OSP Journal of Case Reports

Case Report

SARS COV-2 Infection Revealing Myxedema Coma: A Case Report

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Received: October 15, 2021; **Accepted:** October 19, 2021; **Published:** October 30, 2021

Abstract

Myxedema coma is a life-threatening complication of decompensated hypothyroidism and remains a difficult diagnosis in the absence of known hypothyroidism. The treatment of myxedema coma consists of hormone substitution, treatment of the triggering factor such as infection, as well as adjuvant measures of resuscitation. Herein, we report a case of myxedema coma caused by SARS-CoV-2 pneumonia and discuss how Covid- 19 has unveiled an undiagnosed hypothyroidism in our patient.

Keywords

SARS COV-2; Hypothyroidism; Myxedema Coma; Intensive Care; Prognosis

Introduction

Coma myxedema is a rare but serious complication of hypothyroidism which is most often triggered by an infectious process [1]. However, the impact of hypothyroidism on the natural history of SARS-COV2 pneumonia remains not fully understood [2].

Case Report

We report a case of a 50-years-old woman, non-vaccinated, with a history of osteoarthritis under anti-inflammatory drugs and a depressive syndrome under fluoxetine hydrochloride. The patient reported one week before her admission a cough associated with respiratory discomfort and myalgias, all in a febrile context with progressive worsening of symptoms.

The patient was admitted to the intensive care unit due to her critical respiratory condition. The clinical examination on admission revealed a confused patient with a Glasgow coma scale estimated at 12/15, the pupils were symmetrical and reactive with a morbid obesity, a body mass index of 30 kg/m2. She was apyretic with a blood pressure of 132/93 mmHg, bradycardic at 45 beats per minute, polypneic at 28 cycles per minute, with a pulsed oxygen saturation of 40% in the open air rising to 92% under non-invasive ventilation. The patient had a typical facies, dry skin and mucous membranes, with a hoarseness and slowness of voice. A thoracic computed tomography scan was performed showing lesions compatible with SARS-COV2 viral pneumopathy with an estimated 75% parenchymal damage but there were no signs of pulmonary embolism. The RT-PCR of the nasopharyngeal swab came back positive. The electrocardiogram showed a regular sinus rhythm, fixed PR at 0.18 seconds, fine QRS with aspect of incomplete left bundle branch block and peripheral microvoltage. The cardiac ultrasound showed a non-dilated left ventricle, an ejection fraction at 55%, a non-dilated right ventricle, systolic pulmonary artery pressure at 21mmHg, a compliant inferior vena cava at 11mm, a small pericardial effusion of 8mm opposite the right ventricle and 8mm posteriorly. Biologically: the blood ionogram showed no abnormalities, the liver function tests were slightly disturbed with ASAT/ALAT at 91/65 IU/L. Renal function was preserved (urea at 0.39 g/l, creatinine at 12.4 mg/l), troponin was 600UI/L. With regard to infection: C-reactive protein was positive at 178 mg/l with Procalcitonin at 0.79 mg/ml, hyperleukocytosis with neutrophile predominance (White blood cells= 18180/mm3; neutrophiles at 16070/mm3, lymphocytes at 1270/mm3). The hemogram was normal with a hemoglobin level of 11.2 g/dl and platelets of 36,000/ mm3. The haemostasis work-up was correct with a prothrombin ratio at 100%, fibrinogen at 5, 38g/l, D-dimer at 1000 mcg/l.

Therapeutically, the patient was put under non-invasive ventilation with antibiotic therapy based on third generation cephalosporin type ceftriaxone, systemic corticosteroid therapy based on methylprednisolone, curative low molecular weight heparin therapy associated with aspirin, gastric protection with omeprazol, vitamin therapy C & D, Zinc.

The clinical evolution was marked by the worsening of her neurological and respiratory status with a Glasgow score of 8/15 for which the patient was intubated and ventilated. The patient then displayed a bradycardia at 40 beats per minute without hemodynamic instability, a polyseritis with

a worsening of the pericardial effusion in circumferential of 12 mm. The whole associated with a bilateral oedema of lower limb. Therefore, a thyroid assessment was imposed: Free thyroxine (T4) at 0.3 ng/dl, Triiodothyronine (T3) lower than 0.1 ng/dl, thyroid stimulating hormone TSH at 21 mU/L. The diagnosis of hypothyroidism complicated by a myxedema coma was retained. An intravenous treatment based on L-thyroxine was thus administered at a dose of 300 micrograms associated with hydrocortisone hemisuccinate at a rate of 100 mg/d with a very good clinical evolution.

Sedation was discontinued at day 17 of hospitalization and the patient was extubated after successful completion of the ventilatory weaning test. Post-extubation clinical assessment noted a conscious patient 15/15 in the Glasgow coma scale, symmetrical reactive pupils without sensitivomotor deficits, eupneic at 16cpm with SPO2 at 98% in open air, hemodynamically stable with a blood pressure at 130/75 mmhg and heart rate at 68bpm, apyretic at 37°C. The patient was transferred to the endocrinology department for further management.

Discussion

Myxedema coma is an extremely rare form of decompensated hypothyroidism, with an estimated incidence of 1.08 cases per million people [4]. Our patient presented many of the clinical signs linked to myxedema coma, including coma, hypoventilation and bradycardia [5]. Myxedema coma occurs when a frustrating factor disrupts thyroid hormone regulation in an already hypothyroid patient [6]. Thyroid hormone deficiency is often first detected during the crisis, as in our case [7]. Low T3 and T4 levels suggest pre-existing undiagnosed hypothyroidism, which may be revealed by SARS COV-2 infection. Thyroid hormone is an important regulator of the innate and adaptive immune system, modulating the proliferation and function of lymphocytes, macrophages, and dendritic cells.8 Therefore, patients with hypothyroidism may be at increased risk for infections and subsequent complications such as myxedema coma [8]. Viral infections are frequently cited as a major environmental factor implicated in thyroid disease [9]. However, a direct association between hypothyroidism and viral infections has yet to be established [10]. Autopsy results of patients infected with SARS-CoV-1 in the early 2000s revealed destruction of the thyroid follicular epithelium and signs of apoptosis [11].

In view of all the elements collected from the patient, we hypothesize that our patient had a decompensation of a pre-existing undiagnosed hypothyroidism caused by a SARS-CoV-2 infection. Our patient had classic features of severe COVID-19: markedly elevated inflammatory markers and a chest CT scan showing lesions consistent with SARS-COV2 viral pneumonitis with an estimated 75% parenchymal involvement. SARS-CoV-2 invades cells via the angiotensin converting enzyme 2 (ACE2) receptor [12]. The ACE2 receptor is highly expressed in thyroid tissue, which may lead to direct thyroid damage by the virus, although this needs to be confirmed by autopsy studies [13].

Owing to the high mortality rate, rapid intravenous infusion of thyroid hormone is the standard treatment for myxedema coma [14]. High doses (e.g., levothyroxine >500 mcg/d or liothyronine >75 mcg/d) are associated with higher mortality in patients older than 65 years old and/or with underlying heart conditions, which was not the case in our patient, though [15].

Conclusion

Myxedema coma is often a difficult diagnosis and represents the most serious complication of hypothyroidism. Hence the interest of an early diagnosis and therefore an early and effective management [16]. Nevertheless, as the diagnosis is relatively rare, it is bound to be hidden by the Covid-19 symptoms making thus the prognosis poorer.

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