

Antoine Kimmoun
 Gaittha Munagamage
 Nicolas Dessalles
 Alain Gerard
 François Feillet
 Bruno Levy

Unexpected awakening from comatose thyroid storm after a single intravenous injection of L-carnitine

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Antoine Kimmoun and Gaittha Munagamage participated equally in this work.

Dear Editor,
 We present the first report of an 80-year-old man admitted to the intensive care unit (ICU) for severe hyperthyroidism with coma, awakened by L-carnitine. For 1 month prior to admission, he was treated with amiodarone (200 mg/day). His neurological state worsened with a Glasgow coma score (GCS) of 8 and he was transferred to the ICU. At admission, clinical examination showed dyspnea, atrial flutter at 150/min, and hyperthermia at 39°C. There was no biological inflammatory syndrome or other metabolic abnormalities. All neurological explorations were normal. A thyrotoxicosis was confirmed: thyroid stimulating hormone (TSH), 0.02 mUI/L (controls 0.17–4); free thyroxine (FT4), 74 pmol/mL (controls 10–21); free triiodothyronine (FT3), 41 pmol/mL (controls 2.5–5.8). TSH was normal prior to amiodarone treatment. Thyroid echography was normal and thyroid scintigraphy showed iodine overload. The diagnosis was type II

amiodarone-induced hyperthyroidism (AIT) in apathetic form with coma and cardiothyreosis.

Amiodarone was stopped and atrial flutter was reduced under propranolol. Hyperthyroidism was treated with propylthiouracil (PTU) (200 mg × 3/day) and Solu-Medrol (methylprednisolone sodium succinate) 1 mg/kg/day. Twelve days after admission, the GCS remained at 8. A rescue therapy by L-carnitine (i.v. 75 mg/kg/day) was attempted. The patient fully recovered 24 h after L-carnitine administration and he was discharged 2 months later from the hospital. He was asymptomatic, but TSH still remained very low with elevated levels of FT3 and FT4 (Fig. 1). He was euthyroid only 3 months later.

Thyroid storm is an emergency situation with a 20–50% mortality rate. Apathetic thyrotoxicosis (AT) is a rare form of thyroid storm. This clinical presentation was first described in 1931 and is characterized by prostration, encephalomyopathy and coma [1]. AT is most often found in the elderly, but has been reported at all ages.

The presentation of type II AIT was typical in our patient. Two mechanisms of AIT are described: iodine-induced hyperthyroidism and destructive thyroiditis due to amiodarone itself [2]. Type II AIT is the most frequent form and can develop early after onset of treatment or several months after drug withdrawal because of the long half-life of amiodarone. In our patient, 2 weeks after usual treatments, there was no neurological recovery. One team has reported the usefulness of L-carnitine as an alternative therapy to classic hyperthyroidism treatment in a 24-year-old patient with a history of successive thyroid storm [3]. L-Carnitine is a quaternary amine, which primarily allows the entrance of fatty acids into the mitochondria. L-Carnitine is also known to inhibit T3 nuclear uptake; consequently, it is an antagonist of the effect of thyroid hormones, which can be used in hyperthyroidism [4]. This effect has also been proven in a randomized, double-blind clinical trial [5].

This is the first report of the efficacy of L-carnitine in the comatose

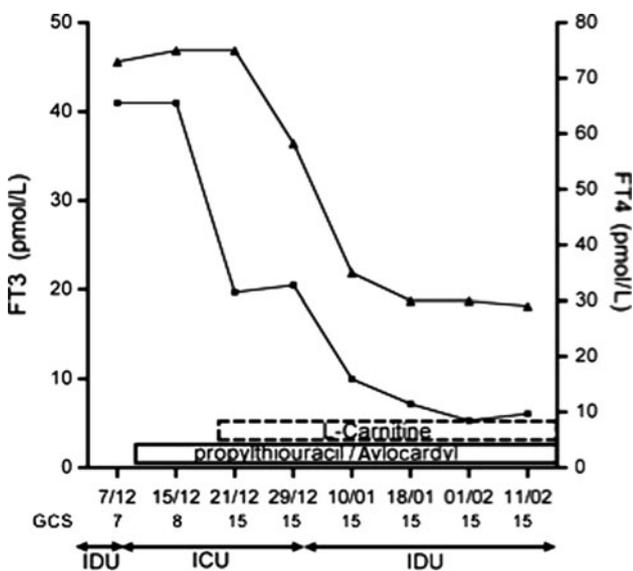


Fig. 1 Clinical and biological course. GCS Glasgow coma score, IDU infectious disease unit, ICU intensive care unit

form of AT with rapid recovery. As this molecule has no secondary effects, it may prove useful in thyroid storm.

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A. Kimmoun (✉) · G. Munagamage · N. Dessalles · A. Gerard · B. Levy
Service de Réanimation Médicale,
Institut du Coeur et des Vaisseaux,
Hôpitaux de Brabois, CHU de Nancy,
Rue du Morvan, 54511
Vandoeuvre-les-Nancy, France
e-mail: a.kimmoun@chu-nancy.fr
Tel.: +33-3-83154084
Fax: +33-3-83154220

F. Feillet
Centre de référence des maladies
héréditaires du métabolisme,
INSERM U 724, Hôpitaux de Brabois,
CHU de Nancy, Rue du Morvan, 54511
Vandoeuvre-les-Nancy, France