPE was deemed feasible for future hormone surges.

3. Discussion

The severe amiodarone-induced thyrotoxicosis (AIT), end-stage heart failure, and other multiple comorbidities are what make this 72-year-old male a challenging case to manage, especially when traditional treatments are contraindicated or insufficient.

Although the amiodarone was discontinued two months before, this did not prevent thyrotoxicosis. Amiodarone has an extremely long half-life (approximately 100 days) due to its lipophilic nature and extensive tissue distribution. This explains why thyrotoxicosis can develop even months after discontinuation, as the drug continues to be released from tissue stores. Studies have shown that AIT can occur in 3-5% of amiodarone-treated patients in iodine-sufficient areas and up to 10% in iodine-deficient regions[6].

Amiodarone partially exerts its antiarrhythmic effects by suppressing the Type 1 5-deiodinase enzyme, which inhibits the conversion of T4 to T3 in peripheral tissues. Additionally, its active metabolite, desethylamiodarone (DEA), can cause direct thyroid cell damage at high concentrations, leading to both hyperthyroidism and hypothyroidism based on individual vulnerability [7]. The key to effective diagnosis and management is the accurate differentiation between Type 1 and Type 2 AIT. The patient showed reduced thyroid vascularity on color flow Doppler sonography, which supports the diagnosis of Type 2 AIT, linked to drug-induced thyroiditis rather than increased hormone production. The absence of thyroid antibodies (pending but likely negative given the clinical course) further supports Type II AIT, as autoimmunity is more characteristic of Type I disease. Type 2 AIT is seen in iodine-deficient regions and doesn't necessitate pre-existing thyroid disease. Unlike Type 1 AIT, which occurs more frequently in areas with high iodine levels, Type 2 AIT is seen in iodine-deficient regions and doesn't necessitate pre-existing thyroid disease [8].

As amiodarone therapy can induce a delayed onset of Type 2 AIT even years after the initial treatment course, ongoing TFT is needed for follow-up, especially in high-risk individuals. The patient's failure to attend follow-up visits leads to a delay in diagnosis and intervention, which causes the progression of severe thyrotoxicosis [2, 9]. Generalized weakness, shortness of breath, confusion, and vomiting were the initial presentation, which raised concerns for an impending thyroid storm, confirmed by the BWPS. Elevated free T4 (7.7 ng/dL) and suppressed TSH (<0.1 μIU/mL) confirmed the diagnosis, requiring immediate intervention. Similar cases have been reported where AIT Type 2 manifested as a thyroid storm, often complicating underlying cardiac conditions [10].

As thyroid storm is a life-threatening complication, the first aim of management is to control plasma thyroid levels and its related symptoms. Patient's elevated liver enzymes and ineligibility for surgery made conservative management the best choice for him. Cholestyramine was used to bind thyroid hormones in the gut, thereby limiting recirculation; however, its effectiveness was compromised by the patient's non-compliance [11]. To stabilize the heart rate within the range of 60-90 bpm and manage adrenergic symptoms, beta blockers were used. Hydrocortisone was utilized to control steroid-induced hyperglycemia and address the inflammation linked to Type 2 AIT [12]. Plasmapheresis emerged as an essential intervention, significantly reducing free T4 levels during the acute crisis [13]. This is consistent with prior research demonstrating substantial reductions in free T4 and T3 after multiple sessions of therapeutic plasma exchange (TPE) [14]. Methimazole and prednisone were introduced subsequently, leading to the successful and sustained normalization of thyroid function despite the patient's hepatic limitations [15]. Methimazole is considered the treatment of choice for antithyroid therapy due to its effectiveness and relatively low side-effect profile. However, regular liver function tests (LFTs) are necessary to identify any signs of hepatotoxicity early on [16].

During the second admission, the patient developed VT, further emphasizing the critical cardio-endocrine connection in AIT Type 2. Previous studies have highlighted similar cases in which uncontrolled thyrotoxicosis triggered arrhythmias, reinforcing the necessity of regular TFT follow-up with established guidelines [17, 18, 19, 20]. Beta-blocker usage and plasmapheresis were necessary to stabilize the patient's rhythm and avoid further complications.

After undergoing several cycles of plasmapheresis, the patient developed a right subclavian DVT. This condition treatment aligns with the hypercoagulable state associated with thyrotoxicosis induced using central venous catheters, further aggravated by a factor V Leiden mutation. Risk of venous thromboembolism increased by 1.55-fold in hyperthyroid patients compared to controls in a population-based cohort study of 12,844 patients [21]. In response, anticoagulation therapy with intravenous heparin was initiated to reduce the risk of thromboembolism. The definitive treatment for refractory AIT is thyroidectomy, which offers quick normalization of thyroid hormone levels and enhances cardiac performance [22]. However, the patient's family refused to undergo surgery due to concerns about the potential risks in his fragile condition [23].

Their decision highlights the ethical challenges of recommending invasive procedures for high-risk elderly patients.

Patient-centered care principles often favor conservative measures over surgery. These conservative measures focus on symptom relief and enhancing quality of life. While these approaches successfully manage immediate symptoms, they may potentially delay the resolution of underlying thyroid dysfunction, which could negatively affect long-term outcomes [22]. Future research is needed to develop strategies that optimize outcomes for similar high-risk patients, taking into account efficacy, safety, and patient preferences.

4. Conclusion

Plasmapheresis provided rapid hormonal control in emergent cases, while respecting patient preferences was essential for improving adherence and quality of life. Future studies should focus on personalized treatment strategies for high-risk populations to enhance outcomes.

Conflicts of Interest

The authors declare that they have no competing interests.

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Large Language Model

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Authors Contribution

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Data Availability

Patient data related to this study are not publicly available but can be obtained upon request from the corresponding author.

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