

Case Report

Myxedematous Coma: A Case Report

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Abstract

Introduction: Thyroid dysfunctions are highly prevalent diseases. Despite of its high prevalence the presentation is unusual nowadays because of oral hormone therapy widely used in insufficiency of the gland however hyperfunction states are common also. About 12% of the US population will develop some alteration of thyroid function in their lifetime. Among these diseases we highlight the presence of myxedema coma in a female patient.

Case presentation: A 58-year-old female patient with symptoms of drowsiness and loss of motor coordination. At admission in the emergency department, ventilatory assistance was required, and the patient presented progressive improvement after oral levothyroxine reposition. Differential diagnosis of the reduction of consciousness highlighted the presence of infection and stroke. They were not detected. The ultrasonographic analysis showed a diffuse and heterogeneous enlargement of thyroid.

Conclusion: Despite the possibility of adrenal insufficiency, pan hypopituitarism, infection and even a tumour, the thyroid stimulating hormone and free thyroxin levels were determinant to make the diagnosis of myxedematous coma according the signs and symptoms of the patient's entrance presentation. Myxedema coma is a syndrome that results from the intense reduction of the thyroid hormone synthesis.

INTRODUCTION

Thyroid gland disorders are pathologies ordinarily found in the clinical practice. It is estimated that 5.9 to 11.7% of the population presents alterations in laboratory exams performed for the analysis of the thyroid function [1]. Hypothyroidism is the most frequent disorder, and when not properly treated it may generate relevant consequences such as the myxedema coma, also known as myxedema crisis.

Not very frequent, with an estimated number of 0.22 million cases/year, it occurs due to the acute suppression of thyroid hormone production. Cases of myxedema coma usually occur in women with a significant mortality rate (25-60%) even when they are under proper treatment [2,3].

Myxedematous coma is a syndrome that results from the intense reduction of the thyroid hormone synthesis. There are some triggering factors like exposition to low temperatures, infections, irregular performance of hormone replacement therapy in patients with hypothyroidism, adverse reactions of

medications like amiodarone, lithium, anesthetics, hypnotics, sedatives or tranquilizers. This syndrome may also be triggered by the intake of raw foods that, when broken down by myrosinase, release substances that inhibit the absorption of iodine. The cooking process deactivates the enzyme, and thus it does not interfere with the production of thyroid hormones [2].

This case refers to a patient who reported not having previous thyroid disorders and presented with hypothyroidism with myxedema coma.

CASE PRESENTATION

A 58-year-old black patient from the southeastern region of Brazil was brought into the emergency department. She was admitted owing to drowsiness that lasted for seven days, reduction of motor coordination and slight mental confusion. The patient were hypertensive, diabetic, obese and had atrial fibrillation. Her past surgical records a mastectomy due to breast cancer followed by radiotherapy and chemotherapy eight years before her admission.

She regularly takes metoprolol 25 mg/day, metformin 1700 mg/day, fluoxetine 20 mg/day and amiodarone 200 mg/day.

At admission in the emergency room her vital signs revealed a blood pressure of 130/90 mmHg, heart rate of 83 bpm, axillary temperature of 95° F and pulse oximetry of 95% with no supplementation of oxygen.

The patient presented with dry skin, generalized slight pitting edema and drowsiness without motor or sensory deficits. She obeyed verbal commands with a score in the Glasgow Coma Scale of 13 (2/5/6). Inspection and palpation of the cervical region was performed and no goiter or neck stiffness was found.

The initial laboratory evaluation revealed the absence of anemia and electrolyte disturbances, however there were reduced renal function with an urea level of 71 mg/dl and a serum creatinine of 2 mg/dl. These alterations might be due to the dehydration presented at admission. Thyroid function was altered with thyroid-stimulating hormone (TSH) of 16.75 mU/L and free thyroxine (T4) of 0.59 ng/dl.

Her consciousness level worsened in the 10 hours that followed her admission and orotracheal intubation along with mechanical ventilation were made necessary.

The syndromic diagnosis for the decreased level of consciousness required the performance of a skull tomography so that the possibility of a stroke could be excluded. It is important to point out that the patient had undergone previous breast cancer treatment, and based on the presented case a central nervous system tumor could justify part of the symptoms. No tumor or stroke was found in the tomography. Spinal fluid analysis dismissed the hypothesis of meningitis as well as sepsis with the previous laboratory findings.

The acute thyroid insufficiency leading to coma could not be disregarded due to the combination of several factors like the female sex, the chronic use of amiodarone and the picture of acute drowsiness and motor discoordination.

Once the hypothesis of myxedema coma came up, supplementation with an attack dose of 200 mcg of levothyroxine was started, followed by maintenance of 75 mcg on daily basis, after the analysis of the thyroid stimulating hormone and free thyroxine levels. A daily supplementation with 300 mg of hydrocortisone was also administered prior to serum cortisol collection. After one day of mechanical ventilation and the onset of the empiric supplementation, the patient regained consciousness and was extubated.

There were no alterations in the levels of antimicrobial and antiperoxidase antibodies with respective values of 17.9 (reference < 35) and 20 (reference < 40). Morning cortisol value was 25.3 mcg/dL with reference value of 5-25 mcg/dL.

The ultrasonographic analysis of the thyroid showed a diffuse heterogeneous appearance of the echotexture with acinar ectasia, and the conclusion was diffuse goiter.

DISCUSSION

Hampton J in a recent series reported that about 12% of

people will experience some issue with thyroid in their lifetime [5].

According to this finding, our patient evolved with thyroid insufficiency aggravated by insufficient treatment and its complication like coma.

The most dangerous complication is related with opposite sides: hyperfunction, the so called "thyroid storm" which is characterized by an enormous release of thyroid hormone into the circulation with a violent response of the organs¹ and hypofunction, the myxedema coma.

In this case, there was no thyroid storm but the opposite because the lack of production of thyroid hormone. Among the clinical and laboratory alterations found in the myxedema crisis, the presence of xeroderma, hairy rarefaction, hoarseness, non-pitting peripheral edema, macroglossia and impairment of deep reflexes can be observed [4].

As part of the syndrome, the presence of hypothermia, decreased level of consciousness, amnesia, hallucinations and seizures, gastric and enteric atony with possible ileus, ascites and gastrointestinal bleeding may be observed. Among the hematologic disorders, normocytic and normochromic anemia, von Willebrand disease, reduction in factors V, VII, VIII, IX and X production, decrease of granulocytes and an increased risk of disseminated intravascular coagulation are usually reported [2,3].

Hormone replacement therapy must be conducted early so that the physiological levels of hormones may be reached as soon as possible.

Oral supplementation proved to be as effective as the intravenous route [2]. However, owing to the presence of gastric atony in many patients, oral therapy may be disadvantageous once there is a lower absorption of the drug in such cases. Whenever available, triiodothyronine replacement is more effective than T4 replacement and it leads to the improvement of clinical and neurological symptoms in approximately 24 hours [3,5].

On account of the increased risk of association with adrenal insufficiency in these patients, it is prudent to start the replacement of corticosteroids with hydrocortisone, 50-100 mg/day for 7 days and weaning after clinical and laboratory improvement [2].

Despite the fact the outcome of the reported case was favorable, the evolution of a myxedema coma patient is bad even with the proper and early treatment. Aggravating factors include advanced age, decreased level of consciousness, and high APACHE II and SOFA scores [2]. The most probable triggering cause in the current case was the progressive worsening of thyroid and the lack of proper hormone replacement.

In conclusion, thyroid function must be analysed routinely because the high prevalence of dysfunction. Myxedematous coma can be extremely dangerous when it's not recognized promptly. Whenever available, iv triiodothyronine replacement is more effective than iv T4 replacement. Intravenous route must be the preferential option due to the lower risk of enteric absorption which is inherent to the syndrome.

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