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LESSON INSTRUCTION TO EXPLAIN THERIOTOXICOSIS TO STUDENTS IN THE EASIEST WAY

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Annotation: Dear author this article provides information on the pathophysiology and physiology of thyrotoxicosis. This article is written in a case that uses internet resources.
Key words: thyrotoxicosis, thyroid hormone, blood, osteoporosis, muscle weakness, atrial fibrillation.

Thyrotoxicosis is the clinical state associated with excess thyroid hormone activity, usually due to inappropriately high-circulating thyroid hormones. The clinical presentation varies, ranging from asymptomatic to life-threatening thyroid storm. Symptoms are due to the hypermetabolic state induced by excess thyroid hormones and include weight loss, heat intolerance, and palpitations. There are many different causes of thyrotoxicosis. It is important to determine the cause since treatment is based on the underlying etiology. Thyrotoxicosis can lead to serious complications when not diagnosed and treated appropriately, including delirium, altered mental status, osteoporosis, muscle weakness, atrial fibrillation, congestive heart failure, thromboembolic disease, cardiovascular collapse, and death. This activity reviews the evaluation and management of thyrotoxicosis and highlights the role of interprofessional team members in collaborating to provide well-coordinated care and enhance outcomes for affected patients.

Thyrotoxicosis is a clinical state of inappropriately high levels of circulating thyroid hormones (T3 and/or T4) in the body from any cause. It is often incorrectly used interchangeably with hyperthyroidism, which is a form of thyrotoxicosis caused by excessive endogenous thyroid hormone production.

The clinical presentation varies, ranging from asymptomatic or subclinical, to life-threatening thyroid storm. Typical symptoms are due to the hypermetabolic state induced by excess thyroid hormones and include weight loss, heat intolerance, and palpitations. The differential for thyrotoxicosis is broad and will need a combination of a thorough physical exam, laboratory studies, and imaging to determine the underlying etiology for appropriate treatment. If not adequately treated, thyrotoxicosis can lead to serious complications including delirium, altered mental status, osteoporosis, muscle weakness, atrial fibrillation, congestive heart failure (CHF), thromboembolic disease, cardiovascular collapse, and death.

The prevalence of thyrotoxicosis in the United States is 1.2%, including 0.5% overt thyrotoxicosis and 0.7% subclinical. The incidence of thyrotoxicosis peaks between ages 20 and 50 years. Graves' disease is the most common cause with an incidence of 20 to 50 cases per 100,000 persons followed by toxic multinodular goiter and toxic adenoma. Graves' disease most commonly affects women aged 30 to 50 with a male to female ratio of 5 to 1 but can occur at any age in both genders. Toxic nodular goiter increases with age and in iodine-deficient regions. Thyroiditis accounts for 10% of cases. One percent to 2% of patients with thyrotoxicosis go on to develop the serious complication of thyroid storm.

Thyrotoxicosis results from thyroid hormone excess either from endogenous over-secretion of T3 and T4 or from exogenous ingestion of synthetic thyroid hormone. Thyroid hormone affects almost every tissue and organ system in the body by increasing basal metabolic rate and tissue thermogenesis by upregulating alpha-

adrenergic receptors leading to an increase in sympathetic activity. Thyroid hormone causes increased expression of myocardial sarcoplasmic reticulum calcium-dependent ATP, increasing heart rate and myocardial contractility with the net effect of increased cardiac output. Decreased systemic vascular resistance (SVR) and decreased afterload results from arterial smooth muscle relaxation by metabolic end products, such as lactic acid, produced with increased consumption of oxygen. Decreased SVR leads to activation of the renin-angiotensin system, increasing reabsorption of sodium and expanding blood volume to increase preload. If left untreated, this may lead to left ventricular hypertrophy and congestive heart failure. Graves' disease is an autoimmune disease comprised of antibodies that stimulate TSH receptors to cause excess secretion of thyroid hormones via a type II hypersensitivity reaction. This results in hyperplasia of thyroid follicular cells causing a diffuse goiter. The cause of Graves' disease is not known, but genetic and environmental factors, such as smoking, stress, and dietary iodine play a role. The thyroid-stimulating immunoglobulin (TSI) triggers the hyperthyroidism. In toxic multinodular goiter and toxic adenoma, autonomously functioning nodules over-secrete thyroid hormone independently without stimulation from TSH. Rarely, these Nnontoxic adenomas or goiter can convert to toxic adenomas after exposure to iodinated contrast, such as from a cardiac catheterization or undergoing a CT study with contrast.

In thyroiditis, thyrotoxicosis is caused by the release of preformed thyroid hormone into the circulation as inflammation destroys thyroid follicles. This causes transient thyrotoxicosis that most often self-resolves. Inflammation can be precipitated by a variety of insults to the thyroid gland, including autoimmune, infectious, chemical, or mechanical insults.

Gestational hyperthyroidism generally occurs in the first trimester of pregnancy, due to increased stimulation of the thyroid gland by excess human chorionic gonadotropin (HCG), which is similar in structure to TSH and binds the TSH receptor.

Patients with thyrotoxicosis most commonly present with signs and symptoms related to excess thyroid hormone including: weight loss with a normal or increased appetite, heat intolerance with increased sweating, palpitations, tremor, anxiety, proximal muscle weakness, alopecia and increased fatigability. Sinus tachycardia is the most common cardiac rhythm problem, but atrial fibrillation may occur and is frequently seen with advanced patient age, valvular disease, and coronary artery disease. Women may present with amenorrhea or oligomenorrhea. Men may present with gynecomastia rarely. It is common for older patients to manifest fewer of the typical clinical manifestations and instead present with depression, fatigue, and weight loss, also known as apathetic thyrotoxicosis.

On physical exam, patients are often cachectic, hyperthermic, diaphoretic, and anxious appearing. They may have goiter, tachycardia or atrial fibrillation, dyspnea, abdominal tenderness, hyperreflexia, proximal muscle weakness, tremor, and gynecomastia. Patients with Graves' disease present with pretibial myxoedema, thyroid acropachy, and onycholysis.

In rare cases, patients present in thyroid storm with tachycardia, fever, altered mental status, agitation, features of cardiac failure, and impaired liver function.

Findings specific to Graves' disease include ophthalmopathy resulting in proptosis, chemosis, conjunctival injection and lid lag, exposure keratitis and extra-ocular muscle dysfunction, pretibial myxedema and thyroid acropachy (clubbing).

In subacute thyroiditis, patients have a history of recent upper respiratory illness and generally present with fever, neck pain and swelling with a firm and tender thyroid gland. Painless thyroiditis often presents in the postpartum period, and patients frequently have a personal or family history of autoimmune or thyroid disease. Suppurative thyroiditis presents with a tender, erythematous mass in the anterior neck and patients often complain of fever, dysphagia, and dysphonia.

Occasionally, patients may present with acute muscle paralysis and severe hypokalemia, termed thyrotoxic periodic paralysis.

Low serum TSH (less than 0.01 mU/L) has high sensitivity and specificity for the diagnosis of thyroid disorders. If TSH is low, elevated serum free thyroxine (T4) and triiodothyronine (T3) levels can distinguish between overt and subclinical hyperthyroidism. Usually, the increase in T3 precedes the increase in T4. Pituitary-dependent causes of hyperthyroidism may have normal or increased TSH levels and increased T4 and T3 levels with an increase in free-alpha subunit concentrations. Serum levels of antibodies to the TSH receptor diagnose Graves' disease. Levels are 98% sensitive and 99% specific. Thyroid peroxidase antibodies are only present in about 75% of cases of Graves' disease.

Radioactive iodine uptake studies or thyroid scans may be used to distinguish between causes of thyrotoxicosis besides Graves' disease. It is recommended in all thyrotoxic patients without the clinical picture of Graves' disease. In Graves' disease, radioactive iodine uptake is diffuse, unless the patient also has nodules or fibrosis. In single toxic adenoma, there will be a focal uptake in the adenoma, with suppressed uptake in the surrounding thyroid tissue. Toxic multinodular goiter will show multiple areas of focal increased uptake and suppressed uptake in surrounding tissue. Radioactive iodine uptake will be near zero in patients with painless, postpartum, or subacute thyroiditis, as well as patients with ingestion of thyroid hormone or recent excess iodine exposure.

In subacute thyroiditis, inflammatory markers such as erythrocyte sedimentation rate and C-reactive protein are frequently increased. In pregnancy, either free T3 and T4, or the total T3 and T4 with adjusted reference range 1.5 times the nonpregnant range should be used for diagnosis in addition to serum TSH levels. During the first half of pregnancy, the serum TSH levels may be lower than the nonpregnant reference range, but free T4 values should be normal. In suspected factitious hyperthyroidism, thyroglobulin levels are decreased and radioactive uptake studies due to the suppression by exogenous thyroid hormone ingestion.

The recommended treatment of thyrotoxicosis is dependant on the underlying cause. Beta-blocker therapy such as propranolol, is used to reduce adrenergic features such as sweating, anxiety, and tachycardia. There are 3 mainstays of treatment: thionamide drugs, radioiodine, and thyroid surgery.

Thionamide drugs include propylthiouracil (PTU) and methimazole and reduce the production of thyroid hormone by acting as preferential substrates for thyroid peroxidase. In high doses, PTU also decreases peripheral conversion of T4 to T3.

In the treatment of Graves' disease, methimazole is used at a dose of 15 mg to 30 mg per day for 4 to 8 weeks, after which most patients become euthyroid. After patients become euthyroid, there are 2 approaches to treatment. First, in the block-replace method, the same dose of thionamide is continued to block thyroid hormone production, and levothyroxine is added to maintain euthyroidism. Alternatively, the thionamide dose may be titrated progressively to allow endogenous synthesis of thyroid hormone, maintaining a euthyroid state.

In Graves' disease, long-term remission is achieved in about 50% of patients with thionamide drugs. A disadvantage of thionamides is the uncertainty of relapse after treatment is stopped. No advantage has been shown in remission rates with prolonged treatment beyond 18 months. Methimazole has been shown to have better efficacy and has a longer half-life, which allows once-daily dosing. Additionally, there is a higher risk of hepatotoxicity with PTU. Agranulocytosis occurs in 1 in 300 patients treated with thionamides and presents as a sore throat, mouth ulcers and high fever. It is recommended to obtain a differential white blood cell count during febrile illness and pharyngitis in all patients on thionamides. Minor side effects include pruritis, arthralgia, and gastrointestinal upset.

Radioiodine therapy is the most common therapy used for adults with Graves' disease in the United States. It can also be used for toxic nodules and TMNGs. Radioactive iodine is given in one oral dose. It is absorbed by the thyroid gland inducing tissue-specific inflammation that leads to thyroid fibrosis and destruction of thyroid tissue over the next several months. Hypothyroidism usually occurs within 6 to 12 months. Most patients go on to require lifelong levothyroxine treatment. There is a small risk of thyrotoxicosis exacerbation in the month after treatment due to the release of the preformed hormone. Patients with large goiters, severe thyrotoxicosis, ischemic heart disease, heart failure or arrhythmia are

recommended to use thionamide pretreatment until they are euthyroid before radioiodine therapy. Radioiodine therapy is contraindicated in pregnancy and lactation and relatively contraindicated in active inflammatory Graves' ophthalmopathy.

Total or partial thyroidectomy is a rapid and effective method of treating thyrotoxicosis. However, it is invasive and expensive, and causes permanent hypothyroidism, requiring levothyroxine treatment. It is recommended that patients be pretreated for euthyroidism before surgery to reduce the risk of worsening thyrotoxicosis and thyroid storm. Complications include hypocalcemia due to hypoparathyroidism, which is usually transient, and vocal cord paresis due to damage to the recurrent laryngeal nerve.

The treatment for thyroiditis differs in that antithyroid drugs are ineffective, since patients usually have low production of new thyroid hormone. It is usually transient, but treatment is aimed at symptom control with beta blockers. In subacute thyroiditis, non-steroidal anti-inflammatory drugs and occasionally systemic glucocorticoids may be used to help with pain and inflammation. Beta blockers are recommended for any elderly patients with symptomatic thyrotoxicosis, and any thyrotoxic patients with resting a heart rate greater than 90 bpm or cardiovascular disease.

Children with thyrotoxicosis may be treated with methimazole, radioiodine therapy or thyroidectomy. Methimazole therapy for 1 to 2 years is the first line therapy for Graves' disease in children since some children will go into remission. Radioiodine therapy is not recommended for children younger than 5 years old. PTU should also be avoided in children due to the risk of hepatotoxicity.

During pregnancy, it is recommended to treat with thionamide drugs in a titrated dose regimen. Block-replace regimens increase the risk of fetal hypothyroidism and goiter. PTU is recommended during the first trimester of pregnancy. PTU is preferred during the first trimester of pregnancy due to the risk of teratogenicity associated with methimazole, including aplasia cutis and choanal or esophageal atresia.

Untreated or undiagnosed thyrotoxicosis can lead to thyroid storm. Patients present with tachycardia, fever, altered mental status, agitation, features of cardiac failure, and impaired liver function. A careful history is needed to identify a precipitating event such as major stress, illness, or a recent injury. Treatment include administration of thionamide therapy with methimazole or PTU to stop the synthesis of new thyroid hormone and Iodine to stop the release of pre-formed hormone. Supportive care with beta-blockers and fluid resuscitation is commonly used in a critical care setting.

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