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CE - LETTER TO THE EDITOR

Severe thyrotoxicosis: a rare cause of acute delirium

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Dear Sirs,

A 56-year old woman presented to the emergency department (ED) with acute delirium ongoing since the previous day. Her family had initially contacted the general practitioner on call the night before. He found her agitated, confused and delirious. Being unfamiliar with her medical history, he diagnosed an acute psychosis, and admitted her to the nearby psychiatric clinic for further treatment. Due to the patients considerable aggression, it was impossible to implement any kind of diagnostic procedures there, which led to the referral to our clinic. On examination in the ED, the patient showed severe hyperactive delirium, exophthalmus, wasting (BMI 17 kg/m^2), and a diffuse goitre. The vital signs included: a body temperature 38.2°C, blood pressure 120/70 mmHg, heart rate 124 beats/min. As her family doctor later reported, she was diagnosed with Graves' disease 30 years prior, and a subtotal thyroidectomy had been performed. Even after extensive search, no medical report could be found on the exact type of surgical treatment. Shortly after surgical treatment, thyreostatic therapy for recurrence of the hyperthyroidism had to be re-implemented. Due to "intolerance of the medication," our patient subsequently stopped thyreostatic treatment, and refused any therapy, except homeopathic tablets to control thyroid function. The laboratory tests on admission showed a severe hyperthyroidism (TSH <0.05 mIU/l, free T4 60 pmol/l) and a mild leucocytosis of 12,200/ul, while the C-reactive protein was not elevated. With the diagnosis of acute delirium associated with thyrotoxicosis, the patient was admitted to the ICU. In order to start thyreostatic therapy, high dose parenteral sedation for the first 24 h was necessary. Her therapy then consisted of 45 mg thiamazol per day, 20 mg propranolol administered every 8 h and 200 mg hydrocortison per day. Four days after admission, she was transferred to the medical ward, her thyroid function had improved considerably with a free T4 of 18.4 pmol/l. She was less confused, able to follow instructions and participate in conversations. The further clinical course was favourable with an uncomplicated total thyroidectomy performed after 3 months of medically well-controlled thyroid function. Radioiodine as a treatment option was considered, but, because of the endocrine orbitopathy, although inactive, and the patient preference, was abandoned in the end.

Our patient fulfilled the criteria for the diagnosis of a "thyroid storm" introduced by Burch and Wartofsky [1]. The most common underlying cause of this rare condition is, as in our case, Graves' disease, and mortality rates range from 20 to 30% [2]. A challenge lies in early diagnosis and appropriate treatment of this medical emergency in order to prevent catastrophic outcomes up to multiple-organ failure. Most cases of thyroid storm occur following a precipitating event or intercurrent illness such as thyroid or non-thyroidal surgery, trauma, infection, or an acute iodine load [3]. Patients suffering from severe thyrotoxicosis frequently present with psychiatric symptoms ranging from depression to acute psychosis [4]. In contrast to the broad range of literature on psychosis in association with hypothyroidism,

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Table 1 Common causes of delirium and confusional states Adopted by 2010 uptodate®, online version 18.1

Drugs and toxins
Prescription drugs: i.e. Opioids, sedatives–hypnotics, antipsychotics etc.
Drugs of abuse: i.e. ethanol, heroin, hallucinogens
Withdrawal states: i.e. ethanol, benzodiazepines
Medication side effects: i.e. hyperammonemia from valproic acid, serotonin syndrome
Infections
Sepsis
Systemic infections; fever related delirium
Metabolic derangements
Electrolyte disturbance: elevated or decreased (sodium, calcium, magnesium)
Endocrine disturbance: depressed or increased (thyroid, parathyroid, adrenal, pituitary)
Hyperglycemia and hypoglycemia
Hyperosmolar and hypoosmolar states
Hypoxemia and hypercarbia
Inborn errors of metabolism: i.e. Wilson's disease etc.
Nutritional: i.e. Wernicke's encephalopathy, vitamin B12 deficiency
Brain disorders
CNS infections: encephalitis, meningitis, brain or epidural abscess
Epileptic seizures, non-convulsive status epilepticus
Head injury
Hypertensive encephalopathy
Psychiatric disorders
Systemic organ failure
Cardiac, renal or liver failure; acute or chronic
Hematologic disorders
Physical disorders
Hypo-, Hyperthermia
Burns, Trauma with SIRS

such as the well-known "myxoedema madness", little can be found on delirium associated with hyperthyroidism. Psychological symptoms presented by patients with thyrotoxic crisis include depressive, manic, paranoid and schizoid features. In an emergency medicine setting patients admitted to the hospital with acute delirium are common [5]. When handling such a case it is essential to rule out underlying somatic causes before assuming an intrinsic psychiatric disorder (Table 1). Besides a profound general and neurological examination, effective diagnostic measures include laboratory testing to screen for fluid or electrolyte disturbances, infections or sepsis, the withdrawal from alcohol or drugs, and the search for metabolic disorders such as hypoglycemia, hypercalcemia, uremia, liver failure and, last but not least, thyrotoxicosis. Further diagnostic procedures such as lumbar puncture, EEG and neuroimaging may be necessary if no cause is apparent, or if the delirium does not improve despite seemingly appropriate treatment.

Our patient was sent to a psychiatric clinic before underlying somatic conditions were ruled out because the physician involved was unfamiliar with the clinical picture of "thyroid storm" and routine laboratory tests did not include thyroid function. Concerning the clinical picture, we consider the clinical criteria offered by Burch and Wartofsky to be helpful for emergency physicians. Yet, with regard to the realities of working in an ED setting, we first and foremost propose to routinely check thyroid function in patients with acute delirium in order to prevent missing this potentially fatal but treatable condition. After establishing the diagnosis of severe thyrotoxicosis, the patient should undergo immediate emergency therapy with vigourous intravenous fluid replacement, and treatment of hyperthermia as well as all supportive measures. Additionally, a multidrug approach with several targets is vitally important; thyreostatic therapy to stop the synthesis of new hormone within the thyroid gland, corticosteroids for preventing conversion of T_4–T_3 and Beta-adrenergic blockade for controlling adrenergic symptoms [3]. Thus, early recognition and prompt implementation of all the treatment modalities is essential for successful management of life-threatening thyrotoxicosis.

Conflict of interest None.

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