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CASE REPORT

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Total thyroidectomy in a patient awaiting heart transplant with amiodarone-induced thyrotoxicosis: A case report

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Abstract

Thyroid function may have a severe impact in cardiac function. Herein, we present the case report of a 53-year-old male patient awaiting heart transplant with amiodarone induced thyrotoxicosis that presented a marked improvement of his cardiac function after total thyroidectomy.

KEYWORDS

amiodarone, dilated-cardiomyopathy, heart transplant, thyrotoxicosis, total thyroidectomy

1 | INTRODUCTION

Amiodarone is a class III anti-arrhythmic widely used in the management of cardiac tachyarrhythmias,[1] particularly in the context of heart failure with reduced ejection fraction (HFrEF). In this subset of patients, it is the first-choice drug for rhythm control of atrial fibrillation (AF) and for suppression of further ventricular arrhythmias, reducing the burden of implantable cardioverter defibrillator (ICD) shocks.[2,3]

Amiodarone is an iodine-rich compound (about 37% of its weight) and the usual maintenance dose of 200 mg daily provides 75 times the daily requirement of iodine. It is lipid soluble and has a long half-life of about 100 days, mainly due to its storage to the adipose tissue.[4]

Amiodarone's adverse effects include thyroid dysfunction in up to 15% to 20% of cases, ranging from amiodarone-induced hypothyroidism to amiodarone-induced thyrotoxicosis (AIT). The incidence of AIT is up to 12% in areas with iodine deficiency (such as Portugal)

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João Sérgio Neves, Roberto Pinto, Sandra Amorim, Selma Souto, Paula Freitas and Davide Carvalho revised the article and added important inputs to the clinical history.

Changes in liver function tests.

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versus 2% in areas that are iodine sufficient.[5] In contrast to the other forms of hyperthyroidism, AIT is more frequent in males than in females.[6]

Two main forms of AIT may occur: type 1 AIT (AIT1) is a form of iodine-induced thyroid hormone production occurring in patients with underlying thyroid abnormalities, and type 2 AIT (AIT2) is a destructive thyroiditis that may occur in an otherwise normal thyroid gland (the most common).[7] AIT1 is traditionally treated with thionamides and AIT2 with oral glucocorticoids. Nevertheless, this classification is likely to be an oversimplification, with patients failing to respond to specific therapy and with AIT2 occurring in patients with goiter or Graves' disease. These mixed/indefinite forms of AIT are treated with thionamides and oral glucocorticoids can be added from the beginning if a precise diagnosis is uncertain, or after a few weeks if response to thionamides alone is poor. Due to the difficult diagnostic differentiation between AIT 1 and mixed/indefinite forms, a multipharmacological approach is often used.[8–10]

Amiodarone-induced thyrotoxicosis is linked to significant cardiac morbidity and mortality, especially in patients with HFrEF, owing to an overlap of preexisting impaired ventricular function and a high output thyrotoxic state, precipitating heart failure in these patients.[11,12] Also, AIT may exacerbate arrhythmic instability,[13] with documented cases of electric storm.[14] So, it is vital to assure a rapid restoration and maintenance of euthyroidism in such cases. However, pharmacological therapy does not always warrant effective and prompt reversion of hyperthyroxinemia. The need to continue amiodarone administration due to life-threatening arrythmias also imposes a long-term problem regarding AIT treatment and recurrence.[14]

Hence, definitive surgical management with thyroidectomy may be required in patients with severe thyrotoxicosis, refractory to medical treatment, if medical therapy is contraindicated or if amiodarone continuation is required. In the presence of rapidly deteriorating cardiac conditions, emergency thyroidectomy may be required for all forms of AIT.[15,16]

2 | CASE REPORT

We report a case of a 53-year-old male patient admitted to the Cardiology department of our hospital in July/2020 due to decompensated heart failure.

The patient has diagnosed 20 years ago with familiar dilated non ischemic cardiomyopathy, with progressive dilation and deterioration of systolic biventricular function and New York Heart Association (NYHA) functional class despite optimized prognosis-modifying therapies. One year ago, he was submitted to implantation of cardiac defibrillator; he was also submitted to mitral and tricuspid valve annuloplasties with prosthesis rings, without significant clinical improvement. He also had permanent atrial fibrillation (AF). The patient was referenced 8 months ago to the advanced heart failure outpatient consultation. Because of severe biventricular systolic dysfunction, with left ventricular ejection fraction (LVEF) of 20% and right ventricle fractional area change (FAC) of 32%, and persistent NYHA class of III+/IV despite medical treatment at a young age, we has included in active heart transplant waiting list 3 months ago. At his last appointment 1 month before, diuretic therapeutic was intensified due to aggravated systemic congestion.

His medications were lisinopril 2.5 mg qd, spironolactone 100 mg qd, bisoprolol 2.5 mg qd, furosemide 80 mg bid, dapagliflozin 10 mg qd, atorvastatin 40 mg qd and warfarin.

On the day of admission, the patient had a temperature of 36.6°C, blood pressure of 111/56 mmHg, respiratory rate of 26 breaths per minute, heart rate of 126 beats per minute (bpm), arrhytmic and irregular, and an oxygen saturation of 93% while breathing ambient air. Thyroid examination revealed no enlargement, tenderness, or bruit. Respiratory sounds were absent in the lower half of the right hemithorax and his legs showed bilateral edema.

Electrocardiogram revealed atrial fibrillation at 126 bpm with complete right bundle branch block (Figure 1). Echocardiogram showed, as previously described, aneurismatic dilation of the left atrium, severe dilation of right atrium and mild dilation of the right ventricle, normopositioned mitral and tricuspid prosthetic rings, with no obstruction of flow nor regurgitation, and severe biventricular dysfunction, with LVEF of 14% and FAC of 23%, pulmonary artery systolic pressure of 38 mmHg; with de novo left pleural effusion. Chest radiograph showed an elevated cardiothoracic index with large left pleural effusion. (Figure 2A,B) The blood work-up revealed a mild iron deficiency anemia (Hemoglobin of 11 g/dl, transferrin saturation of 12%), mild acute renal injury (Creatinine of 1.19 mg/dL) and an elevated brain-natriuretic peptide (733 mg/dL).

A cause of the acutely decompensated heart failure was then investigated.

The thyroid function tests revealed a thyroid-stimulating hormone (TSH) of 0.02 μUI/mL (normal values:0.35–4.94 μUI/mL), free triiodothyronine T3 (FT3) of 4.09 ng/dL (normal values: 1.71–3.71 ng/dL) and free thyroxine (FT4) of 2.46 ng/dL (normal values: 0.70–1.48 ng/dL), with negative anti-TSH receptor and antithyroid peroxidase antibodies.

Besides the persistent rapid ventricular response AF (without palpitations), there were no other signs or

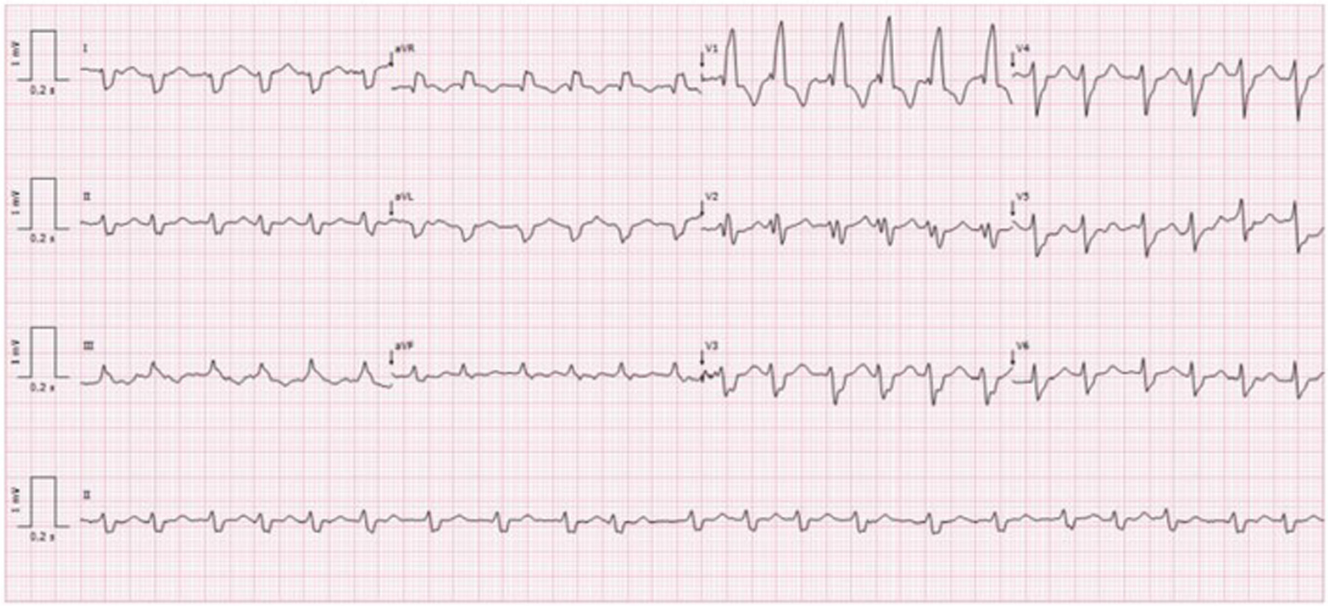


FIGURE 1 Electrocardiogram at admission showing atrial fibrillation, heart rate of 126 bpm with complete right bundle branch block

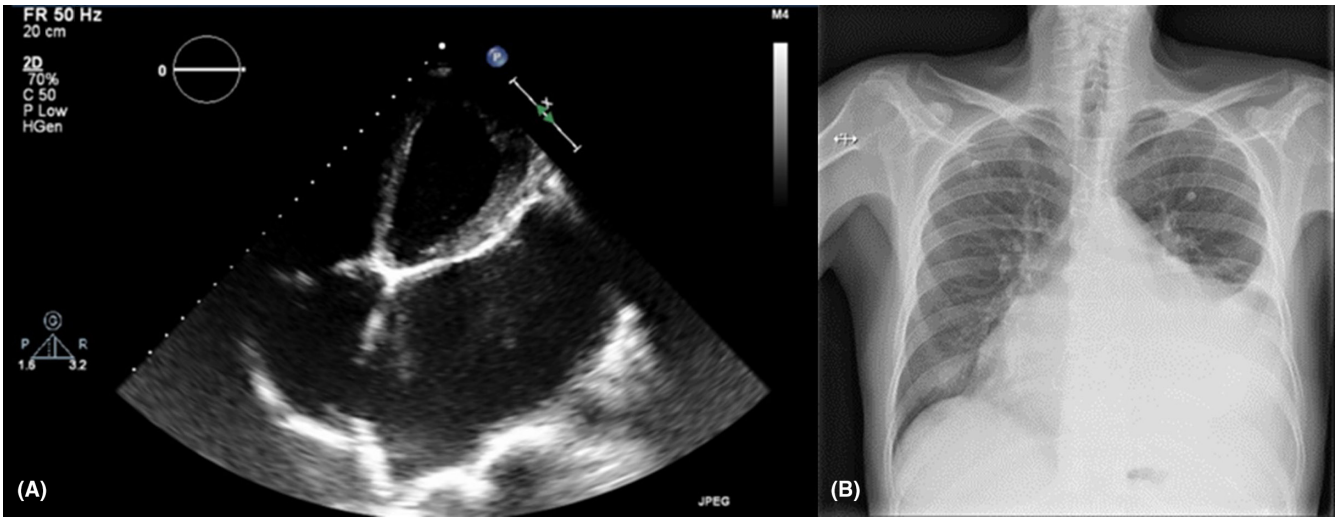


FIGURE 2 (A left) Apical four chamber view in transthoracic echocardiogram showing aneurismatic dilation of the left atrium, severe dilation of right atrium and mild dilation of the right ventricle, with normopositioned mitral and tricuspid prothesic rings; (B right) Chest radiograph revealing a significant elevated cardiothoracic index and large left pleural effusion

symptoms of hyperthyroidism. He had no history of familiar thyroid disease. Thyroid ultrasound revealed rare bilateral infracentimetric nodules, but the pattern of thyroidal vascularization was not described (Figure 3).

After revision of previous prescriptions, it was found that the patient took amiodarone (200 mg id) between May/2018 and December/2019, due to documented non sustained ventricular tachycardia (NSVT) in a Holter. His thyroid function at December/2019 was normal - TSH of 1.23 μUI/ml and FT4 of 1.21 ng/dl.

A diagnosis of mixed/indefinite AIT was assumed, and the patient initiated methimazole 10 mg bid progressively escalated to 20 mg bid, but thyrotoxicosis persisted. Given patient's very high surgical risk (cachexia, severe cardiac dysfunction and thyrotoxicosis status), we further attempted thyroid function control with solely medical treatment. For that reason, prednisolone 30 mg qd was added to the therapy 2 weeks later. Since the patient had several non-sustained and rare sustained ventricular tachycardias, and aiming to the rate control of atrial fibrillation, bisoprolol was switched to propranolol and titrated up to 40 mg 4id, with successful rhythm stabilization. After 3 weeks of medical therapy, the thyroid function persisted uncontrolled, and despite rhythm stabilization, the patient was

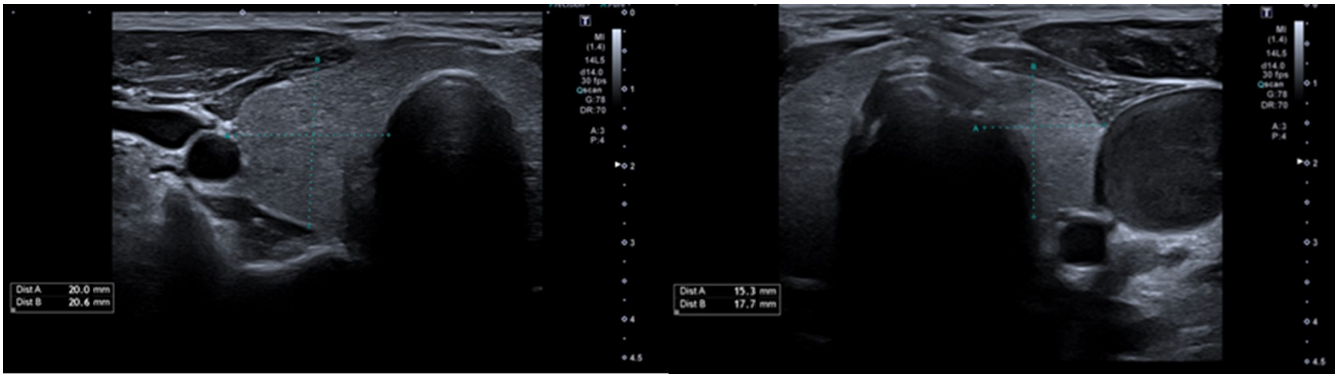
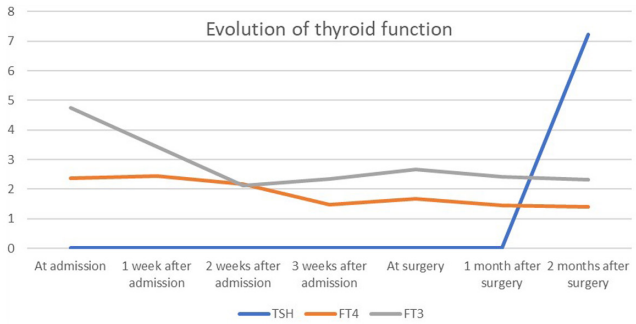


FIGURE 3 Thyroid ultrasound revealed rare bilateral infracentimetric nodules



GRAPH 1 Evolution of TSH, FT4 and FT3 since admission to post-thyroidectomy

still cachectic, progressively more congestive and at NYHA IV, with no conditions for discharge nor cardiac transplant. For that reason, after multidisciplinary discussion with the Advanced Heart Failure Cardiology team, Endocrinology Physicians and Anesthesiologists, it was decided to propose the patient to total thyroidectomy. The surgical procedure was uneventful, but postoperative period was complicated with a right cervical hematoma, requiring percutaneous drainage. The histopathological analysis of the surgical specimen revealed thyroid parenchyma composed predominantly by follicles that were multifocally interspersed with macrophages, compatible with amiodarone induced thyrotoxicosis. The patient was then discharged after clinical compensation and revision of ICD.

At ambulatory, with adjusted levothyroxine doses, the patient achieved euthyroid state 2 months after surgery (Graph 1), allowing is reentrance in cardiac transplant list. There was a marked clinical improvement to NYHA class II, with heart rate of 70–80 bpm, and reversal of the associated heart failure cachexia. At his last follow up echocardiogram, right ventricle systolic function remained impaired but there was a mild improvement in LVEF, to 32%. For these reasons, the patient was later transiently withdrawn from the cardiac transplant list (Figure 4).

3 | DISCUSSION AND CONCLUSIONS

We present a case of mixed/indefinite AIT triggering acute decompensated heart failure and rhythm instability, where medical treatment was not sufficient to achieve euthyroid state, requiring total thyroidectomy (Figure 5). The remarkable feature of this case is the extraordinary clinical improvement after normalization of thyroid function, allowing removal of active heart transplant list.

In a population of patients with HFrEF treated with amiodarone, Yamamoto et al encountered about 12% with AIT development.[17] Although uncommon, AIT is associated with three-fold increased risk of major adverse cardiovascular events; that is why its early diagnosis and prompt treatment are crucial.[10]

Thyroid dysfunction is a common event in amiodarone treated patients, so all patients should have TSH levels measured before starting therapy, at three- to four-month intervals during treatment and for at least 1 year after the amiodarone is discontinued.[9] This report illustrates a late AIT, happening 7 months after discontinuation of amiodarone. At any time, a diagnosis of AIT can also be considered in a patient who develops clinical signs of thyrotoxicosis.[15]

AIT manifests with clinical signs indistinguishable from spontaneous hyperthyroidism but may be inapparent or obscured by an underlying cardiac condition/therapeutic—an example are palpitations mitigated by beta blocker therapy.[15] On the other side, an exacerbation of an underlying cardiac disorder after amiodarone therapy, should prompt an investigation into thyroid function for suspected development of AIT, as was this case. Similarly, unexplained supratherapeutic levels of international normalized ratio (INR) can be a consequence of increased warfarin effects promoted by increased thyroid hormone levels.[18]

The challenging paradox of AIT is that often occurs in patients with HFrEF who presently require or previously

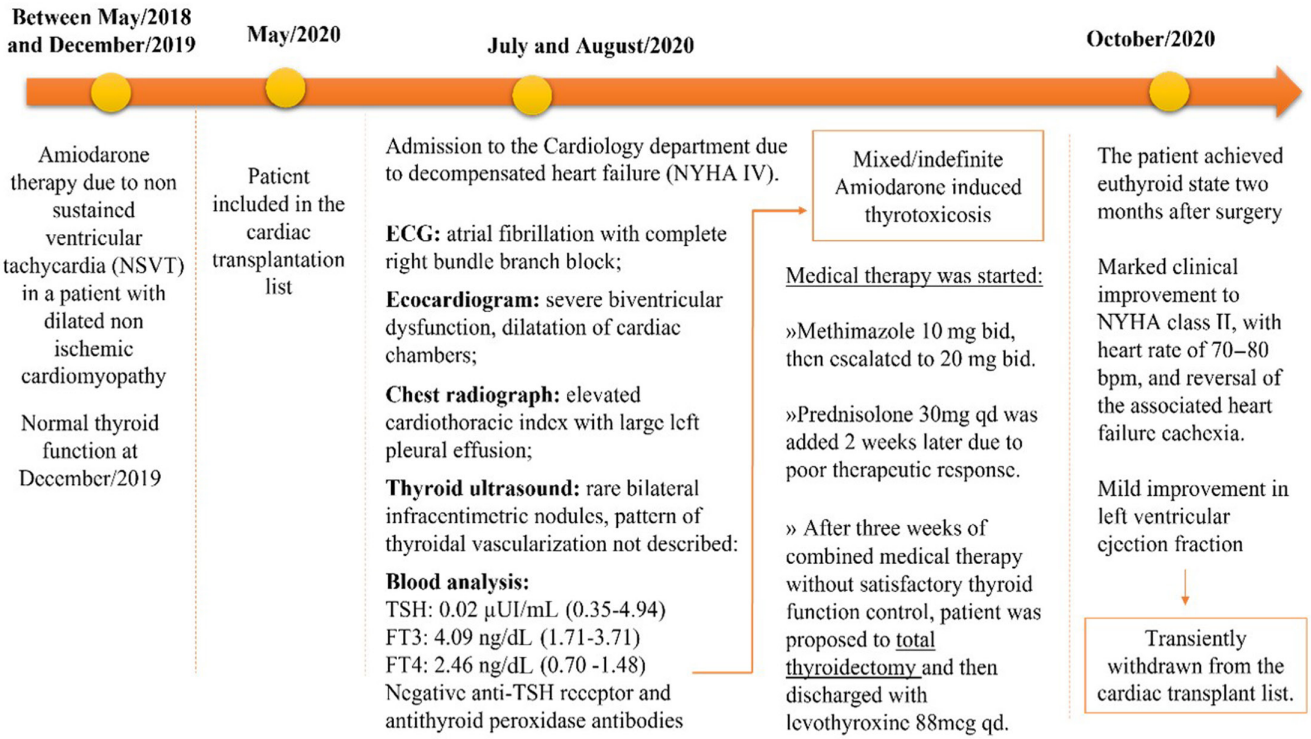


FIGURE 4 Timeline of patient findings and management

required amiodarone for life-threatening arrhythmias, nevertheless, are the least able to tolerate a thyrotoxic state.

The effects of amiodarone can persist for up to eight to 9 months after discontinuation, as a result of its long half-life and storage time, allied to its slow release and of its main metabolite (desethylamiodarone) from the adipose tissue.[19] This fact explains why our patient presented thyrotoxicosis 7 months after amiodarone discontinuation, as previously documented in other reports.[20,21]

Consequently, as no immediate effects are expected after discontinuation of amiodarone, the decision to continue or to stop this drug after AIT diagnosis should be individualized, shared between the cardiologists and endocrinologists, taking into account patient's arrhythmic risk.[9]

In the presented case, rhythm stabilization was achieved with beta blocker, after switching bisoprolol to propranolol. However, some of these patients with HFrEF who took amiodarone for suppression of further ventricular arrhythmias/ICD shocks, with superimposed AIT may experience additional arrhythmic instability, requiring adjuvant anti-arrhythmic drugs (AAD) other than beta blockers. The key point is that amiodarone is the safest anti-arrhythmic drug in this subset of HFrEF patients, as others AAD are contra-indicated, such as sodium channel blocking agents, lidocaine, mexiletine, sotalol, verapamil and diltiazem.[22] As so, it is widely accepted that amiodarone should be continued in critically ill patients with life-threatening cardiac disorders responsive to the drug.[9] To the date, there is conflicting data regarding if continuation of amiodarone is related or not to delay of restoration of thyroid function with medical treatment, and if it is associated or not with greater recurrence of thyrotoxicosis.[23–26]

The thyrotoxic state in HFrEF patients also leads to aggravated congestive status, combined with pulmonary hypertension, tachycardia, further reduction in ventricular contractility and diastolic filling.[27] In a patient with biventricular severe dysfunction as ours, this congestive and low output status may be extremely difficult to tolerate and compensate as thyrotoxicosis persists. Nevertheless, drug therapy tapering with oral glucocorticoids and/or thionamides may take up to six to 8 weeks; and HFrEF patients may also have a low tolerance for high-dose steroids. Hence, some HFrEF patients with AIT and severe cardiac impairment, such as refractory congestive heart failure or arrhythmic instability requiring amiodarone, cannot wait for the period required for effective anti-thyroid treatment. For these patients, a thyroidectomy allows a rapid normalization of thyroid function.[10]

Therefore, definitive surgical management with thyroidectomy may be required in patients with severe thyrotoxicosis, refractory to medical treatment, if medical therapy is contraindicated or if amiodarone continuation is required. In the presence of rapidly deteriorating cardiac

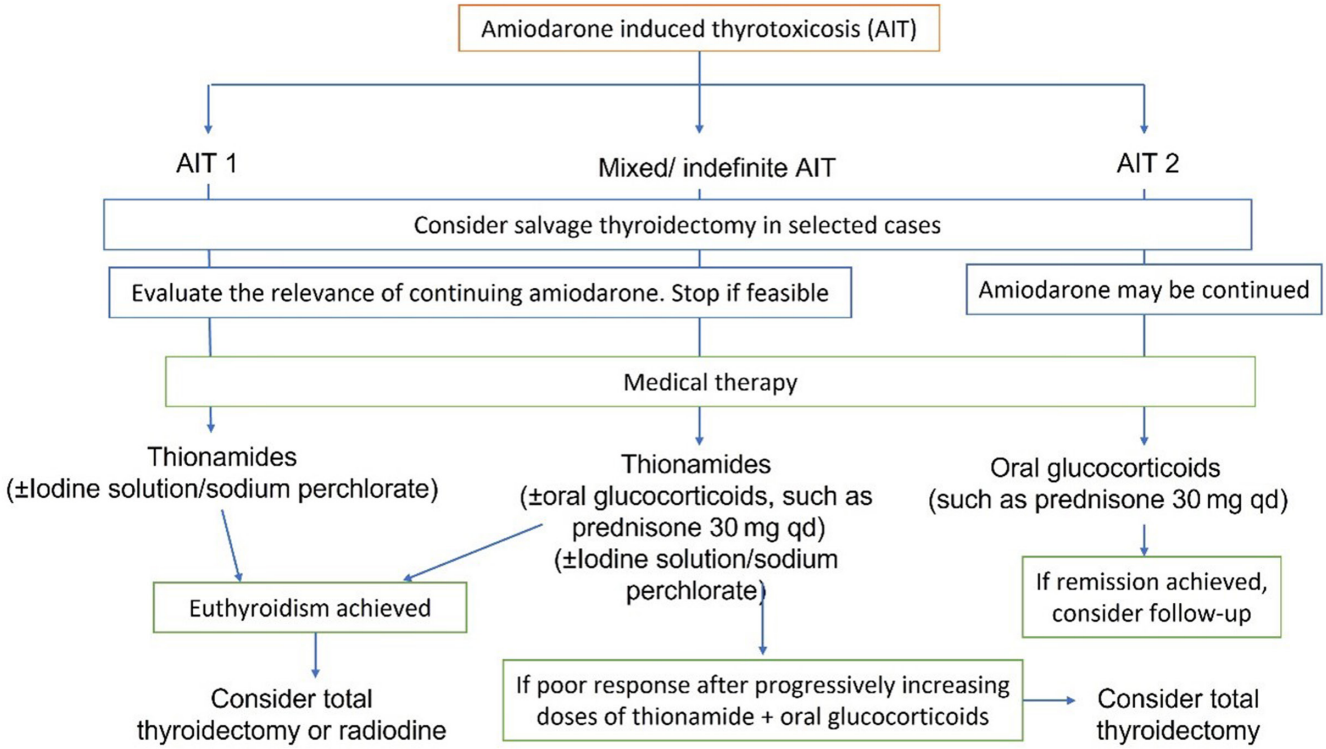


FIGURE 5 Treatment of amiodarone induced thyrotoxicosis (Adapted from reference 9.)

conditions, emergency thyroidectomy may be required for all forms of AIT.[15,16]

Traditionally there has been concern that thyroidectomy is associated with excess peri-operative risk in HFrFE. However, there are now some reported cases of surgically managed AIT, the majority (about 70%) with concomitant severe cardiac disease, and they overall state that thyroidectomy is safe in patients with severe cardiac failure if performed in a center with cardiac anesthetic expertise.[17,28–30]

Thyroidectomy has been shown to be effective in the rapid reversal of hyperthyroidism as well as in the improvement of cardiac function in patients with left ventricular dysfunction related to thyrotoxicosis. Moreover, this surgery may enable cardiac transplant in those who would otherwise not be considered suitable due to presence of uncontrolled thyrotoxicosis.[19,20]

After thyroidectomy, there is a marked clinical improvement and previous series also encountered patients whose improvement was so significant that were removed from cardiac transplant waiting list.[31]

After total thyroidectomy (and levothyroxine introduction), physicians must evaluate thyroid function regularly, with the first perioperative thyroid analysis being scheduled to 4–6 weeks after levothyroxine introduction. The dosage of this medication should be progressively adjusted to achieve euthyroidism (normalization of TSH and FT4). If the patient remains euthyroid in subsequent evaluations with the same levothyroxine dosage, thyroid function can be evaluated annually. Caution should be taken to iatrogenic elevation of thyroid hormones, leading to recurrence of a hyperthyroid state, again with the cardiac impairment associated (congestive heart failure and arrhythmic instability).[32] In this particular subset of HFrEF patients submitted to thyroidectomy, one of the most relevant long-term complications is permanent hypoparathyroidism after damage to the parathyroid glands, which incidence varies from 0% to 11% in previous reports.[28–30] Chronic hypoparathyroidism can be diagnosed in patients maintaining inadequately low parathyroid hormone levels 6 months postoperatively.[33] The resulting hypocalcemia may induce ST segment modification and QT interval prolongation and, when severe (serum calcium < 1.9 mmol/L and/or symptomatic), can predispose to life-threatening ventricular arrhythmias.[34,35]For that reason, it is reasonable to monitor serum calcium and parathyroid hormone concentrations 6–8 h and also 24 h after surgery in these patients, in order to make a timely diagnosis and to treat an eventual hypoparathyroidism (that should be monitored regularly after the patient's discharge).[36]

In summary, a careful history and a high index of suspicion are needed to correctly diagnose an amiodarone-induced thyrotoxicosis. In advanced heart failure patients with this thyrotoxicosis, in whom medical treatment is not rapidly effective, the total thyroidectomy

may be the key to achieve clinical stabilization and improve prognosis.

AUTHOR CONTRIBUTIONS

FM and FA analyzed and interpreted the patient data regarding the thyroid and cardiac disease, and contributed equally to the manuscript writing. CC, CM, JSN, RP, SA, SS and PF were the doctors of the presented patient (along with FM and FA), contributed to the elaboration, have read, and approved of the manuscript. DC contributed to the elaboration, read and approved the final manuscript

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CONFLICT OF INTEREST

The authors declare that they have no competing interests

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

CONSENT

The patient signed informed consent and allowed the publication of this case.

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