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

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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 propanolol. 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

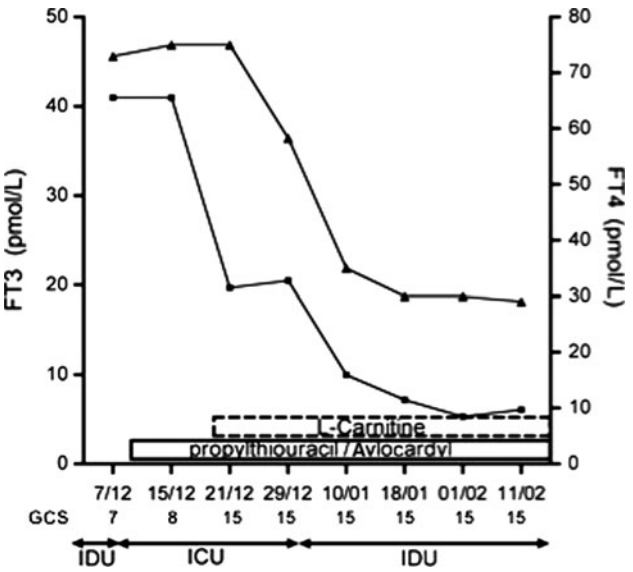

Fig. 1 Clinical and biological course. *GCS* Glasgow coma score, *IDU* infectious disease unit, *ICU* intensive care unit

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

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