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Surgery in Refractory Amiodarone-Induced Thyrotoxicosis

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Abstract

Background: Thyroidectomy is a challenging treatment for refractory amiodarone induced thyrotoxicosis (AIT).

Objectives: The authors' aim in this article is to conduct a systematic review of the currently available literature regarding thyroidectomy for the treatment of refractory AIT.

Methods: The authors' systematic review yielded 14 studies encompassing 39 patients.

Results: All patients in this study developed thyrotoxicosis on amiodarone especially those with cardiac fragility and benefit from thyroidectomy to control all the symptoms of the hyperthyroid state without surgical risk or consequence on the cardiac status.

Conclusion: Thyroidectomy remains the definitive management of refractory AIT and should be instituted sooner rather than later in a patient suffering from this condition.

Introduction

Amiodarone is a class III anti-arrhythmic drug used to manage different cardiac problems, but its high iodine content and its direct toxic effect may cause thyroid dysfunction. Thyrotoxicosis is a harmful side effect of amiodarone use. Management of AIT is usually resistant to conventional methods and require prompt resolution of thyrotoxicosis-related cardiac decomposition. Definitive treatment can include surgery of the thyroid gland.

So here comes many studies about the safety and utility of thyroidectomy as definitive way to treat AIT and restore thyroid function.

The purpose of the present study was to systematically review the existing recent data regarding total thyroidectomy for the treatment of refractory AIT.

Methods

A review of the existing published data on thyroidectomy for the treatment of amiodarone induced thyrotoxicosis performed using PubMed for articles published in English. The terms used included amiodarone, thyrotoxicosis, thyroidectomy, amiodarone induced thyrotoxicosis. The search was not limited to any date range. 38 articles were identified and a total of 14 remained after duplicates were removed. 11 cases, 3 studies of case series reporting total thyroidectomy as definitive treatment for amiodarone induced thyrotoxicosis especially in those refractory to medical management.

Results

A total of 14 studies were included in this review article. All were published between 2002 and 2016 from different countries.

We have in total 39 patients, 23 were males, 16 were females and the age range varies between 32 and 82.

All patients have heart problems and started amiodarone for atrial fibrillation, ventricular tachyarrythmias or dilated ischemic cardiomyopathy with heart failure and all these cardiac conditions were refractory to the usual medical or interventional therapy (drugs, implantable cardiac devices or even radiofrequency ablation).

The usual dose of amiodarone present is 200 mg daily and the duration of amiodarone use extends from 6 months to 4 years and most of patients were admitted for thyrotoxicosis state ranging from recurrent rapid atrial fibrillation, decompensated heart failure to even thyroid storm which is reported in one case.

Laboratory tests in the 11 cases showed an elevated free or total thyroxine and triiodothyronine and suppressed TSH levels. In the 3 case-series, thyroid function tests were not identified. The reasons for intervention were failure of therapy to control thyrotoxicosis, persistence and deterioration of clinical symptoms and appearance of drug's side effects secondary to high doses used.(Table 1)

The definitive mechanism by which amiodarone has contributed to thyrotoxicosis, whether type 1, type 2 or both, has been identified in most of the patients; in the 11 cases, 7 had type II AIT confirmed by either Doppler ultrasound which showed hypovascularization or by pathological features, 1 patient had type I AIT revealed by thyroid gland hypervascularization in addition to nodular goiter seen macroscopically and in 3 patients the exact mechanism was unknown. In the case –series of, all patients had destructive thyroiditis seen on pathology, in Lorberboym et al, type II AIT was established in the 11 patients and the type of AIT has not been mentioned [10,12,14]. 2 patients out of 39 had toxic MNG with pathology confirming type II AIT (they may have mixed disorder) and 1 patient had nodular goiter with unknown pathology. .(Table 2)

Table1: Summary of case reports for 11 patients with amiodarone-associated thyrotoxicosis who were treated successfully with total thyroidectomy

Study	Age and Sex	Medical history	Reason for starting amiodarone	Amiodarone dose
Mehta et al. 2008(1)	66 year-old man	DM, CAD, CVA, HTN	CABG complicated by AF	800 mg for one week then 200 mg daily
Cunha et al.2016(2)	52 year-old man	Negative	Acute MI, complicated by AF (patient refused radiofrequency ablation).	300 mg daily
Hashimoto et al.2015(3)	40 year-old man	DCMP	DCMP on amiodarone for 2.5 years	NA
Ishay et al.2013(4)	48 year-old man	Non obstructive HCMP, CHF, CAD, MI, DM, CKD (3A), COPD, recurrent CVA, paroxysmal AF	Persistant episodes of AF treated with amiodarone since 5 years	NA
Kotwal et al.2015(5)	61 year-old Caucasian man	AF, CAD, ischemic CMP, CHF, DM.	AF and CHF	NA
Batori et al.2006(6)	65 year-old woman	CMP (due to moderate-severe aortic regurgitation) complicated by VA and FA. Multiple nodular formations in thyroid lobes (treated with MTZ 5mg/ day)	VA and FA	200 mg daily
Calis et al.2010(7)	46 year-old man	Idiopathic DCMP Sustained VT, VF (treated with radio frequency ablation and ICD)	Persistent episodes of VT and VF	NA
Tonnelier et al.2013(8)	62 year-old caucasian man	AF since 4 years AI grade 1-2/4	AF	200 mg daily for 2 years
Gavira et al.2013(9)	51 year-old man	Obstructive HCMP, AF	AF	200 mg daily since 4 years
Marinis et al.2013(10)	52 year-old man	Recurrent AF	Uncontrolled AF (despite cardioversion, propafenone, sotalol and catheter ablation)	200mg daily for 6months
Zhu et al.2016(11)	56 year-old Chinese man	AF	AF	200mg daily for 2 years
Study	**Reason for Intervention**	**lab tests**	**Ultrasound thyroid+echocardiography**	**Treatment of AIT**
Mehta et al. 2008(1)	SOB, palpitations, AF with rapid ventricular rate	TSH 0.008 TT3 5.61 TT4 28.4	Thyroid gland enlarged with diffuse heterogeneity, no Doppler evidence of increased vascularity	MTZ 10mg TID later increased to 15mg TID then replaced by PTU 1000mg loading then 250 mg every 4hours dexamethasone 2mg every 6hours
Cunha et al.2016(2)	-TFT monitoring (after 1 year) -2months after, recurrent AF	TSH 0.01 FT4 4.06 FT3 9.23. **After 2 Months** TSH 0.03 FT4 1.49 FT3 3.22	-EF 34%, mild atrial dilatation	Bisoprolol 5 mg daily, MTZ 10 mg twice daily and prednisolone 5 mg daily

Hashimoto et al.2015(3)	symptoms of CHF	TSH <0.05 FT4 3.39 FT3 6.61	-Thyroid gland was not swollen, slightly enlarged, with monotonous echogenicity. The Doppler flow was not increased	Inorganic iodine administration (189mg/day), Hydrocortisone 200 mg replaced by prednisolone 40mg, increased to 60 then 80mg, MTZ 15mg
Ishay et al.2013(4)	symptoms of CHF	TSH <0.03 FT4 5.8 FT3 8.39	-EF 50% with grade 2 diastolic dysfunction and enlarged left atrium	MTZ 40mg/day replaced by PTU 800 mg/day, prednisone 40mg/day replaced by dexamethasone
Kotwal et al.2015(5)	SOB, palpitations, tremor, generalized weakness	TSH 0.02 FT4 4.88 FT3 5.4	-Bilaterally heterogeneous, hypovascular and hypoechoic thyroid gland -EF 25%	Prednisone 60mg and methimazole 40mg replaced by PTU 200mg TID, lithium, 8 daily cycles of plasmapheresis, with increasing volumes of plasma exchange
Batori et al.2006(6)	Symptoms of hyper functioning MNG: Weight and hair loss, insomnia, nervousness	TSH 0.97 FT4 1.81 FT3 2.36	-Thyroid gland moderately enlarged with colloidal-cystic nodules, the largest of 1 cm in the isthmus, solid, isoechogenic, with thin rarefaction halo of the echoes -Moderately dilated left ventricular with hypertrophy, EF 35-40%, moderate-severe aortic valvular regurgitation, light-moderate mitral insufficiency and light tricuspidal insufficiency	MTZ 5mg/day replaced by PTU 50mg/day
Calis et al.2010(7)	Fine tremor and tachycardia.	TSH <0.005 FT4 >7.76 TT3 1.62	-Left ventricular dilatation, EF 30%, mild mitral regurgitation.	PTU, sodium per chlorate, prednisone, metoprolol
Tonnelier et al.2013(8)	Tremor, heat intolerance, excessive sweating, weight loss, palpitations, SOB	TSH < 0.015 FT4 > 3.1 FT3 19.2	-Diffusehypo-echogenic heterogenous gland. Absence of hypervascularity -Normal EF	Methylprednisolone 32mg/day, MTZ 30mg then 60mg/day, potassium per chlorate 1g/day, sotol 240mg/day, 6 cycles of plasmapheresis
Gavira et al.2013(9)	New episode of AF, decompensated CHF, nervousness, palpitations, weight loss	TSH 0.008 FT4 7.5 FT3 10.99	-Generalized hypovascularization in thyroid gland	carbimazole 45 mg/d, prednisone 60 mg/d, propranolol 60 mg/d, Lugol's solution 5 drops BID, cholestyramine 12 g/d, 10 sessions of plasmapheresis, iopanoic acid 500 mg BID
Marinis et al.2013(10)	Tachycardia progressively leading to CHF	TSH <0.01	-Slightly enlarged thyroid, with heterogeneous echogenity and increased vascularity -EF 30-35%	carbimazole 40mg TID, prednisolone 16mg daily, propranolol 100mg BID

Zhu et al.2016(11)	Fever (40°C), SOB, rapid AF, hypotension (thyroid storm)	TSH <0.015 FT4 4.97 FT3 11	-Normal EF	propranolol 20 mg TID replaced by esmolol infusion up to 200 **µg**/kg/min, hydrocortisone 100 mg every 6 hours, intravenous digoxin 500 **µg** once, PTU 400 mg, Lugol's iodine 10 ml, cholestyramine, plasmapheresis
Study	**PRE-Thyroidectomy**	**POST-Thyroidectomy**	**Pathology**	
Mehta et al. 2008(1)	Despite maximal medical therapy, FT4 and TT3 continued to increase and TSH remained below 0.03	Clinically and biochemically euthyroid 10 days after thyroidectomy	The gland was soft, not hypervascular. The epithelium in many of the follicles was degenerated or partially denuded, with vacuolated cytoplasm and pyknotic nuclei. Aggregates of foamy histiocytes were found within the injured follicles, as well as in the adjacent interstitium. These changes are typical for amiodarone-induced thyroid injury	
Cunha et al.2016(2)	Electrical cardio version performed then radiofrequency ablation was proposed for the recurrent AF but the patient refused.	TFT improved gradually in the first week and the patient became asymptomatic. Control echocardiography was normal, with EF of 59%.	NA	
Hashimoto et al.2015(3)	Overt thyrotoxicosis was not controlled despite 2.5 months of steroids and anti-thyroid drugs	After surgery, the patient's thyrotoxicosis rapidly disappeared, EF ameliorated and TFT normalized with 100 μg of levothyroxine	Findings characterized by scattered follicle disruption, vacuoles in epithelial cells, and macrophage infiltration, compatible with amiodarone toxicity	
Ishay et al.2013(4)	Persistant symptoms of decompensated CHF (recurrent pulmonary edema with episodes of supraventricular and ventricular tachycardia)	Within 2 weeks of thyroidectomy, patient developed hypothyroidism and started on levothyroxine	Macroscopically: small and fibrotic diffuse goiter Microscopically: thyroid follicular cells were degenerated with picnotic nuclei. Infiltration with foamy histiocytes was noted	
Kotwal et al.2015(5)	Reccurent symptoms with poor response to medical therapy and increased cardiovascular risk	Within 24 hours, his FT4 and FT3 levels decreased. On follow-up 2 months later, he remained clinically euthyroid on levothyroxine and maintained a stable cardiac status.	Macroscopically: a hard thyroid on both sides with severe fibrosis and adhesions especially over the trachea. Microscopically: intra-follicular histiocytes, patchy fibrosis and involuted follicles, suggestive of destructive thyroiditis	
Batori et al.2006(6)	No amelioration depite anti-thyroid drugs, reccurent symptoms of CHF	Amelioration of thyrotoxic state	NA	

Calis et al.2010(7)	No improvement in TFT (TT3 and FT4 remained high)	Within several weeks, TFT improved and the patient was started on levothyroxine.	NA
Tonnelier et al.2013(8)	Worsening of symptoms	Rapid improvement of symptomatology after thyroidectomy. Levothyroxine was prescribed at day 28 post-surgery	NA
Gavira et al.2013(9)	Worsening of hyperthyroidism (TSH 0.008, FT4 >7.7 and FT3 15.14), uncontrolled atrial flutter	After 12 months of follow-up on treatment with levothyroxine 125 mcg/ day, the patient has experienced no new episodes of tachyarrhythmia.	Normal sized thyroid gland with no nodules, involution of thyroid follicles, and degenerative changes.
Marinis et al.2013(10)	Persistant tachycardia and abnormal TFT	2 weeks after thyroidectomy, the patient experienced better tolerance of physical activity, reduced his β-blocker regimen and demonstrated a higher EF (50%). One month postoperatively, the patient has discontinued his β-blocker and has returned to normal daily activity.	Nodular goiter with follicular hyperplasia
Zhu et al.2016(11)	Refractory tachycardia, therapeutic options were limited by the severe derangement of liver function.	After thyroidectomy, there was a significant drop of FT4, FT3 and heart rate. 12 months after discharge, his TFT were stable on thyroxine replacement.	Microscopically, there was a predominance of colloid containing thyroid follicles. Several features compatible with type II AIT were seen, including foamy histiocytes, vacuolated desquamated epithelial cells and multinucleated giant cells

DM: diabetes mellitus CAD: coronary artery disease CVA: cerebrovascular accident AF: atrial fibrillation
CABG: coronary artery bypass graft MI: myocardial infarction DCMP: dilated cardiomyopathy CHF: congestive heart failure CKD: chronic kidney disease COPD: chronic obstructive pulmonary disease CMP: cardiomyopathy
VA: ventricular arrhythmia MTZ: methimazole VT: ventricular tachycardia VF: ventricular fibrillation AI: aortic insufficiency SOB: shortness of breath TFT: thyroid function test MNG: multinodular goiter NA: not available
TSH (thyroid stimulating hormone) :0.35-5.5 mIU/l
TT4 (total thyroxine) : 4.5-12 mcg/dl
TT3 (total triiodothyronine) : 0.6-1.81 ng/ml
FT4 (free thyroxine) : 0.61-1.12 ng/dl
FT3 (free triiodothyronine) : 2.5-3.9 pg/ml

Table2: Summary of case-series for 5 patients with amiodarone-associated thyrotoxicosis who were treated successfully with total thyroidectomy

study	Age +sex	Past medical history	Reason for starting amiodarone	Reason for intervention	Duration of amiodarone use	Treatment of AIT	Pre-Thyroidectomy	Post-Thyroidectomy	Pathology
Gouch et al.2002(12)	82 year-old female	Toxic MNG treated with carbimazole for 20 years	AF	thyrotoxicosis	4 months	PTU, prednisone, potassium per chlorate.	Failure of therapy to control thyrotoxicosis	patients recovered rapidly and remain well and euthyroid on thyroxine replacement	Destruction of follicles, and macrophages in the colloid. Marked inflammatory cell infiltration. Fibroblasts creating fibrous tissue.
Gouch et al.2002(12)	39 year-old man	4 operations for tetralogy of Fallot and on waiting list for cardiac transplantation	Recurrent VT	worsening VT due to thyrotoxicosis(requiring automatic implanted cardiac defibrillator)	3 years	carbimazole, prednisone, potassium per chlorate and lithium carbonate	Failure of therapy to control thyrotoxicosis	Same as before	Same as before.
Gouch et al.2002(12)	32 year-old man	Familial DCMP	recurrentVT	Thyrotoxicosis for 18 months. CHF : EF 4%	4 years	PTU, potassium per chlorate and lithium carbonate.	Failure of therapy to control thyrotoxicosis	Same as before	Same as before
Gouch et al.2002(12)	61 year-old man	Rheumatic heart disease with aortic valve replacement and congestive cardiac failure	Ventricular tachycardia and fibrillation	Thyrotoxicosisdeveloped two months after amiodarone was discontinued.	two years	propylthiouracil, prednisone and lithium carbonate.	Failure of therapy to control thyrotoxicosis	Same as before	Same as before
Gouch et al.2002(12)	63 year-old man	CABG and cardiac pacemaker.	FA	Thyrotoxicosis developed 3 months after amiodarone was discontinued	2 years	carbimazole and prednisolone	Failure of therapy to control thyrotoxicosis	Same as before	Same as before

Many trials were conducted to control the hyperthyroid state, from anti-thyroidal drugs (high doses of thionamides (60mg/day) and propylthiouracil (1200 mg)) to high doses of corticosteroids, iodine solution, cholestyramine and even plasmapheresis, without any benefit. In all cases, thyrotoxicosis was refractory to these different treatment modalities, unresponsive with worsening of the underlying cardiac conditions in several cases and the only solution remains total thyroidectomy whether they had type I (underlying thyroid pathology) or type II AIT (destructive thyroiditis). .(Table 3)

All patients ameliorated after thyroidectomy and restored the euthyroid state with improvement of symptoms and ventricular function. Only one died after surgery but this is secondary to his preexisting comorbid conditions.

Table3: 2 case-series presenting patients who underwent successful thyroidectomy for the treatment of amiodarone-associated thyrotoxicosis

Study	Number of patients	Age +sex	Reason for intervention	Duration of amiodarone use	Treatment of AIT	PRE-thyroidectomy	POST-thyroidectomy	Pathology
Lorberboym et al.2007(13)	11 patients	Sex:6 men and 5 women	5patients:reccurrence of AF	1 to 12 months(mean:7months)	All patients: High doses of PTU up to 1200 mg/day	4 patients did not respond to medical treatment after 3 months	There was a rapid correction of thyrotoxicosis. Subsequently, all patients recovered rapidly and remained well and euthyroid on thyroxine after 12 months of follow-up replacement therapy	In all cases thyroid histopathology demonstrated degenerative and destructive follicular lesions with multinuclear cell infiltrate and focal fibrosis.
					7patients:beta-receptor antagonist	3 patients were treated with high doses of steroids, one of them with PTU and ipodate without therapeutic effect		
			5patients:ventricular premature beats and deterioration of clinical condition under amiodarone treatment			1 patient developed marked impairment of hepatic function. Therapy was stopped and the patient had severe decompensated CHF with tachycardia unresponsive to medical treatment.		
		Age: 63 to 82 years			5patients: prednisone 60mg/day	1 patient had large toxic MNG treated with MTZ for 20 years. He began amiodarone treatment for AF and developed thyrotoxicosis after 4 months.		
			1patient:severe CHF			-2 patients chose surgery		
					3 patients: high doses of steroids, one of them with PTU and ipodate			
Drescher et al.2006(14)	12 patients	Sex:3men and 9 women	All patients had underlying cardiac disease(4 CAD, 2 DCMP, 1 valvular heart disease, 3 HTN, 1 arrythmia, 1unkownbut on ICD)	1year to 4 years(mean 2.9 years)	Thionamides, glucocorticosteroids or both administered for 3-10 weeks	Unresponsiveness to medical therapy and worsening of the underlying cardiac conditions in several cases	Euthyroidism was restored quickly in all subjects. Temporary intensive care was required for 3 patients. One died after surgery because of multiple preexisting comorbid conditions, All other were euthyroid after levothyroxine replacement and on stable or improved cardiac condition after at least 1 year	NA
		Age:49-81(mean 60)	8 had ICD 1					
			7 out of 12 had EF of 35% or below (the rest between 45 or 50%)					

Discussion

Amiodarone is a widely used anti-arrhythmic drug treating mainly supraventricular and ventricular tachycardia's, used safely in patients with atrial fibrillation and heart failure in order to obtain sinus rhythm without affecting ventricular function.

However, like any other drug, its benefit can be out weighted by numerous side effects especially that most individuals administered it on a chronic basis so they will experience definitely at least one side effect among others. The most hazardous one is thyroid dysfunction (hypo or hyperthyroidism) as amiodarone is an iodine rich compound and similar structurally to thyroid hormone. (1)

Some patients on amiodarone remain euthyroid but others develop thyrotoxicosis and have consequently higher adverse cardiovascular events.

Amiodarone-induced thyrotoxicosis (AIT) is a serious complication of long-term amiodarone use. It occurs in patients with already established cardiac dysfunction on amiodarone, who didn't tolerate the hyperthyroid state. It is classified as iodine-induced thyrotoxicosis (type 1) existing in patients with underlying thyroid disease exacerbated by thyroid autonomous function, mainly in iodine-deficient areas and destructive thyroiditis (type 2) in those with no history of thyroid problems causing direct cytotoxic and inflammatory effects, occurring in iodine-repleted areas. [4] Mixed forms also are present. Those effects are probably due to high iodine content of amiodarone, and its long half-life (107 days). [2]

Changes seen microscopically in type I AIT are consisting of preexisting thyroid disease, usually multinodular goiter or underlying latent Graves disease, in whom iodine exposure triggers the development of clinical disease. This subgroup usually has normal or only slightly elevated serum levels of interleukin-6, while patients with type 2 AIT have randomly distributed disrupted follicules, filled with desquamated vacuolated epithelial cells, foamy macrophages and lymphocytes, involutional changes and fibrosis. They have higher levels of IL-6, rendering it a good marker of the thyroid-destructive processes. [13] Thyroid scan and doppler ultrasound can differentiate also between the 2 types. Absence of tracer uptake in the thyroid bed and Doppler flow demonstrate type 2 AIT.

The exact type of thyrotoxicosis cannot be identified most of the time and treatment for both conditions is given. [2] Mixed cases of amiodarone induced thyrotoxicosis occur in 15 to 27% of cases. [4]

In patients with preexisting cardiac disease, the effect on heart is more worrisome. When established, AIT worsens the underlying arrhythmia and most patients developed decompensated heart failure, recurrent atrial fibrillation or any arrhytmia because of the superimposed hyperthyroid state. [3]

Hyperthyroidism may be mild (free thyroxine (T4) levels 1 to 1.5 times the upper limit of normal), moderate (free T4 levels 1.5 to 2 times the upper limit of normal) or severe (free T4 levels approximately 2 to 3 times the upper limit of normal).

AIT is a challenging problem difficult to manage with substantial morbidity and mortality especially in elderly patients with signs and symptoms of cardiac instability. Thus, resolution of this severe hyperthyroid state is crucial.

Ideally, amiodarone should be discontinued but cessation of this potent drug is impractical in some patients because it is the only anti-arrhythmic capable of controlling tachyarrhythmias, and even if it can be stopped, AIT can take up to nine months to resolve, its effect may last seven years. When stopping amiodarone, a loss of beta blockage may exacerbate thyrotoxicosis which may not improve and lead to comorbid cardiac conditions and major problems. [2]

Many trials of medical therapy with anti-thyroid drugs, steroids, radioactive iodine, plasmapheresis failed due to the high intrathyroidal iodine content that reduces the efficacy of thionamides which also have many side effects and complications, the low suppressed RAI uptake mainly in type II and the refractoriness of AIT to steroids and plasmapheresis. [13]

Thionamides, usually with a short course of potassium per chlorate are beneficial, and if no response we can add steroids but the effect of medical therapy may take as long as 4 months to be efficient. [7]

Major anti-thyroid drugs are able to temporary control symptoms but all are hepatotoxic especially that treatment requires high doses, steroids can reduce the inflammation but have major side effects, plasmpharesis can provide acute relief of type 2 amiodarone induced thyrotoxicosis but its effect is transient, with higher cost and impossibility of long term use. Radioactive iodine is not feasible in all conditions especially in type 2 because of a low uptake. [3]

Clinical manifestations and morbidity of thyrotoxicosis leading even to thyroid storm, can be the reason for choosing thyroidectomy as the only curative solution. Many series have reported the successful surgical management of amiodarone-induced thyrotoxicosis.[15]

Most studies have shown that total thyroidectomy reduce mortality and morbidity, despite that euthyroidism is difficult to achieve. None of those case series presented here showed a significant complication post thyroidectomy, despite the risk of anesthesia associated with thyrotoxicosis, mainly in patients with heart disease, and the high surgical risk. The anesthetic management should be considered. Patients with mild hyperthyroidism do not require special considerations while those with severe Intraoperative hyperthyroidism (thyrotoxic storm secondary to manipulation of the thyroid gland during thyroidectomy] may require hydration, cooling, and administration of antithyroid drugs, beta-blockers and sodium iodine to prevent any hyperthyroid crisis (storm].[29]. Thus, it is preferable to control hyperthyroid and to restore euthyroidism before thyroidectomy but in most cases, there is a necessity for an urgent treatment before establishing a normal euthyroid state.

When surgery is performed, the postoperative risk of arrhythmias, hemodynamic compromise, and thyroid storm is very minimal.

All patients improved clinically, in addition to amelioration of their cardiac status. Those in whom discontinuing amiodarone is not feasible can be also referred to surgery. [6]

Conclusion

As amiodarone can cause thyroid dysfunction and thyrotoxicosis especially in patients with preexisting heart problems, checking for and treatment of amiodarone induced thyrotoxicosis is important. Total thyroidectomy is an effective method for treatment of amiodarone induced thyrotoxicosis unresponsive to medical therapy, because of severe cardiac morbidity and mortality associated with overt hyperthyroidism.

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