Hypothyroidism is an interdisciplinary problem.

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Abstract: Hypothyroidism happens when the thyroid gland doesn't make enough hormones. Conditions or problems that can lead to hypothyroidism include.autoimmune disease. the most common cause of hypothyroidism is an autoimmune disease called hashimoto's disease.

Keywords: hypothyroidism, thyroid hormones, hormone excess thyrotoxicosis, coexisting morbidities

The goal of treatment is to return your levels of thyroid hormone back to normal. Untreated hypothyroidism may lead to anemia, low body temperature, and heart failure. Treatment may include medicine that replaces lost thyroid hormones. You usually will need to take thyroid hormones for the rest of your life.

The thyroid is a 15- to 20-gram gland located in the anterior neck. It is responsible for the production of the thyroid hormones T4 (thyroxine) and T3 (triiodothyronine). Various factors can affect thyroid hormone synthesis, including acute illness, coexisting morbidities, and certain medications. Both the states of low thyroid hormone concentrations (hypothyroidism) and thyroid hormone excess thyrotoxicosis) can be transient or permanent. The decompensated, severe forms of hypothyroidism and hyperthyroidism, termed myxedema coma and thyroid storm, are associated with increased morbidity and mortality. Prompt recognition of both conditions is necessary to initiate treatment and supportive measures. This review will summarize the essential principles of the clinical manifestations, diagnostic methods, and treatments of hypothyroidism and hyperthyroidism, both in the nonacute and life-threatening forms of these diseases.

The normal physiology of the hypothalamic-pituitary-thyroid axis involves the production of T4 and T3 by the thyroid gland, a process that is regulated by thyroid-stimulating hormone (TSH) secreted by the pituitary, which is, in turn, regulated by thyrotropin-releasing hormone (TRH) secreted by the hypothalamus. Both serum T4 and T3 concentrations act as negative feedback regulators of TSH and TRH secretion, but can be altered by environmental conditions—including food availability and temperature—and disease states, such as infection.1

Thyroid hormone synthesis is achieved first through active transport of circulating iodide, which is taken in from the diet, by the sodium/iodide symporter located at the basolateral membrane of the thyroid follicular cell.2 Iodide then becomes oxidized by thyroid peroxidase (TPO) and hydrogen peroxide at the apical membrane, which then attaches to the tyrosyl residues on thyroglobulin (Tg) to

produce monoiodotyrosine (MIT) and diiodotyrosine (DIT). MIT and DIT are the precursors to the thyroid hormones, which are produced by the linkage of 2 DIT molecules to form T4 and the linkage of MIT and DIT to produce T3. Release of T4 and T3 into the circulation results from the digestion of Tg in MIT and DIT by endosomal and lysosomal proteases.

The metabolic effects of thyroid hormone action result from the binding of the thyroid hormone to thyroid hormone transporters located in specific target tissues, which is mediated by the thyroid hormone nuclear receptor (TR) that is encoded by the genes TRα and TRβ.3 While the sole source of T4 is the thyroid gland, the majority (approximately 80%) of T3 is produced from the extrathyroidal conversion of T4 to T3 by the action of the 5′-deiodinase enzymes—D1 or D2—located in the liver, brain, brown adipose tissue, and muscles.4 T4 can also be converted to the inactive thyroid hormones, reverse T3 or T2, by the 5′-deiodinase D3. The activity and expression of the deiodinases are specific to different tissues and environmental conditions.

Hypothyroidism refers to the state of low circulating thyroid hormones. In 2 large US population-based studies of data collected in the 1980s to the 1990s, the prevalence of overt hypothyroidism ranged from 0.3% to 0.4%, while that of subclinical hypothyroidism ranged from 4.3% to 8.5%, among the general population.The most common etiology of hypothyroidism in the United States is Hashimoto's thyroiditis, an autoimmune disease that is more prevalent among older women; nutritional iodine deficiency is the most common etiology worldwide. Additional etiologies of hypothyroidism include a history of thyroidectomy, radioactive iodine therapy, and, less commonly, decreased TSH production by the pituitary. Predisposition factors for the development of hypothyroidism include thyroid autoimmunity (which can be assessed by the determination of serum thyroid autoantibodies, such as TPO and [Tg] antibody titers), the use of certain medications (ie, lithium, amiodarone, interferon-alpha), and excess iodine exposure (ie, from iodinated contrast radiographic studies), in which individuals with a history of thyroid disease are at higher risk of iodine-induced hypothyroidism.

Thyroid hormone is important for the metabolic functions of many major organs, including the heart, brain, liver, and muscle. Signs and symptoms of hypothyroidism are widely variable , often subtle, and may include fatigue, malaise, weight gain, dry and puffy skin, constipation, cold intolerance, altered cognition, and hyporeflexia. In children, there may be stunted growth, and in women, menstrual abnormalities may be present. Normal thyroid function is particularly important among pregnant and lactating women, the developing fetus, and young children. As thyroid hormone is crucial for the complex processes of neurodevelopment and growth,8 which begins in the first trimester of pregnancy and continues into the first

few years of infancy, these groups are especially susceptible to the effects of even mild thyroid dysfunction. Several studies have demonstrated that low thyroid hormone levels among pregnant women are associated with increased risks of preterm delivery, spontaneous miscarriage, fetal death, and cognitive deficits,9 including a decrement in intelligence quotient and memory scores of the offspring, compared with euthyroid women.Neuroimaging studies also show abnormalities of hippocampal and corpus callosum size, and of gray matter, among children with a form of low thyroid hormone levels at birth termed congenital hypothyroidism.

Myxedema coma refers to the state of severe, life-threatening, and decompensated hypothyroidism in which thyroid hormone levels are dangerously low. The diagnosis appears to be more common in elderly women with long-standing preexisting hypothyroidism. Triggers may include cold temperature (thus, it is more common during winter months); precipitating comorbidities, such as infection, stroke, and heart failure; or the use of sedative, analgesic, antidepressant, hypnotic, antipsychotic, or anesthetic medications.Patients with preexisting hypothyroidism may also present with myxedema coma following a period of prolonged noncompliance with thyroid hormone replacement.

Signs and symptoms usually are exacerbations of the usual manifestations of hypothyroidism and may include extreme lethargy, which can progress to stupor or coma, hypothermia, respiratory depression, bradycardia, hyponatremia, and renal impairment. The diagnosis of myxedema coma is made with the confirmation of a biochemical thyroid profile consistent with hypothyroidism and corresponding clinical manifestations. A diagnostic scoring system has been proposed to guide the clinician toward a diagnosis of myxedema coma based on body temperature, central nervous system signs, gastrointestinal symptoms, precipitating events, cardiovascular dysfunction, and metabolic disturbances.Treatment of myxedema coma should be considered as quickly as possible, given the increased mortality of the disease (25%–60% despite treatment),18 and can be started even before confirmation of laboratory results demonstrating abnormal serum TSH and T4 concentrations. The management of myxedema coma should be in an intensive care unit (ICU) setting. The central tenets of treatment are thyroid hormone replacement, stress-dose corticosteroids if concomitant adrenal insufficiency is suspected, supportive care, and the treatment of any underlying and coexisting conditions (Table 2). Supportive care may include the administration of intravenous (IV) fluids, sodium replacement if hyponatremia is present, and the use of warming blankets (although aggressive rewarming should be avoided, given the risks of vasodilation).

Thyrotoxicosis refers to the state of thyroid hormone excess arising from either overproduction from the thyroid gland (termed hyperthyroidism) or extrathyroidal, including exogenous, sources. Of the etiologies attributable to

hyperthyroidism, the most common cause worldwide is Graves' disease, resulting from the autoimmune stimulation of the thyroid by serum thyroid-stimulating immunoglobulin, followed by toxic multinodular goiter and toxic adenoma. In the United States, the prevalence of hyperthyroidism from all causes is approximately 1.2%, of which 0.5% is overt and 0.7% is subclinical. Other causes of thyrotoxicosis include overproduction of TSH from a pituitary adenoma, thyroiditis, exogenous thyroid hormone ingestion, ectopic hyperthyroidism (such as from struma ovarii or metastatic thyroid cancer), or human chorionic gonadotropin-mediated hyperthyroidism (such as from hyperemesis gravidarum or a molar pregnancy).

The signs and symptoms of thyrotoxicosis are reflective of the excess concentrations of thyroid hormone present and can include anxiety, fatigue, diaphoresis, heat intolerance, tremors, palpitations, tachycardia, weight loss, hyperreflexia, and warm and moist skin. In women, menstrual abnormalities may be seen. In patients in whom the thyrotoxicosis is due to Graves' disease, specific clinical manifestations may also include thyroid eye disease (ie, exophthalmos, lid lag), a diffuse goiter with a bruit, localized dermopathy, thyroid acropachy (ie, digital clubbing and swelling), and the coexistence of other autoimmune diseases in the patient or the patient's family.

Thyroid storm is the clinical manifestation of elevated serum thyroid hormone concentrations, resulting in the extreme alteration of usual hyperthyroid symptoms. The diagnosis can occur in patients with or without preexisting hyperthyroidism. It is a rare diagnosis and usually triggered by precipitants such as trauma, myocardial infarction, surgery (including thyroid surgery for hyperthyroidism or other surgeries in general), or infection. In some cases, acute exposure to excess iodine (ie, administration of iodinated contrast radiographic scan) may result in iodine-induced hyperthyroidism to trigger thyroid storm.Patients with known severe hyperthyroidism who are noncompliant with prescribed antithyroid medications may also develop thyroid storm.

Prompt recognition of thyroid storm is essential to initiate treatment, which should be performed in an ICU setting. Clinical manifestations of thyroid storm can be quite varied and may include fever, cardiac arrhythmias, vomiting, and impaired mental status. Patients with thyroid storm have increased inpatient mortality rate, overall hospital and ICU length of stay, and ventilation requirements compared with those with compensated thyrotoxicosis. The mortality rate of thyroid storm ranges from 10% to 20%..Diagnosis of thyroid storm is made using a combination of biochemical laboratory tests confirming thyrotoxicosis in a patient displaying the severe, life-threatening symptoms of hyperthyroidism. Several diagnostic scoring systems have been proposed that can be used to assess the likelihood of thyroid storm in patients. The Burch scoring system is based on factors related to temperature,

central nervous system effects, gastrointestinal/hepatic dysfunction, cardiovascular dysfunction, heart failure, and any precipitant history. The criteria are similar and have also been proposed as another diagnostic scoring system in the assessment of thyroid storm. Treatment usually consists of multiple measures and medications aimed to target the various causes and effects of thyrotoxicosis, as summarized . Symptomatic improvement of tachycardia and clinical manifestations reflecting the increased adrenergic tone can be achieved with beta-blocker therapy. Methimazole or propylthiouracil should be initiated to decrease production of thyroid hormone. Saturated solution of potassium iodide can be used to inhibit thyroid hormone release from the thyroid gland. Glucocorticoids decrease the conversion of T4 to T3, which can also be accomplished by the use of propylthiouracil. Supportive measures include IV fluids, oxygen, cooling, and treatment of any precipitating causes. Finally, if necessary, when the above treatments cannot be used or are unsuccessful, plasmapheresis can be attempted to decrease thyroid hormone excess, as well as cytokines and putative antibodies, in the circulation.

Hypothyroidism and thyrotoxicosis are common endocrine disorders, each with a variety of etiologies, and most patients with thyroid dysfunction are easily managed. However, in certain patients, severe, life-threatening forms of these states, representing rare thyroid emergencies, can develop on exposure to precipitating triggers or among patients with preexisting thyroid dysfunction or noncompliance with medical treatment. Both myxedema coma, corresponding to extremely low serum thyroid hormone concentrations, and thyroid storm, corresponding to extremely elevated thyroid hormone concentrations, are associated with increased mortality and must be recognized promptly. Treatment of myxedema coma and thyroid storm is multifaceted and should be managed by the interdisciplinary team of an ICU setting.

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