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Thyrotoxicosis: Etiology, Epidemiology, Pathophysiology and its Deadly Complications

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Description

Thyrotoxicosis is the state in which thyroid hormones are excessively produced. Thyrotoxicosis, also known as a thyroid storm, is an uncommon but serious disorder that requires medical intervention right away, ideally in an intensive care unit. It occurs in patients with overt hyperthyroidism at a rate of 1%-2% [1].

It is a severe, perhaps deadly side effect of having an overactive thyroid. Possibly it is an exaggerated presentation of hyperthyroidism. There is a sudden multisystem involvement with it. Despite recent breakthroughs in its treatment and supportive measures, thyroid storm mortality is still thought to range from 8% to 25%. Thus, it is crucial to identify it early and begin vigorous therapy to lower mortality.

Etiology

Graves' disease is the most common cause of thyrotoxicosis but it can also occur with other etiologies for example toxic multinodular goiter and toxic thyroid adenoma.

The predisposing factors are:

- Thyroid surgery
- Non thyroid surgery
- Trauma
- Burns
- Parturition
- Cardiovascular disorders, diabetic ketoacidosis, acute myocardial infarction.

SARS-CoV-2 infection (this can trigger a thyrotoxicosis crisis and acute decompensated heart failure even without having multi-system inflammatory syndrome in children).

COVID-19. A study reported thyroid dysfunction during and after COVID-19. It is hypothesized that thyroid dysfunction during COVID-19 is brought on by an autoimmune reaction to the thyroid or by a direct thyroid infection due to a "cytokine storm". Studies have also shown that COVID-19 induced thyroid disorders include Graves' disease, euthyroid sick syndrome, Hashimoto's thyroiditis and subacute thyroiditis [2].

If a patient with a traumatic head injury has underlying hyperthyroidism, this condition can also lead to thyroid storm.

- Side effects of medications like Amiodarone.
- Hyperemesis gravidarum.

Epidemiology

It is a rare form of hyperthyroidism. Thyroid storm accounts for about 1% to 2% of hyperthyroidism admissions. According to a survey conducted in the United States, the incidence of storms ranged from 0.57 to 0.76 cases per 100,000 people per year in the general population and 4.8 to 5.6 cases per 100,000 people per year in hospitalized patients [3].

The incidence of thyroid storm was 0.2 per 100,000 population per year, accounting for about 0.22% of all thyrotoxicosis patients and 5.4% of hospitalized thyrotoxicosis patients, according to the Japanese national survey. People with thyroid storm were 42 to 43 years old on average, which was similar to those with thyrotoxicosis without thyroid storm. The male to female ratio for thyroid storm incidence was about 1:3, which was similar to the thyrotoxicosis without storm group [4].

Pathophysiology

Thyrotoxicosis is caused by an excess of thyroid hormone, either from endogenous over secretion of T3 and T4 or from the ingestion of synthetic thyroid hormone [5]. The thyroid hormone affects nearly every tissue and organ system in the body by increasing basal metabolic rate and tissue metabolism through the upregulation of alpha adrenergic receptors, leading to a boost in sympathetic activity. Thyroid hormone increases the expression of myocardial sarcoplasmic reticulum calcium dependent ATP, which raises heart rate and myocardial contractility, resulting in increased cardiac output. Reduced Systemic Vascular Resistance (SVR) and afterload result from arterial smooth muscle relaxation caused by metabolic end products such as lactic acid produced by increased oxygen consumption. Reduced SVR activates the renin-angiotensin system, increasing sodium reabsorption and expanding blood volume to increase blood pressure. If left untreated, it can lead to heart failure [6,7].

Complications

A thyroid storm can occur if thyrotoxicosis is left untreated or undiagnosed. Patients have tachycardia, fever, alterations in mental status, agitation, cardiac failure symptoms, and impaired liver function. To identify a precipitating event, like major stress, illness, or a recent injury, a careful history is required. Thionamide therapy with Methimazole or PTU is used to prevent the synthesis of new thyroid hormone and iodine is used to prevent the release of the pre formed hormone [8]. In a critical care setting, beta blockers such as

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propranolol and fluid resuscitation are commonly used. Different complications are as follows:

Psych or CNS:

- Delirium.
- Altered mental status.
- Psychosis.
- Meningitis.

Musculoskeletal system:

Thyrotoxic myopathy is characterized by muscle weakness, proximal muscle wasting, fatigue, and heat intolerance. The core muscles are those of the pelvic girdle and shoulder, with infrequent complicity of the bulbar and oropharyngeal muscles, resulting in swallowing and respiratory difficulties. The weakness is usually substantial to the degree of objective muscle wasting, with normal or hyperactive deep tendon reflexes [9].

Cardiac

Thromboembolism: According to research, hyperthyroidism increases the risk of thromboembolic events. There is no agreement on when to start anticoagulation for atrial fibrillation in severe thyrotoxicosis [10]. Anticoagulation is not routinely started if the risk on a CHADS2 score is low; however, it's worthwhile to be considered for those with thyroid storm or severe thyrotoxicosis with the impending storm, regardless of CHADS2 risk, because it appears to increase the risk of thromboembolic episodes [11].

- Cardiovascular collapse.
- Cardiomyopathy.
- Death.

Treatment

Antithyroid medications and radioactive iodine are the treatment for hyperthyroidism [12,13].

The primary therapeutic option for thyrotoxicosis/hyperthyroidism is medical treatment. For therapeutic purposes, two types of thyrotoxicosis must be considered: Thyrotoxicosis with hyperthyroidism and thyrotoxicosis without hyperthyroidism [14]. Conventional therapies include beta-blockers, Antithyroid Drugs (ATDs), corticosteroids, inorganic iodide, perchlorate, cholecystographic agents, lithium and cholestyramine.

Surgical intervention ought to be considered in cases of extreme goiter, severe ophthalmopathy, pregnancy, persistent hyperthyroidism despite anti-thyroid medication and radioactive iodine treatment, and individual preferences [15].

Potassium perchlorate can also be used as a treatment for thyrotoxicosis or combined with propylthiouracil [16].

Beta blockers such as propranolol are given to treat thyroid hormone excess effects in peripheral tissues [17].

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