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Cardiothyrotoxicosis: A Life-Threatening Complication of Hyperthyroidism Leading to Cardiac Arrest

Oussama Jaddi[1], Sana Rafi[2], Sara Ijdda[3], Ghizlane El Mghari[4], Nawal EL Ansari[5]

[1,2,3,4,5]Department Department of Endocrinology, Diabetology, Metabolic Diseases and Nutrition Cadi Ayyad University, Mohammed VI University Hospital, Marrakesh, Morocco.

ABSTRACT

Cardiothyrotoxicosis represents a life-threatening complication of hyperthyroidism, characterized by severe cardiac rhythm disturbances and hemodynamic instability. This condition can precipitate potentially fatal arrhythmias leading to cardiac arrest, especially in patients with underlying cardiac disease or those experiencing a thyroid storm. We present three clinical cases of cardiothyrotoxicosis culminating in cardiac arrest, illustrating the diverse clinical presentations and the complex pathophysiological mechanisms involved. The discussion reviews the impact of excess thyroid hormones on cardiac function, the associated risks of arrhythmias and heart failure, and the importance of rapid diagnosis and multidisciplinary management. Early recognition and aggressive treatment of thyrotoxicosis and its cardiovascular complications are crucial to reducing morbidity and mortality. Increased clinician awareness and vigilant monitoring of at-risk patients are essential for optimizing outcomes.

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KEYWORDS:

Cardiothyrotoxicosis; Hyperthyroidism; Cardiac arrest; Arrhythmia; Thyroid storm; Heart failure; Antithyroid drugs; Endocrine emergencies.

INTRODUCTION

Cardiothyrotoxicosis is a severe complication of hyperthyroidism, marked by cardiac rhythm disturbances and hemodynamic instability [2,3]. It often manifests as potentially fatal arrhythmias that may culminate in cardiac arrest [1,3,5,7]. Thyrotoxicosis, denoting an excess of thyroid hormones, is a major risk factor for these cardiovascular events [2,4].\

Corresponding Author: Oussama Jaddi

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We report three clinical cases of cardiothyrotoxicosis that resulted in cardiac arrest, highlighting the underlying pathophysiological mechanisms and clinical management implications.

CASE REPORT

Case 1:

A 66-year-old female patient was admitted for the management of cardiothyrotoxicosis complicated by atrial fibrillation (AF). Her laboratory workup showed suppressed TSH, free T4 at 29.60 pmol/L, and free T3 at 6.23 pmol/L. Initial treatment included carbimazole 10 mg, bisoprolol 10 mg/day, rivaroxaban 20 mg/day, and furosemide 40 mg (¼ tablet/day). She underwent total thyroidectomy due to a toxic multinodular goiter (TMNG). However, during the night following surgery, she developed tachypnea (respiratory rate 38) and tachycardia

(180 bpm). The next morning, she experienced severe dyspnea with profuse sweating, culminating in a cardiorespiratory arrest.

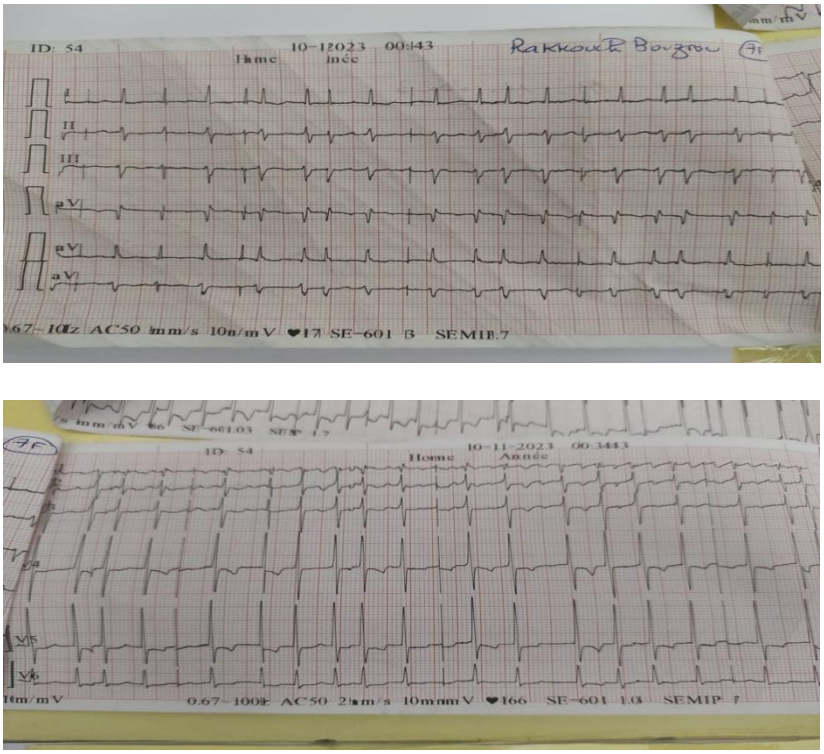

Figure 1: ECG showing atrial fibrillation

Case 2:

A 55-year-old man was admitted for dyspnea. Electrocardiogram (ECG) revealed atrial fibrillation. Thyroid panel showed a TSH level below 0.01 and a free T4 level of 28.49 pmol/L. On clinical examination, he had tachycardia at 122 bpm, a WHO grade 1 goiter, and signs of heart failure, along with significant markers of hepatic cytolysis. The patient was started on hydrocortisone and propranolol. Unfortunately, after 8 hours of treatment, he suffered a cardiorespiratory arrest.

Case 3:

A 26-year-old woman was admitted in shock for cardiothyrotoxicosis. ECG showed supraventricular tachycardia. Her condition rapidly deteriorated, resulting in cardiac arrest, which was successfully resuscitated. Tests revealed suppressed TSH and markedly elevated free T4. She was treated with norepinephrine and hydrocortisone.

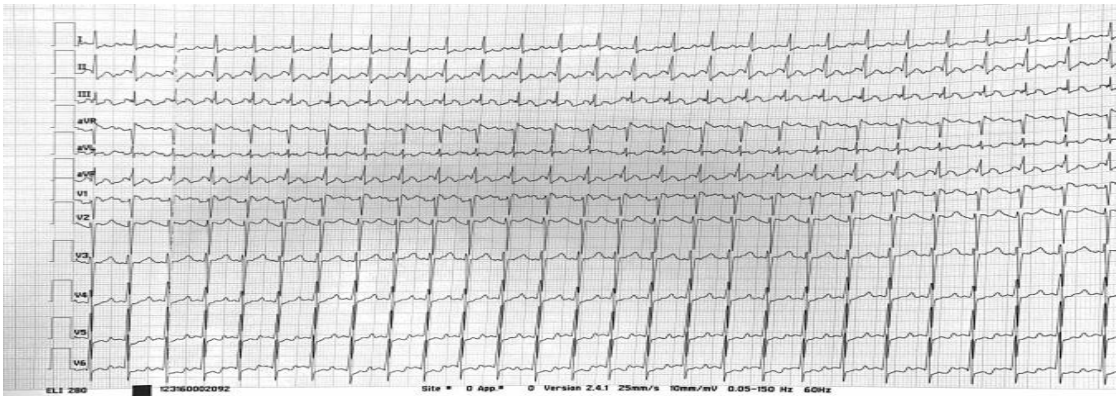

Figure 2: supraventricular tachycardia on ECG

DISCUSSION

Thyroid hormones, mainly thyroxine (T4) and triiodothyronine (T3), play a crucial role in regulating tissue metabolism, including cardiac function [2]. T3 is biologically more potent than T4 and affects gene expression in the heart, influencing contraction and rhythm through both genomic and non-genomic mechanisms [2,4]. Hyperthyroidism has major cardiovascular repercussions. It increases cardiac output, sometimes up to 300% above normal, enhances sensitivity to catecholamines, and activates the renin-angiotensin-aldosterone system, leading to circulatory hyperdynamics [2,4]. Hyperthyroidism is also a risk factor for coronary artery disease, increasing mortality and the incidence of cardiovascular events [2,4,5]. It can induce both preserved and reduced ejection fraction heart failure, most often reversible with antithyroid therapy [2,5,7]. Rhythm disturbances are frequent, especially sinus tachycardia and atrial fibrillation, promoted by increased β-adrenergic receptor activity and sympathetic tone [2,5,7]. The perioperative risk is also amplified in uncontrolled hyperthyroid patients, making preoperative stabilization essential to limit cardiovascular complications [2,5].

A retrospective analysis of 6,380 hospitalizations for thyroid storm showed that 3,895 patients had cardiovascular events, including 3,770 cases of arrhythmias, 555 cases of acute heart failure, and 150 ischemic events. Hospital mortality was significantly higher in patients with cardiovascular complications (3.5% vs. 0.2%), with a longer median hospital stay [2,4].

Clinical management is based first on a careful assessment of history and clinical examination, looking for signs of thyrotoxicosis such as palpitations, weight loss, sweating, or tremors, as well as signs of heart failure like dyspnea, edema, or tachycardia [2,3,4]. ECG allows the detection of rhythm disturbances, and laboratory tests include TSH, free T4, T3, electrolytes, and liver enzymes [2,4]. Immediate stabilization requires continuous cardiac monitoring for early detection of arrhythmias, oxygen therapy as needed for hypoxia, and intravenous access for rapid administration of treatments [2,3,4,5].

Medical treatment combines antithyroid drugs such as carbimazole or methimazole to suppress hormone production, beta-blockers like propranolol to control heart rate and adrenergic symptoms, and corticosteroids (hydrocortisone) to reduce inflammation and metabolic stress, especially in severe thyroid storm [2,3,4,5]. Sedation may be necessary in cases of significant agitation [2,3]. Management of rhythm disorders is based on cardioversion in case of instability and correction of electrolyte imbalances that may aggravate arrhythmias [2,3,5,6].

In case of large goiter, Graves' disease, or refractory thyrotoxicosis, thyroidectomy may be indicated, especially in the presence of cardiovascular complications [2,4,5]. Specialized endocrine follow-up is essential to adjust antithyroid therapy, prevent recurrences, and educate the patient about recognizing symptoms of thyrotoxicosis [2,4,5]. Finally, prevention of recurrences involves discussing long-term options such as radioactive iodine ablation or surgery in case of repeated episodes [2,4,5].

CONCLUSION

Cardiothyrotoxicosis is a formidable complication of hyperthyroidism with a high risk of fatal outcomes such as cardiac arrest. The presented cases illustrate the diverse manifestations and complex mechanisms leading to severe cardiovascular events. Improved understanding and vigilant monitoring of at-risk patients are crucial for prevention. Effective management of thyrotoxicosis, including treatment of arrhythmias and hormonal monitoring, is essential to reduce morbidity and mortality. Clinician awareness is key to optimizing care for patients with hyperthyroidism.

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