Revista da Sociedade Portuguesa de Anestesiologia
http://revistas.rcaap.pt/anestesiologia
https://dx.doi.org/10.25751/rspa.47039

CASO CLÍNICO / CASE REPORT

Total Thyroidectomy for Refractory Amiodarone-Induced Thyroid Storm in a High-Risk Cardiac Patient: A Perioperative Management Strategy

Tiroidectomia Total por Tempestade Tiroideia Refratária Induzida por Amiodarona em Doente Cardíaco de Alto Risco: Estratégia de Gestão Perioperatória

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Afiliações

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Keywords
Amiodarone; Anesthesia; Thyroid Storm; Thyroidectomy; Thyrotoxicosis.

Palavras-chave
Amiodarona; Anestesia; Tempestade Tiroideia; Tiroidectomia; Tirotoxicose.

ABSTRACT

Thyroid storm is a life-threatening endocrine emergency in which clinical decompensation, rather than hormone concentration alone, determines severity. Amiodarone-induced thyrotoxicosis is particularly challenging because iodine load, prolonged tissue persistence, and mixed pathogenic mechanisms may delay or limit response to conventional therapy. We report the perioperative management of a 49-year-old ASA IV male with ischemic cardiomyopathy, left ventricular ejection fraction of 36%, an implantable cardioverter-defibrillator, and amiodarone-induced thyroid storm complicated by sustained ventricular tachycardia. Severe thyrotoxicosis persisted despite high-dose thionamide therapy, corticosteroids, amiodarone withdrawal, and six sessions of therapeutic plasma exchange. Following multidisciplinary discussion, urgent total thyroidectomy was performed. The anaesthetic strategy focused on attenuating sympathetic response and reducing myocardial oxygen demand, together with strict temperature control, invasive monitoring, preservation of cardiac electrical stability, and careful vasopressor selection. The postoperative course was favourable. This case highlights thyroidectomy as a definitive rescue strategy when refractory thyrotoxicosis perpetuates cardiovascular instability.

RESUMO

A tempestade tiroideia é uma emergência endócrina potencialmente fatal cuja gravidade depende da descompensação clínica, e não apenas da concentração hormonal. A tirotoxicose induzida por amiodarona é desafiante pela carga iodada, persistência tecidular prolongada e mecanismos patogénicos mistos, que podem limitar a resposta à terapêutica convencional. Descrevemos a gestão perioperatória de um homem, 49 anos, ASA IV, com cardiomiopatia isquémica, fração de ejeção ventricular esquerda de 36%, cardioversor-desfibrilhador implantável e tempestade tiroideia induzida por amiodarona complicada por taquicardia ventricular sustentada. A tirotoxicose persistiu apesar de terapêutica com tionamidas, corticoterapia, suspensão da amiodarona e seis sessões de plasmaferese. Após discussão multidisciplinar, realizou-se tiroidectomia total urgente. A estratégia anestésica centrou-se na atenuação da resposta simpática, redução das necessidades miocárdicas de oxigénio, controlo térmico, monitorização invasiva, preservação da estabilidade elétrica cardíaca e seleção criteriosa de vasopressores. A evolução foi favorável, destacando a tiroidectomia como estratégia definitiva quando a tirotoxicose refratária perpetua instabilidade cardiovascular.

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INTRODUCTION

Thyrotoxicosis results from excessive thyroid hormone activity and affects multiple organ systems, particularly cardiovascular and neurological function. Thyroid storm represents its most severe form and remains a clinical diagnosis, as biochemical values alone do not reliably distinguish severe thyrotoxicosis from life-threatening decompensation.[1,2] Contemporary population data continue to show substantial morbidity and mortality in hospitalized patients with thyroid storm.[1-5]

Amiodarone-induced thyrotoxicosis (AIT) is uniquely complex because amiodarone has a high iodine content, extensive tissue distribution, and prolonged biological half-life. AIT may result from iodine-driven hormone synthesis, destructive thyroiditis, or mixed mechanisms, and its clinical presentation may be modulated by the drug's antiadrenergic properties.[3,4] Although elective surgery should generally be postponed until euthyroidism is achieved, urgent thyroidectomy may be required when medical therapy fails or cardiovascular deterioration becomes life-threatening.[3,5]

We describe the anesthetic and perioperative management of refractory AIT requiring total thyroidectomy in a patient with ischemic cardiomyopathy, reduced systolic function, and malignant ventricular arrhythmia.

CASE REPORT

A 49-year-old male (ASA IV) presented after an appropriate discharge of his implantable cardioverter-defibrillator for monomorphic ventricular tachycardia. In the preceding weeks, he had developed anxiety, tremor, heat intolerance, and progressive exertional dyspnea. Laboratory testing revealed suppressed thyroid-stimulating hormone (0.032 µIU/mL), elevated free triiodothyronine (18.1 pmol/L), and elevated free thyroxine (76 pmol/L). Thyroid autoantibodies were negative. Ultrasound demonstrated diffuse thyroid enlargement without nodules.

His cardiac history included previous ST-elevation myocardial infarction, multiple percutaneous coronary interventions, ischemic cardiomyopathy with left ventricular ejection fraction of 36%, septo-apical aneurysm, chronic calcified left ventricular thrombus, and implantable cardioverter-defibrillator implantation after arrhythmic storm three years earlier. He had been receiving chronic amiodarone therapy. Comorbidities included hypertension, obesity (body mass index 35.4 kg/m^2), dyslipidemia, and previous femoropopliteal thrombosis. He was receiving long-term anticoagulation with warfarin.

Amiodarone was discontinued and replaced with sotalol. Thiamazole was escalated to 60 mg/day and prednisolone to 60 mg/day. Perchlorate was considered but not administered. Despite optimized medical treatment, severe biochemical and clinical thyrotoxicosis persisted. Therapeutic plasma exchange was performed over six sessions before surgery, with only a transient biochemical response. The patient fulfilled diagnostic criteria for thyroid storm according to both the Burch-Wartofsky scoring system and the Japan Thyroid Association criteria.[1,2] After multidisciplinary discussion involving endocrinology, cardiology, surgery, anesthesiology, and intensive care, urgent total thyroidectomy was considered indicated. The evolution of thyroid hormone levels and therapeutic interventions is illustrated in **Fig. 1**.

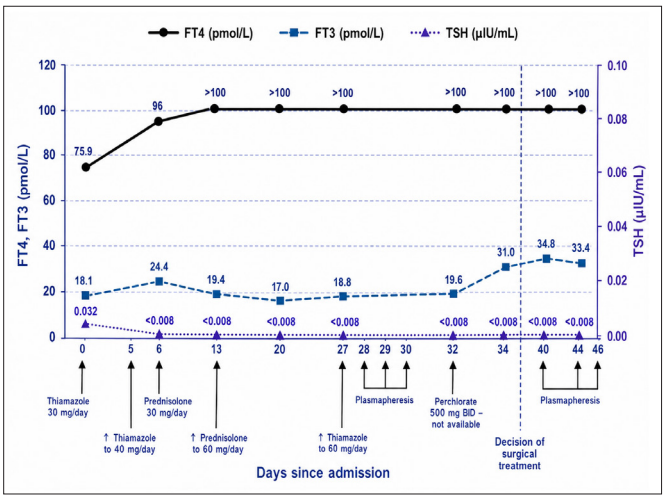

Figure 1. Evolution of thyroid hormone levels and therapeutic interventions during hospitalization.

FT4 and FT3 are shown in picomoles per liter and TSH in micro-international units per milliliter. Arrows indicate treatment escalation and therapeutic plasma exchange sessions; the vertical dashed line marks the decision to proceed with urgent surgical treatment.

Warfarin was transitioned to enoxaparin, which was discontinued 24 hours before surgery. Anxiolytic premedication was administered. In the operating room, a calm environment was maintained, external defibrillator pads were applied, and the implantable cardioverter-defibrillator was deactivated. Invasive arterial pressure and bispectral index monitoring were established before induction. The primary anesthetic objectives were attenuation of sympathetic stimulation, maintenance of hemodynamic stability, prevention of hyperthermia, correction of metabolic abnormalities, and ensure preparedness for malignant dysrhythmias.

Balanced general anesthesia was selected. Lidocaine 1 mg/kg was administered before induction to blunt the sympathetic response associated with airway manipulation. Induction was achieved with fentanyl, etomidate, and rocuronium. Etomidate was chosen for cardiovascular stability in the setting of impaired systolic function. Tracheal intubation was performed smoothly by an experienced anesthesiologist, and continuous temperature monitoring was instituted.

Arterial blood gas analysis revealed mild hypokalemia (3.2 mEq/L), which was corrected. Magnesium sulfate 2 g was administered to reduce catecholamine-mediated arrhythmogenic vulnerability and attenuate sympathetic overactivity. Fluid therapy was guided by pulse pressure variation. After induction, hypotension attributed to anesthetic-induced vasodilation was treated with phenylephrine infusion, titrated to a maximum of 12.5 µg/min. A pure alpha-adrenergic agonist was selected to restore vascular tone while avoiding tachycardia and increased myocardial oxygen demand. Hemodynamic parameters remained stable throughout the

procedure without significant arrhythmias. Intraoperative hemodynamic trends are shown in **Fig. 2**.

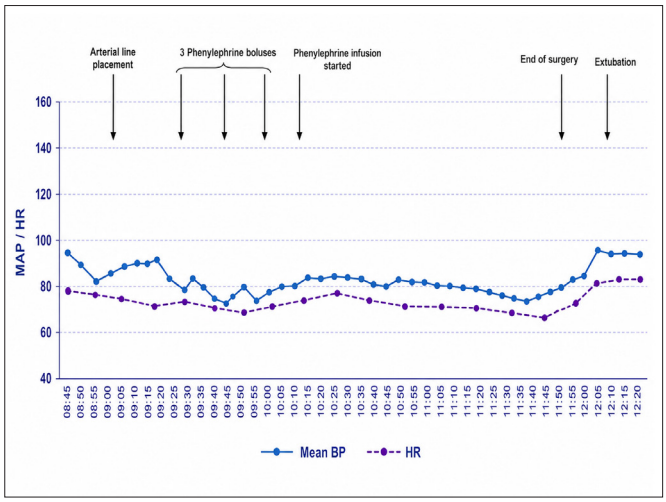

Figure 2. Intraoperative hemodynamic profile during total thyroidectomy.

Mean arterial pressure and heart rate remained within a controlled range after induction and phenylephrine administration, without significant dysrhythmias. MAP, mean arterial pressure; HR, heart rate.

Neuromuscular blockade was reversed with sugammadex, avoiding the dysrhythmic risk associated with neostigmine and atropine. The patient was extubated uneventfully and transferred to the cardiac intensive care unit, where the implantable cardioverter-defibrillator was reactivated.

Thiamazole was discontinued and corticosteroids were tapered. Thyroid hormone levels progressively decreased, with free triiodothyronine normalizing before free thyroxine. The subsequent rise in thyroid-stimulating hormone reflected recovery of hypothalamic-pituitary-thyroid axis activity after removal of the source of thyroid hormone excess. Levothyroxine replacement therapy was initiated according to postoperative endocrine assessment. Calcium levels were monitored closely, with supplementation provided as needed. No clinically significant arrhythmias or hemodynamic instability occurred. The patient was discharged home on postoperative day five with a favorable outcome. Histopathology confirmed findings consistent with amiodarone-associated destructive thyroiditis.

DISCUSSION

This case illustrates a high-risk perioperative scenario in which thyroidectomy was not merely a definitive endocrine intervention, but a cardiovascular rescue strategy. The severity of thyroid storm is determined by the interaction between hormone excess, precipitating stressors, and physiological reserve. This distinction is particularly important in patients with ischemic cardiomyopathy, in whom even a limited additional adrenergic and metabolic burden may exceed compensatory capacity.[1,2]

The cardiovascular effects of thyroid hormone are central to prognosis. Triiodothyronine increases myocardial oxygen consumption, enhances chronotropy and inotropy, reduces systemic vascular resistance, expands blood volume through renin-angiotensin-aldosterone activation, and increases responsiveness to catecholamines.[1,4] In patients with preserved reserve, this may produce a high-output state. In those with ventricular scar, reduced systolic function, or pre-existing arrhythmogenic substrate, the same physiology may precipitate ischemia, heart failure, and malignant dysrhythmias. The implantable cardioverter-defibrillator discharge for sustained ventricular tachycardia in our patient was therefore interpreted as evidence that thyrotoxicosis had destabilized a vulnerable myocardium.

AIT adds further complexity. Type 1 AIT reflects iodine-driven hormone synthesis, typically in abnormal thyroid tissue, whereas type 2 AIT results from destructive thyroiditis with release of preformed hormone; mixed forms are frequent and may be difficult to classify during clinical instability.[3,4] A recent cohort of 55 AIT patients reported thyrotoxic crisis in 11%; all affected patients were male, had higher maximum free thyroxine levels and larger thyroid volume, and urgent thyroidectomy was required in five of six cases.[5] These findings support the clinical relevance of early recognition and decisive multidisciplinary management in patients with AIT and cardiovascular compromise.

Medical treatment of thyroid storm aims to interrupt thyroid hormone synthesis and release, reduce peripheral conversion, control adrenergic manifestations, and provide organ support.[1,2] However, in AIT, particularly in destructive or mixed phenotypes, thionamide response may be incomplete or delayed because hormone excess may reflect release of preformed hormone rather than de novo synthesis.[3] Corticosteroids are therefore particularly relevant because they reduce peripheral conversion of thyroxine to triiodothyronine and target the inflammatory component of destructive thyroiditis.[3] In the present case, persistence of severe clinical and biochemical thyrotoxicosis despite high-dose thionamide therapy, corticosteroids, and withdrawal of amiodarone supported the diagnosis of medically refractory disease.

Therapeutic plasma exchange has a plausible rescue role because most circulating thyroid hormones are protein-bound and can be acutely reduced by plasma removal. The 2024 systematic review of perioperative thyrotoxicosis found that second-line pharmacotherapy and/or plasma exchange may allow surgery when antithyroid drugs are contraindicated or inadequate.[6] A 2025 scoping review including 234 patients reported mean reductions of free thyroxine and free triiodothyronine of approximately 52% and 67% after plasma exchange, but emphasized limited evidence and suggested that repeated sessions should not delay earlier surgical intervention when response is insufficient.[7] In our patient, six sessions produced only transient improvement, reinforcing plasma exchange as a bridge rather than a definitive endpoint.

The decision to proceed with thyroidectomy in uncontrolled thyrotoxicosis must balance operative risk against the ongoing risk of cardiovascular collapse. A systematic review in the British Journal of Anaesthesia found that evidence quantifying perioperative thyroid storm risk is weak and heteroge-

neous, but concluded that preoperative treatment remains warranted because the complication is severe and patients at risk cannot be reliably identified.[8] In refractory AIT with ventricular arrhythmia and limited cardiac reserve, however, achieving euthyroidism before surgery may be unrealistic. Under these circumstances, definitive source control may be safer than prolonged exposure to uncontrolled thyrotoxicosis, particularly as thyroidectomy has been associated with improved control and cardiac recovery in selected AIT patients with severe ventricular dysfunction.[9,10]

The anesthetic strategy was deliberately aligned with pathophysiology and with the principles recommended for endocrine emergencies.[11] Anxiety, laryngoscopy, pain, hyperthermia, hypovolemia, hypoxia, hypercarbia, electrolyte abnormalities, and emergence agitation may all amplify sympathetic activation. Etomidate was selected to preserve cardiovascular stability during induction. Phenylephrine was physiologically appropriate for post-induction vasodilation because it restores vascular tone without beta-adrenergic stimulation, thereby limiting tachycardia and myocardial oxygen demand. Magnesium sulfate was used as an adjunctive antiadrenergic and antiarrhythmic measure; experimental and clinical studies show that magnesium can inhibit norepinephrine release at sympathetic nerve endings through calcium-channel modulation and may attenuate catecholamine-mediated cardiovascular responses.[12,13] Finally, sugammadex avoided the cholinesterase inhibitor-anticholinergic combination that may be undesirable in an arrhythmia-prone patient.

This case contributes to the perioperative literature by showing that successful management of refractory AIT depends on timely recognition of failed bridging therapy, multidisciplinary escalation to definitive surgery, and an anesthetic plan designed to prevent catecholamine-mediated decompensation. The favorable outcome was probably not attributable to any single intervention, but rather to the coherence between endocrine pathophysiology, cardiovascular risk stratification, and perioperative decision-making.

In conclusion, refractory amiodarone-induced thyroid storm in patients with limited cardiac reserve requires early recognition, multidisciplinary escalation, and a perioperative strategy directed at preventing catecholamine-mediated cardiovascular deterioration. In selected high-risk patients, urgent total thyroidectomy should be considered not only as endocrine rescue therapy, but also as a cardiovascular stabilization strategy. Anesthetic management must be individualized, with meticulous attenuation of sympathetic stimulation, invasive hemodynamic monitoring, temperature control, correction of metabolic abnormalities, preparedness for arrhythmias, and judicious vasopressor selection.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO

All authors contributed substantially to the conception of the case report, interpretation of clinical data, drafting and critical revision of the manuscript, approved the final version, and agree to be accountable for all aspects of the work.

Todos os autores contribuíram substancialmente para a conceção do relato de caso, a interpretação dos dados clínicos, a redação e a revisão crítica do manuscrito, aprovaram a versão final e concordam em assumir a responsabilidade por todos os aspetos do trabalho.

Responsabilidades Éticas
Conflitos de Interesse: Os autores declaram a inexistência de conflitos de interesse na realização do presente trabalho.
Fontes de Financiamento: Não existiram fontes externas de financiamento para a realização deste artigo.
Confidencialidade dos Dados: Os autores declaram ter seguido os protocolos da sua instituição acerca da publicação dos dados de doentes.
Consentimento: Consentimento do doente para publicação obtido.
Proveniência e Revisão por Pares: Não comissionado; revisão externa por pares.

Ethical Disclosures
Conflicts of Interest: The authors have no conflicts of interest to declare.
Financing Support: This work has not received any contribution, grant or scholarship.
Confidentiality of Data: The authors declare that they have followed the protocols of their work center on the publication of patient data.
Patient Consent: Consent for publication was obtained.
Provenance and Peer Review: Not commissioned; externally peer-reviewed.

Submissão: 7 de maio, 2026 | Received: 7th of May, 2026
Aceitação: 15 de junho, 2026 | Accepted: 15th of June, 2026
Publicado: 8 de julho, 2026 | Published: 8th of July, 2026

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