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Case Report

Amiodarone-Induced Thyrotoxicosis Presenting as Impending Thyroid Storm in a Patient with Structural Heart Disease: A Case Report

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ABSTRACT

Thyroid storm is a rare, life-threatening endocrine emergency representing the most severe expression of thyrotoxicosis. Amiodarone-induced thyrotoxicosis may precipitate severe thyrotoxic decompensation, particularly in patients with underlying cardiovascular disease and may occur even months after amiodarone withdrawal because of its prolonged tissue persistence. We report a 62-year-old woman with heart failure with preserved ejection fraction, mechanical mitral and aortic valve prostheses, paroxysmal atrial fibrillation and previously diagnosed amiodarone-induced hyperthyroidism who presented with palpitations, dizziness, blurred vision, agitation and unintentional weight loss. Laboratory evaluation showed suppressed thyroid-stimulating hormone, elevated free thyroxine and a markedly elevated international normalized ratio in the absence of clinically evident bleeding. An atrial tachyarrhythmia with rapid ventricular response was documented on admission. A Burch-Wartofsky Point Scale score of 35, as calculated by the treating team, supported a diagnosis of impending thyroid storm. Treatment with beta-blockade, propylthiouracil, corticosteroids and supportive care was followed by progressive clinical and biochemical improvement. This case emphasizes the diagnostic and therapeutic challenges of amiodarone-induced thyrotoxicosis in patients with structural heart disease and the importance of early recognition, prompt treatment and continued thyroid surveillance even after amiodarone discontinuation.

Keywords: Amiodarone; Amiodarone-induced thyrotoxicosis; Thyrotoxicosis; Thyroid storm; Atrial flutter; Structural heart disease; Hyperthyroidism; Endocrine emergency

Introduction

Thyroid storm is a rare but potentially fatal endocrine emergency characterized by acute multisystem decompensation in the setting of severe thyrotoxicosis. Diagnosis is primarily clinical and treatment should not be delayed when the syndrome is strongly suspected[1,2].

Amiodarone-induced thyrotoxicosis (AIT) is an important cause of thyroid dysfunction in patients with cardiovascular disease. AIT is traditionally classified as type 1, related to iodine-induced excess hormone synthesis in an abnormal thyroid gland; type 2, a destructive thyroiditis caused by direct toxic effects on thyroid follicular cells; or a mixed/indeterminate form. Distinguishing these phenotypes is clinically relevant but can be difficult[3-5].

We report a case of severe AIT presenting as impending thyroid storm several months after amiodarone discontinuation in a patient with major structural heart disease and chronic anticoagulation.

Case Presentation

A 62-year-old woman presented to the emergency department with palpitations, dizziness, blurred vision, agitation and unintentional weight loss.

Her medical history was notable for heart failure with preserved ejection fraction (left ventricular ejection fraction 58%), mechanical mitral and aortic valve replacement performed more than a decade earlier, paroxysmal atrial fibrillation with recurrent episodes of rapid ventricular response, multinodular thyroid disease and previously diagnosed amiodarone-induced hyperthyroidism. Amiodarone had been discontinued several months before admission.

Her regular medications included sotalol 160 mg twice daily, bisoprolol 2.5 mg twice daily, warfarin, pitavastatin 1 mg daily and methimazole 5 mg twice daily.

On admission, the patient was restless and agitated, which made clinical interaction difficult, but she remained conscious, cooperative and fully oriented. She was hemodynamically stable and afebrile, with a heart rate of 119 beats/min. Cardiac examination revealed metallic prosthetic valve sounds without murmurs. Pulmonary examination was unremarkable, with preserved and symmetrical breath sounds. The abdomen was soft and non-tender. No peripheral edema was present and the neurological examination was normal.

Initial laboratory evaluation showed free thyroxine (FT4) of 24.4 pmol/L and thyroid-stimulating hormone (TSH) <0.01 mIU/L. Routine blood tests were otherwise unremarkable. The international normalized ratio (INR) was markedly elevated at 8.0, without clinically evident bleeding. An atrial flutter with rapid ventricular response was documented on admission. Chest radiography showed no acute abnormalities.

The working diagnosis was severe amiodarone-induced thyrotoxicosis with impending thyroid storm and a rapid atrial tachyarrhythmia. A history of prolonged amiodarone exposure was confirmed: 200 mg twice daily for approximately one year. Thyroid autoantibodies were negative. In the context of pre-existing multinodular thyroid disease and incomplete phenotypic data, a mixed or indeterminate AIT phenotype could not be excluded.

The treating team calculated a Burch-Wartofsky Point Scale (BWPS) score of 35, consistent with impending thyroid storm **(Table 1)**.

Table 1: Burch-Wartofsky Point Scale (BWPS) score documented in the clinical assessment.

Clinical parameter	Score
Agitation	10
Tachycardia (119 beats/min)	10
Atrial fibrillation	10
Precipitating factor	5
Total BWPS score	35

Interpretation: a total BWPS score of 35 falls within the range generally considered compatible with impending thyroid storm (25-44 points). The score shown reproduces the treating team's documented calculation.

Initial management included beta-blockade and rate-control therapy. Prednisolone 40 mg daily was started, with tapering initiated during hospitalization and continued thereafter. Propylthiouracil was initiated and antithyroid therapy was adjusted, together with supportive care and close clinical and biochemical monitoring.

The patient showed progressive clinical improvement during hospitalization, with normalization of thyroid hormone levels and adequate heart-rate control. She was discharged after two weeks in stable condition. Methimazole was discontinued and replaced by propylthiouracil, while prednisolone was continued with gradual tapering. Subsequent follow-up documented continued clinical improvement.

Discussion

Thyroid storm remains a clinical diagnosis because no single laboratory test reliably distinguishes it from uncomplicated thyrotoxicosis. Absolute thyroid hormone concentrations correlate imperfectly with clinical severity and the diagnosis therefore depends on the degree of systemic decompensation[1,2,6].

The Burch-Wartofsky Point Scale is widely used to support diagnostic assessment. Scores of 45 or higher are strongly suggestive of thyroid storm, scores below 25 make the diagnosis less likely and scores between 25 and 44 support impending thyroid storm[6].

In this case, the contemporaneously documented BWPS score of 35 supported early treatment for impending thyroid storm. The absence of fever, shock or overt multiorgan failure was consistent with a pre-storm state rather than fully developed thyroid storm.

AIT is classically divided into type 1 AIT, caused by iodine-induced excess thyroid hormone synthesis and type 2 AIT, caused by destructive thyroiditis with release of preformed hormone. Mixed or indeterminate presentations are well recognized and definitive subtype classification may be difficult in routine practice[3-5].

The present case illustrates this uncertainty. Negative thyroid autoantibodies did not support Graves disease, while pre-existing multinodular thyroid disease raised the possibility of an iodine-driven component. Because the available clinical documentation did not permit confident phenotypic separation, a combined therapeutic approach was clinically reasonable in the setting of severe presentation and substantial cardiovascular risk.

Management of severe thyrotoxicosis or thyroid storm should be initiated promptly. Treatment aims to inhibit thyroid hormone synthesis and release, reduce peripheral hormone effects, provide supportive care and address precipitating factors. Propylthiouracil can additionally reduce peripheral conversion of thyroxine (T4) to triiodothyronine (T3), while corticosteroids further decrease peripheral conversion and are particularly useful when a destructive component of AIT is suspected[1-3].

AIT remains particularly hazardous in patients with underlying cardiovascular disease because prolonged exposure to excess thyroid hormone increases the risk of arrhythmias, hospitalization and major cardiovascular events. Contemporary management therefore emphasizes rapid recognition, appropriate first-line therapy and consideration of thyroidectomy when medical treatment fails or cardiovascular instability is severe[5,7-9].

Amiodarone has a prolonged tissue half-life and clinically relevant thyroid dysfunction may emerge or persist after the drug has been withdrawn. Continued thyroid-function surveillance is therefore warranted after discontinuation, especially in patients with pre-existing thyroid abnormalities or major cardiac disease[3,5,10].

Conclusion

Amiodarone-induced thyrotoxicosis may present as impending thyroid storm even months after amiodarone discontinuation. In patients with structural heart disease, early clinical recognition, prompt combined medical therapy when subtype classification is uncertain and continued thyroid-function monitoring are essential to reduce morbidity and prevent progression to overt thyroid storm.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Informed consent for treatment and open-access publication was obtained or waived in accordance with institutional requirements.

Availability of data and materials

Not applicable. All data relevant to the case report are presented in the manuscript.

Conflicts of interest

The authors declare no conflicts of interest.

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Authors' contributions

All authors contributed to the conception and design of the report, acquisition and interpretation of clinical data, drafting and critical revision of the manuscript and approval of the final version. All authors agree to be accountable for the work.

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