A Challenging Case of Type 2 Amiodarone-Induced Thyroiditis

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Introduction

AIT can be a diagnostic challenge in clinical cases, where response to therapy is slow.

Type II Amiodarone-induced thyroiditis (AIT) is defined as the destruction of thyroid tissue due to inflammatory processes, secondary to the intrinsic drug effects of Amiodarone.

Type II AIT patients are more likely to be male, have a higher serum free T4/free T3 ratio and receive a higher cumulative dose of Amiodarone when compared to type I AIT patients (1).

Case Presentation

- A 51-year-old male patient initially presented with paroxysmal atrial fibrillation and was treated with oral Amiodarone.
- There was no personal nor family history of thyroid illness. He has a past medical history of congestive cardiac failure and his recent coronary angiogram showed 40% left anterior descending coronary artery stenosis.
- On second presentation, he reported a two week history of progressive breathlessness and lethargy.
- On examination, the patient exhibited features consistent with systemic thyrotoxicosis: fast atrial fibrillation, bilateral hand tremor, altered mentation, perfused sweating and goitre in his neck.

Investigation

Biochemical thyrotoxicosis was revealed: TSH <0.01 mIU/L, free T3 41.3 pmol/L and free T4 >77 pmol/L. He was negative for both TPO and TBII antibodies.

The patient scored **greater than 60** in the Burch & Wartofsky criteria for thyroid storm; a score greater than 45 is highly suggestive of thyroid storm.

Notably, an ultrasound scan of the thyroid revealed the thyroid to be diffusely enlarged with no increased vascularity. Subsequent 99mTc-methoxyisobutylisonitrile (MIBI) thyroid imaging found no visible thyroid uptake of the isomer (figure 1). These findings were highly suggestive of type II AIT.

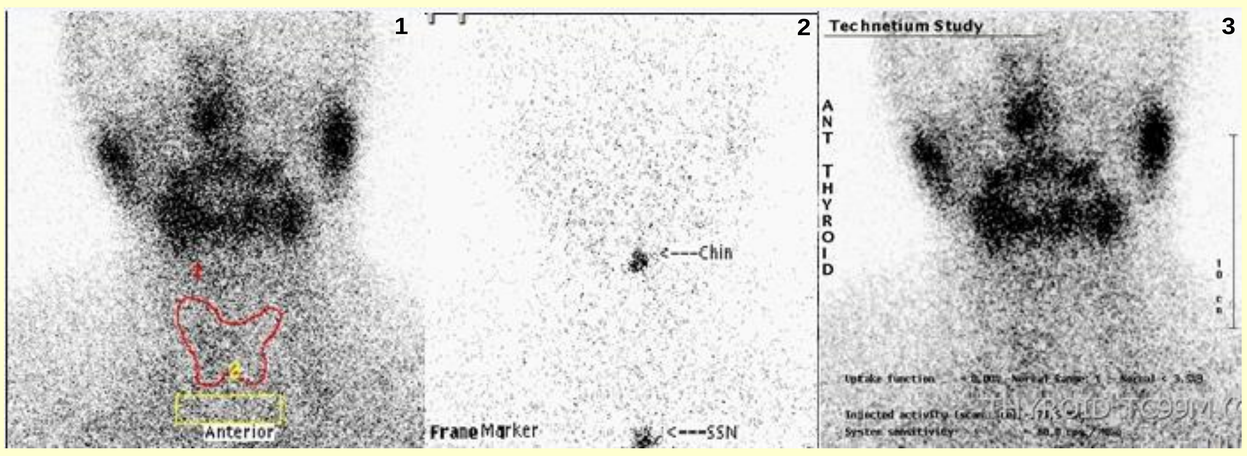

Figure 1. Thyroid uptake scan with 99mTc-methoxyisobutylisonitrile (MIBI) showed complete absence of contrast uptake

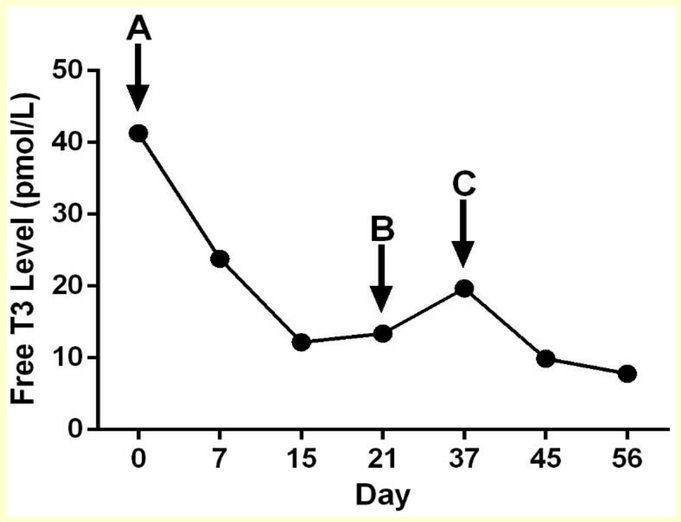

Figure 2. Levels of serum free T3 and respective treatment adjustments. A) Amiodarone was withheld and propranolol 20 mg QDS and prednisolone 30mg OD were commenced. B) Carbimazole 20mg OD was added to the regimen and propranolol increased to 40mg QDS. C) Carbimazole was increased to 20mg BD. Thyroid stimulating hormone was consistently less than 0.01 mIU/L from day 0 through to 56.

Management

On presentation, amiodarone was withheld immediately. Propranolol 20mg QDS and prednisolone 30mg OD were commenced (figure 2A).

The patient initially responded, but at 3 weeks following treatment commencement, free T3 remained raised at 13.4 pmol/L and symptoms persisted. The regimen was adjusted to carbimazole 20mg OD and propranolol 40mg QDS in addition to prednisolone (figure 2B).

Despite these changes, free T3 was 19.7 pmol/L at approximately 5 weeks and there was worsening of symptoms. Carbimazole was subsequently increased to 20mg BD (figure 2C). It is worth noting that 4 months of treatment was required before free T4 and T3 levels normalized.

Discussion

- Early identification and differentiation between various subtypes of AIT is crucial to its management (2).
- Amiodarone has a half-life of 120 days and withdrawal may not provide immediate results (3).
- Type 2 AIT patients may require prolonged treatment with thionamides and corticosteroids to achieve euthyroid status. Carbimazole is usually weaned off after 2 weeks, but this may not be possible due to relapse of AIT.
- Discontinuation of therapy is guided by close follow-up surveillance, monitoring free T3 levels and a thyroid uptake scan at 6 months.

References

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Thyroid

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