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Normothermic Thyroid Storm with Circulatory Collapse: An Unusual Presentation

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Abstract Thyroid storm is a rare, life-threatening condition characterized by severe clinical manifestations of thyrotoxicosis. Hyperpyrexia to 104 to 106°F is common. It is usually precipitated by noncompliance with medication, infection, thyroid or non-thyroidal surgery, and parturition. In this article, we present a case of a young 39-year-old male patient with no significant history of thyroid disorder who presented with palpitation and shortness of breath. The patient was diagnosed with normothermic thyroid storm without any precipitation factor. The patient had circulatory collapse with shock and acute respiratory failure needing ventilatory and ionotropic support. The patient was appropriately treated and discharged home. The patient was normothermic during the entire hospital stay.

Keywords*: normothermic, thyroid storm, circulatory collapse*

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1. Case Description

A 39-year-old male with no significant medical history presented to an emergency department with palpitation and shortness of breath for the last 2 weeks. The patient had associated nausea, anxiety, and restlessness. On presentation, had temperature 36°C blood pressure 110/80 mm of Hg. Patient has a normal eye and thyroid examination. EKG showed new-onset atrial fibrillation with a rapid ventricular rate of 180 beats per minute. Patient had a thyroid-stimulating hormone <0.015 mIU/ml (0.465–4.860), elevated free T4 5.53 ng/dl (0.79-2.35) and elevated free T3 31.30 pmol/L (3.56-9.35). Patient had elevated liver function with total bilirubin of 2.0 mg/dl, AST 1205 unit/L (17-69) and ALT 605 unit/L (21-72) and negative urine drug screen. Based on Burch and Wartofsky's scoring system, the patient had a score of 65, which is highly suggestive of thyroid storm. The patient was transferred to an intensive care unit. The patient was treated with intravenous hydrocortisone, methimazole, potassium iodide (SSKI), and propranolol. The patient's condition deteriorated to coma, shock, needing ventilatory, and ionotropic support. The patient had a negative blood culture; the X-ray chest was negative for pneumonia, and urine did not show any bacteria. The patient has elevated thyroglobulin antibody S, thyroperoxidase antibody S, and thyrotropin receptor antibody S. The Patient had echocardiography that showed the left ventricle ejection of 25-30% and had normal cortisol level. The patient stayed in the hospital for 7 days, successfully extubated with normal vitals. On the day of discharge, the patient had TSH <0.015 mIU/ml with normal free T3 5.58 pmol/L and free T4 1.57 ng/dl and normalization of liver function. The patient was normothermic during the whole hospital stay. On the day of discharge, the patient had a temperature of 37°C, blood pressure 113/81 mm of Hg heart rate 73 beats per minute, and a respiratory rate of 18 per minute. Repeat echocardiography showed the resolution of left ventricular systolic dysfunction. The patient was discharged on methimazole and metoprolol to have followed up with primary care physician and endocrinology.

2. Discussion

Thyroid storm is a rare medical emergency caused by exacerbation of hyperthyroid state with high mortality unless recognized and treated early. Thyroid storm is characterized by fever, tachyarrhythmias, gastrointestinal, and central nervous system symptoms. [1] Diagnosis of thyroid storm is clinical, supported by biochemical evidence of abnormal thyroid function as well as systemic decompensation due to exaggerated symptoms of thyrotoxicosis. Burch and Wartofsky proposed a scoring system based on clinical Criteria in which score >45 is highly suggestive of thyroid storm. [2] Thyroid storm mortality is high as 10%. [3] our patient had Burch and Wartofksy score >65 with strong evidence of much suppressed TSH with elevated FT3 and FT4.

Unusual presentations like a normothermic, hepatic failure and apathetic thyroid storm are rare. Our case of thyroid storm has strong and unusual points. First, the patient has no previous history of thyroid disease, so presentation as thyroid storm is uncommon. 2nd Thyroid storm is a hypermetabolic state that usually presents with high cardiac output failure, but our patient had low cardiac

out failure because of underlying systolic dysfunction and propranolol use. [4] Propranolol was used in 80 mg dose, but the patient has no prior feature of congestive heart failure, including any peripheral edema, orthopnea, and paroxysmal nocturnal dyspnea. According to Japenese endocrinology society guidelines, more selective beta-blocker like esmolol IV or bisoprolol should be used [5]. 3rd unusual feature is the severity of liver dysfunction with an elevation of transaminases AST> 1000 unit/L as most of the thyroid storm has a mild elevation of transaminases. [6] The reason for elevation in transaminases is a multifactorial increase in oxygen consumption can lead to hypoxia, particularly in the perivenular region. One study showed simple atrophy, sinusoid congestion, and fatty metamorphosis in hepatocytes [5]. Alcohol abuse and prior history of liver disease could be a contributing factor, but our patient had none of those. 4th our patient is normothermic during the whole hospital stay. Many case studies have shown that thyroid storms present with hyperthermia, normothermia is rare. [6] Hyperthermia is usually due to abnormal thermoregulation of central nervous system with increased thermogenesis and elevated basal metabolic rate beyond body's capacity to dispel heat. [7] 5th our patient has no known precipitating factor. Most common precipitating factors are infection, parturition, trauma to thyroid gland, surgery and emotion stress. [8] 6th our patient has complete recovery with no neurological and cardiovascular deficit before discharge. As literature has shown, that patients may have protracted neurological dysfunction after normalization of thyroid function test. [9]

Treatment of thyroid storm is based on the same principle of non-critical hyperthyroidism, but medications are given in higher doses, increased frequency, and intensive care unit monitoring. The patient needs several different medications for adequate treatment.[10]. Beta-blocker, mainly non-selective, should be used to decrease the sympathomimetic effect. Propranolol can be used. Underlying acute systolic congestive heart failure and other contraindication should be considered. Propranolol can be started at a low dose, mainly 10-20 mg every 6 hours up to 320 mg per day to control symptoms and heart rate. [11] Glucocorticoid (hydrocortisone/dexamethasone) should be used. It decreases peripheral conversion of T4 to T3 but helpful in associated adrenal insufficiency and control of vasomotor symptoms. [12] Thionamides, which include propylthiouracil and methimazole, are used. These block synthesis of thyroid hormones. In addition to this, propylthiouracil also decreases peripheral conversion of T4 to T3. Iodine solutions block the release of thyroid hormones. Because Iodine can be used for the synthesis of new hormones, it must be used at least one hour after administering Thionamides. Bile acid sequestrants such as cholestyramine can be used to decrease enterohepatic solutions and enhance fecal excretion. [13] Other treatment includes radioactive Iodine, surgery, and plasmapheresis can be used if traditional treatment fails. [14]

3. Conclusion

It can be an initial presentation of the hyperthyroid state. Atypical presentations like the normothermic, absence of precipitating factors, and no history of hyperthyroidism should not preclude the diagnosis of thyroid storm. The infrequency with which normothermic thyroid storm is encountered makes it a diagnostic challenge for Clinicians.

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