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# CLINICAL CASE

# FATAL CARDIOTHYREOSIS REVEALED THROUGH A STATUS EPILEPTICUS AND RESULTING IN A THERAPEUTIC IMPASSE A CASE REPORT

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## ABSTRACT

Cardiothyreosis is a rare and serious complication of thyroid disease. We report a case that is unique in its inaugural clinical picture (status epilepticus) due to late diagnosis and iatrogenic drug which precipitated the patient in an array of congestive heart failure where therapeutic management was limited by insufficient technical facilities leading to death.

KEY WORDS: Cardiothyreosis, Thyroid disease, Status epilepticus.

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## INTRODUCTION

Cardiothyreosis is a rare complication of thyroid disease and usually occurs during the natural course of Graves' disease. Severe forms constitute of cardiac and neurological manifestations. We report a case of acute thyrotoxic crisis revealed unusually by a status epilepticus which delayed diagnosis, induced worsening of iatrogenic disease and leading to a major dysfunction of the heart pump as part of a fatal therapeutic impasse.

#### CASES REPORT

A student of Moroccan nationality, aged 28 and with unknown medical history was brought to the emergencies by unsafe means of transportation (civil protection) for a status epilepticus which occurred in a bus, with no notion of trauma, according to witnesses, and evolved for more than thirty minutes. The examination at admission showed a patient with generalized tonic-clonic seizures with a blood pressure at 142/71 mmHg, a heart rate of 135 bpm, a pulsed O2 saturation of 92% in ambient air and capillary blood glucose of 1.45 G/L. The temperature was 38.8 °C

and the weight was 82 Kg. The patient management led to a general anesthesia to stabilize the patient. The etiological assessment included a normal brain scan and lumbar puncture. Serum sodium (141 mEq/L), serum calcium (2.1 mmol/L) and renal function were normal. The patient was admitted to intensive care and put under basal diet with hourly glycemic control. The patient received sedation with midazolam (0.1 mg/kg/h) with a Ramsey score 6 in 30 minutes. Sodium valproate was given at a dose of 500 mg/6h intravenously. Analgesia was provided by fentanyl 50 mcg/h with electric syringe pump. The patient was ventilated in controlled ventilation mode with target partial arterial pressure of CO2 fixed at 38 mmHg. Hyperthermia was treated with paracetamol IV and the use of physical cooling means. Prophylactic treatment of thromboembolic disease with enoxaparin 40 mg/d subcutaneously was introduced as well as gastric antiulcer protection with omeprazole 40 mg/d IV. The evolution was marked by the increase in heart rate (sinus tachycardia at 155 bpm), insensitive to filling and outside any fluid and electrolyte disorder. It was treated with amiodarone (450 mg loading dose and 800 mg over 24 hours' maintenance; weight 80 kg). On the second day, the hemodynamic state was complicated by refractory hypotension associated with global hypokinesia on echocardiography. The performed cTnI returned to 8.1 ng/ml. The patient received dobutamine at 10 mcg/kg/min, and a loading dose of aspirin at 300 mg then 160 mg/day and enoxaparin at 70 mg/12h. The records of the patient's history with the family, afterwards, revealed the existence of a recently discovered untreated thyroid dysfunction. No history of drug use, injection, or trauma was found. The clinical condition of the previous days was described as normal by the family. Cervical examination with ultrasound and hormonal assays confirm the existence of a thyroid nodule lump producing a biological hyperthyroidism with TSH of 0.02 µUI/L, a triiodothyronine and a thyrotonine respectively of 8.45 nmol/L and 185 nmol/L. The clinical showed a complicated picture thyrotoxicosis cardiothyreosis by administration of amiodarone. Amiodarone was stopped and the patient underwent specific corticosteroids treatment: based on Hydrocortisone at 300 mg/day and Carbimazole at 40 mg/day. The Esmolol was not administered. The basal diet was increased and an emergency surgical resection was considered, but the patient died in an array of congestive heart failure.

## DISCUSSION

Cardiothyreosis is a rare complication of thyroid disease. Cardiothyreosis represents 1% of patients hospitalized for hyperthyroidism [1]. In Morocco, in endemic areas in the southern half of the country, the estimated rate is 16.66% [2]. The thyroid storm occurs most often in the context of Graves' disease, a toxic thyroid nodule or thyroiditis. The contributing factor in the Moroccan socio-medical context is the absence of prior diagnosis and treatment of thyroid disease [2]. The triggers are unknown and may be related to a high level or rapid and sudden increase in levels of circulating thyroid hormones; hyperactivity of the sympathetic nervous system and the amplification of the cellular response to hormones, that result, can contribute to Cardiothyreosis [3]. We did not find any trigger in our patient.

The clinical presentation of severe forms refers to the consequences of heart failure. In our case, heart failure was marked by a hyperkinetic state followed by a cardiac ischemia leading to congestive heart failure refractory to medical treatment. Neurological manifestations are rare. They were reported as convulsions or encephalopathy particularly in children [4]. These manifestations were due to late diagnosis, initially oriented towards traumatic brain injury and infectious brain disease in immunocompetent. In the absence of clinical specificity to give the positive diagnosis of acute cardiothyreosis, we used the score of Burch-Wartofsky [5] who found a score of 95 points (> to 45/140). The use of hormone assays allows giving only the diagnosis of biological hyperthyroidism and performing treatment follow-up.

In the specific treatment, antithyroid drugs are used, but their effect is delayed (4 to 6 weeks) and they must be administered orally, incompatible in a state of shock. Radioactive iodine and lithium (900-1200 mg/day), usually used for perioperative preparation for surgery, are compatible with an urgent situation such as the case we are reporting.

Symptomatic treatment uses beta-blockers in reducing tachycardia without altering the positive inotropic thyroid hormones that can worsen potential heart failure. Given this situation, the circulatory assistance is considered as the last resort. In our case, we neither prepared the above mentioned medical treatment nor plasmapheresis nor circulatory substitution. The use of beta-blockers was not maintained given the dysfunction of the heart pump.

Non-specific resuscitation should emphasize adaptation of water needs to compensate for insensible losses and balance the metabolic status especially blood glucose and serum potassium. Renal function should be monitored as well as renal protection measures carried out in particular in view of the risk of rhabdomyolysis.

# CONCLUSION

Knowledge and the means of managing hyperthyroidism currently allow the reduction of the incidence of perioperative cardiothyreosis. When thyroid disease is mistreated or misunderstood, it leads to the occurrence of potentially fatal severe forms requiring rapid response and appropriate intensive care environment.

#### **AUTHORS' CONTRIBUTIONS**

The participation of each author corresponds to the criteria of authorship and contributorship emphasized in the <u>Recommendations for the Conduct, Reporting, Editing,</u> <u>and Publication of Scholarly work in Medical Journals</u> of the <u>International Committee of Medical Journal Editors</u>. Indeed, all the authors have actively participated in the redaction, the revision of the manuscript and provided approval for this final revised version.

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Declared none.

#### PATIENT CONSENT

Written informed consent was obtained from the patient for publication of this case report.

#### **COMPETING INTERESTS**

The authors declare no competing interests.

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