

Sequential Thyroid Storm and Myxedema Coma: A Unique Case Report

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Abstract

Background and objective: Both thyroid storm and myxedema coma are rare life-threatening events and carry a high mortality rate. Our objective is to document a unique case of sequential thyroid storm and myxedema coma.

Results: We report a very unusual case of a 46-year-old female patient who presented initially in 2002 with thyroid storm caused by a previously undiagnosed Graves' disease. After two doses of radioactive iodine treatment, she developed hypothyroidism for which she was placed on l-thyroxine replacement therapy. She was lost to follow-up and, indeed, she failed to take her l-thyroxine medication and in the fall of 2010, she presented to our Emergency Department with myxedema coma, which was treated successfully.

Conclusion: A careful and life-long follow-up is essential in patients who have received thyroid radioactive iodine ablation or total thyroidectomy to ensure that their thyroidal status remains stable. This constitutes the first step in the prevention of myxedema coma, a diagnosis which still carries an appreciable mortality rate.

Keywords: Hypothyroidism; Graves' disease; Myxedema coma; Thyroid storm; Levothyroxine

Introduction

Unrecognized thyrotoxicosis can be complicated by thyroid storm which is a serious medical condition associated with a mortality rate of 20-30% [1,2], although the most recent data suggest it is around 10% [3,4]. Mortality rate can be high also in untreated hypothyroidism which progresses to myxedema coma that carries a mortality rate of 20-25% [5], despite intensive supportive care delivered to patients with such complication.

We report a case of a middle-aged female patient who presented initially with thyroid storm. Eight years later, she presented with myxedema coma as a result of non-compliance to l-thyroxine replacement treatment for post-radioactive iodine hypothyroidism.

Case Report

A 46-year-old black female patient presented to the emergency department with thyroid storm in 2002. Her initial presentation to the emergency department was in the form of psychosis and agitation, which had been noted by family members for the last 5 days prior to admission. The family members were also concerned that the patient had gradually lost some 20 lbs during the previous two years. Her vitals showed a sinus tachycardia with a heart rate of 170 beats per minute and a blood pressure of 186/99 mm Hg. Clinical evaluation confirmed that she was agitated and psychotic. Her deep tendon reflexes were bilaterally brisk. Her thyroid gland was firm and diffusely enlarged to three times normal size and she demonstrated signs of thyroid-associated ophthalmopathy, including bilateral exophthalmos and lid lag. There was no hyperpyrexia. These physical signs added to 45 points on the Burch and Wartofsky score for the clinical likelihood of having a thyroid storm [1]. There was no family history of thyroid disease.

A diagnosis of thyroid storm was made and the patient was managed in the intensive care unit with propylthiouracil (PTU) at 200 mg every four hours, Lugol's solution, propranolol and hydrocortisone starting at 100 mg intravenously three times a day tapered to prednisone orally. No precipitating factors for the thyroid storm were identified and the diagnosis of an underlying new onset Graves' disease was made based on the findings of a diffuse goiter and overt signs of thyroid-associated ophthalmopathy. Serum TSH was <0.007 mU/l (normal 0.5-4.0) and

free T4 >100 pmol/l (normal 10-22). Anti-TSH receptor antibodies came back showing a positive result of 26 units/L (reference: <10) while the anti-TPO antibody was more than 1000 IU/mL (reference: 0-35).

The patient improved clinically within days, which were reflected biochemically by a marked decrease in her serum free T4 level to 35.21 pmol/l. She was discharged on PTU and propranolol, following which she was given 16 mCi of radioactive iodine ¹³¹I treatment. She became euthyroid but one year later, she developed a recurrence of Graves' hyperthyroidism. This was confirmed by an elevated serum free T4 of 22.75 pmol/l, a TSH of <0.01 mU/L and an increased 24-hour ¹³¹I uptake of 43% with diffuse distribution of the tracer. Thus, a second dose of 20 mCi of ¹³¹I was administered. The patient developed hypothyroidism and was placed on l-thyroxine replacement therapy, requiring daily doses of 150-200 mcg to maintain a normal serum TSH over the ensuing three years.

She was lost to follow up in 2007. Her next encounter with the medical service was in 2010 where she presented to the emergency department with decreased level of consciousness, a core body temperature of 34.4°C, a blood pressure of 120/67 mm Hg and a heart rate of 38 beats per minute. In addition, she demonstrated a generalized non-pitting edema including periorbital edema, dry skin, coarse hair and a delayed relaxation phase of the Achilles tendon. Her family members reported that the patient had menorrhagia, fatigue, pain in the limbs for couple of weeks. Her Glasgow Coma Score was 12. We also employed the recently designed diagnostic scoring system for myxedema coma [6], and our patient scored 80 (a score of >60 is considered to be highly suggestive/diagnostic of myxedema coma).

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Serum TSH was elevated at 58.85 mU/l (normal: 0.5-4.0) and her free T4 was <1.9 pmol/l (normal: 10-22). Other blood tests showed a normal Na of 138 mEq/L, glucose of 104.50 mg/dL, creatinine of 2.16 mg/dL but an elevated creatinine kinase of 1638 u/l (normal: 24-195 u/l). A random serum cortisol level was performed which showed a concentration of 904 nmol/l (a random level of >800 nmol/l in major stress being regarded as indicative of adequate adrenal reserve). Echocardiography examination revealed a 7 mm circumferential pericardial effusion with diffuse hypokinesia.

The diagnosis of myxedema coma was made and the patient was managed in the intensive care unit for hemodynamic and respiratory support. She was ventilated and initiated on passive re-warming and a vasopressor because of a drop in blood pressure. Further management included an intravenous dose of levothyroxine of 100 mcg followed by doses that ranged between 75-100 mcg i.v. daily as the oral intake was poor at that time. No precipitating factors for the myxedema coma were identified except for the fact that she had not taken any l-thyroxine replacement for the last 2 years according to family members. Therefore, the only explanation for her presentation was severe hypothyroidism complicated by possible cold exposure (as it was the late fall in Canada) which tipped the patient into myxedema coma. The patient improved dramatically on the third day (was alert and oriented, extubated uneventfully and weaned completely off pressure support). Her biochemical profile on the same day showed a TSH of 40 mU/l and a free T4 of 4.7 pmol/l. She was discharged on l-thyroxine and she became compliant to both her therapy and follow-up; she has remained euthyroid while on l-thyroxine 175 mcg daily.

Discussion

The spectrum of thyroid dysfunction encompasses various grades of hypothyroidism and hyperthyroidism, from subclinical to mild and overt, while thyroid storm and myxedema coma represent the very two extremes. Both of them are rare but life-threatening events and carry a high mortality rate [7,8] even with best possible treatment. Early diagnosis is of critical importance but it may not be easy to recognise these two extremes of thyroid dysfunction, especially in patients with no previous history of thyroid disorders and are seen in a comatose state. In 1993, Burch and Wartofsky developed a novel scoring system to standardize the diagnosis of thyroid storm [1], and a recent comprehensive review detailed an approach aimed at improving its morbidity and mortality [4]. Wartofsky and his colleagues have very recently come up with a diagnostic scoring system for myxedema coma [6] and future studies employing this score will be needed to confirm the clinical usefulness of this approach.

To our knowledge, there has been no previous report of a patient experiencing sequential thyroid storm and myxedema coma. These two events were separated by eight years in our patient and she was successfully treated on both occasions. The initial presentation of thyroid storm was quite classical while the myxedema coma occurred primarily because of non-compliance to the prescribed l-thyroxine replacement treatment in the setting of post-radioactive iodine hypothyroidism. Interestingly, Dutta et al. recently reported from their

series of 23 consecutive patients with myxedema coma that l-thyroxine treatment defaulters had more severe manifestations compared with *de novo* subjects [9]. Moreover, these authors identified various predictors of mortality including hypotension, bradycardia at presentation, need for mechanical intervention hypothermia unresponsive to treatment, sepsis, intake of sedative drugs, lower Glasgow Coma Score (GCS), high APACHE II score and high Sequential Organ Failure Assessment (SOFA) score. The latter SOFA score had the best predicted value [9].

The extreme thyroïdal dysfunctional states observed in our patient during a time-frame of eight years serve to underscore the importance of life-long follow-up of patients with thyroid disease, especially those on active treatment. Moreover, all patients receiving l-thyroxine replacement treatment should be counseled on the importance of compliance to their prescribed medications.

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