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Hypothyrodisim: A new aspects of old disease

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Abstract
Abstract Objective: To present relevant and updated information on the status of hypothyroidism in the pediatric population (newborn infants to adolescents). Sources: Original and review articles and books containing relevant updated data. Summary of the findings: This review addressed data on the etiopathogeny of hypothyroidism and on the importance of screening for congenital hypothyroidism to assure early diagnosis and treatment of the newborn. We point out the difficulties experienced in the handling of subclinical hypothyroidism; we also address the importance of diagnosing autoimmune Hashimoto's thyroiditis, the high incidence of the disease among adolescents, mainly females, and the occurrence of a severe neurological condition, Hashimoto's encephalopathy. We indicate situations in which severe hypothyroidism may lead to puberty disorders (precocious or delayed puberty) and describe the importance of transcription factors in thyroid embryogenesis. Diagnostic and therapeutic criteria are also addressed. Conclusion: Thyroid hormones are necessary for normal growth and development since fetal life. Insufficient production or inadequate activity on the cellular or molecular level lead to hypothyroidism. These hormones are necessary for the development of the brain in the fetus and in the newborn infant. Neonatologists and pediatricians deal with child development issues in their practice, and many of these issues start during intrauterine life. Currently, with neonatal screening, neonatologists and pediatricians can prevent irreversible damage through early treatment. They should also be alert for dysfunctions such as subclinical hypothyroidism and Hashimoto's thyroiditis, which may provoke damage not only to growth, but also to the neurological and psychological development of these children and adolescents, thyroid hormones, thyropathies, thyroid failure, pediatric hypothyroidism, thyroid deficiency.

Keywords: Hypothyrodisim: a condition in which the thyroid gland doesn't produce enough thyroid hormone, etiopathogeny: the cause and subsequent development of an abnormal condition or of a disease, GH: growth hormone, thyropathies: a disorder off thyroid gland

1. Introduction

The thyroid gland contains numerous follicles, composed of epithelial follicle cells and colloid. Also, between follicles are clear parafollicular cells, which produce calcitonin.

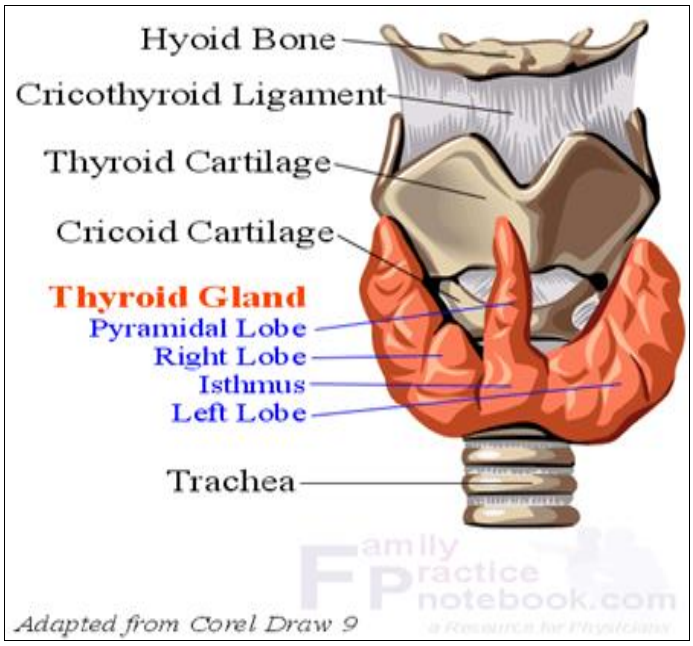

Fig 1

Thyroid hormone

- There are two biologically active thyroid hormones:
- Tetraiodothyronine (T4; usually called thyroxine)
- Triiodothyronine (T3)
- Derived from modification of tyrosine.

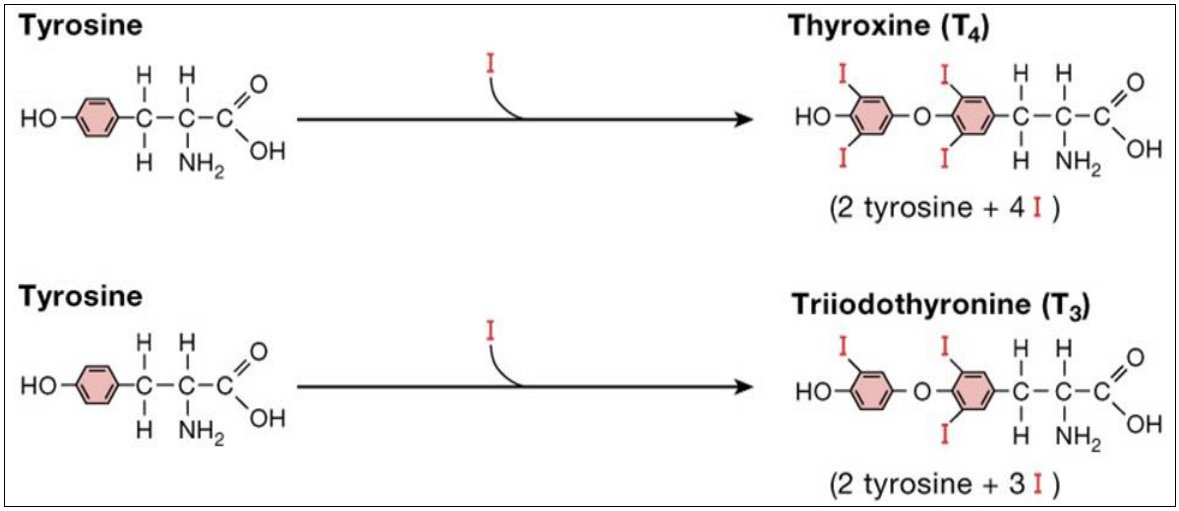

Fig 2

Why is Iodine Important in Thyroid Hormone Production?

- Thyroid hormones are unique biological molecules in that they incorporate iodine in their structure.
- Thus, adequate iodine intake (diet, water) is required for normal thyroid hormone production.
- Major sources of iodine:
- Iodized salt
- Iodated bread
- Dairy products
- Shellfish
- Minimum requirement: 75 micrograms/day
- US intake: 200 - 500 micrograms/day

Iodine Metabolism

- Dietary iodine is absorbed in the GI tract, then taken up by the thyroid gland (or removed from the body by the kidneys).
- The transport of iodide into follicular cells is dependent upon a Na^+/I^- co transport system.
- Iodide taken up by the thyroid gland is oxidized by peroxide in the lumen of the follicle

I^- peroxidase I^+

Oxidized iodine can then be used in production of thyroid hormones.

Transport of Thyroid Hormones

- Thyroid hormones are not very soluble in water (but are lipid-soluble).
- Thus, they are found in the circulation associated with binding proteins:
- Thyroid Hormone-Binding Globulin (~70% of hormone)
- Pre-albumin (transthyretin), (~15%)
- Albumin (~15%)
- Less than 1% of thyroid hormone is found free in the circulation.
- Only free and albumin-bound thyroid hormone is biologically available to tissues.

Conversion of T4 to T3

- 3 has much greater biological activity than T4.
- A large amount of T4 (25%) is converted to T3 in peripheral tissues.
- This conversion takes place mainly in the liver and kidneys. The T3 formed is then released to the blood stream.
- In addition to T3, an equal amount of "reverse T3" may also be formed. This has no biological activity.

One Major Advantage of this System

- The thyroid gland is capable of storing many weeks worth of thyroid hormone (coupled to thyroglobulin).
- If no iodine is available for this period, thyroid hormone secretion will be maintained.

Regulation of TSH Release from the Anterior Pituitary

- TSH release is influenced by hypothalamic TRH, and by thyroid hormones themselves.
- Thyroid hormones exert negative feedback on TSH release at the level of the anterior pituitary.
- Inhibition of TSH synthesis
- Decrease in pituitary receptors for TRH

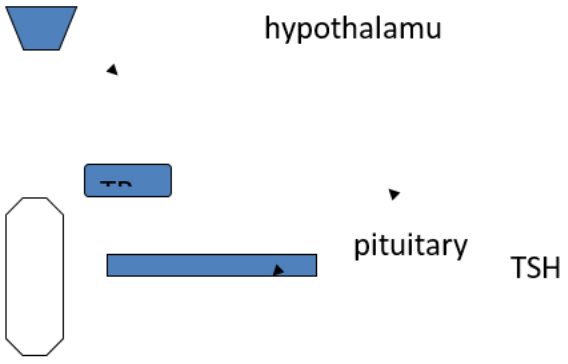

Fig 3

Other Factors Regulating Thyroid Hormone Levels

- Diet: a high carbohydrate diet increases T3 levels, resulting in increased metabolic rate (diet-induced thermo genesis).
- Low carbohydrate diets decrease T3 levels, resulting in decreased metabolic rate.
- Cold Stress: increases T3 levels in other animals, but

not in humans.
- Other stresses: increased or decreased?
- Any condition that increases body energy requirements (*e.g.*, pregnancy, prolonged cold) stimulates hypothalamus → TRH → TSH (Pit)

Actions of Thyroid Hormones
- Thyroid hormones are essential for normal growth of tissues, including the nervous system.
- Lack of thyroid hormone during development results in short stature and mental deficits (cretinism).
- Thyroid hormone stimulates basal metabolic rate.
- What are the specific actions of thyroid hormone on body systems?
- Required for GH and prolactin production and secretion
- Required for GH action
- Increases intestinal glucose reabsorption (glucose transporter)
- Increases mitochondrial oxidative phosphorylation (ATP production)
- Increases activity of adrenal medulla (sympathetic; glucose production)
- Induces enzyme synthesis
- Result: stimulation of growth of tissues and increased metabolic rate. Increased heat production (calorigenic effect)

Effects of thyroid hormones on:
Cardiovascular system
- Increase heart rate
- Increase force of cardiac contractions
- Increase stroke volume
- Increase Cardiac output
- Up-regulate catecholamine receptors

Respiratory system
- Increase resting respiratory rate
- Increase minute ventilation
- Increase ventilatory response to hypercapnia and hypoxia

Renal System
- Increase blood flow
- Increase glomerular filtration rate

Oxygen-Carrying Capacity
- Increase RBC mass
- Increase oxygen dissociation from hemoglobin

Effects Thyroid Hormones in Growth and Tissue Development
- Increase growth and maturation of bone
- Increase tooth development and eruption
- Increase growth and maturation of epidermis, hair follicles and nails
- Increase rate and force of skeletal muscle contraction
- Inhibits synthesis and increases degradation of mucopolysaccharides in subcutaneous tissue

Nervous System
- Critical for normal CNS neuronal development
- Enhances wakefulness and alertness
- Enhances memory and learning capacity
- Required for normal emotional tone
- Increase speed and amplitude of peripheral nerve reflexes

Reproductive System
- Required for normal follicular development and ovulation in the female
- Required for the normal maintenance of pregnancy
- Required for normal spermatogenesis in the male

Definition
Hypothyroidism is a metabolic imbalance that results from decreased production of the thyroid hormones tri iodothyronine (T_3) and thyroxine (T_4). The pituitary gland senses the low levels of thyroid hormone and increases the TSH level.

Types
Hypothyroidism (underactive thyroid) is a condition in which the thyroid gland does not make enough thyroid hormone. There are several types of hypothyroidism in children:
- Congenital hypothyroidism
- Acquired hypothyroidism
- Autoimmune hypothyroidism
- Iatrogenic hypothyroidism

Congenital hypothyroidism
Congenital hypothyroidism (CH) occurs when the thyroid gland does not develop or function normally prior to birth. It is a very common problem, affecting about 1 in every 2,500 to 3,000 babies. In the United States, all states test for CH as part of their routine newborn screening programs.
Many factors, including your family history, the physical exam, the degree of hypothyroidism in your baby at the time of diagnosis, and the course of treatment over the first two to three years of life, will help your physician determine if the cause is hereditary (runs in your family), and if life-long therapy is required. In general, there is little benefit from pursuing extensive laboratory or radiologic testing, but these options can be discussed with your physician.

Acquired hypothyroidism
Acquired hypothyroidism includes autoimmune hypothyroidism and iatrogenic hypothyroidism.

Autoimmune hypothyroidism
Acquired hypothyroidism is most frequently caused by an autoimmune disorder called chronic lymphocytic thyroiditis (CLT). In this disorder your child's immune system attacks the thyroid gland, leading to damage and decreased function. The disorder was originally described by a Japanese physician and thus is often referred to by his name: Hashimoto's thyroiditis. CLT is more common in girls than in boys, and in adolescents than pre-adolescents. Patients with other forms of autoimmune disease, most commonly insulin-dependent diabetes, are at increased risk of developing CLT. Overall, about 20 to 30 percent of diabetics will develop CLT and, because of this, annual screening for CLT is a routine part of diabetic care. The opposite is much less common, but can occur: to have CLT and then subsequently develop insulin-dependent diabetes.
Diagnosis of CLT may include:

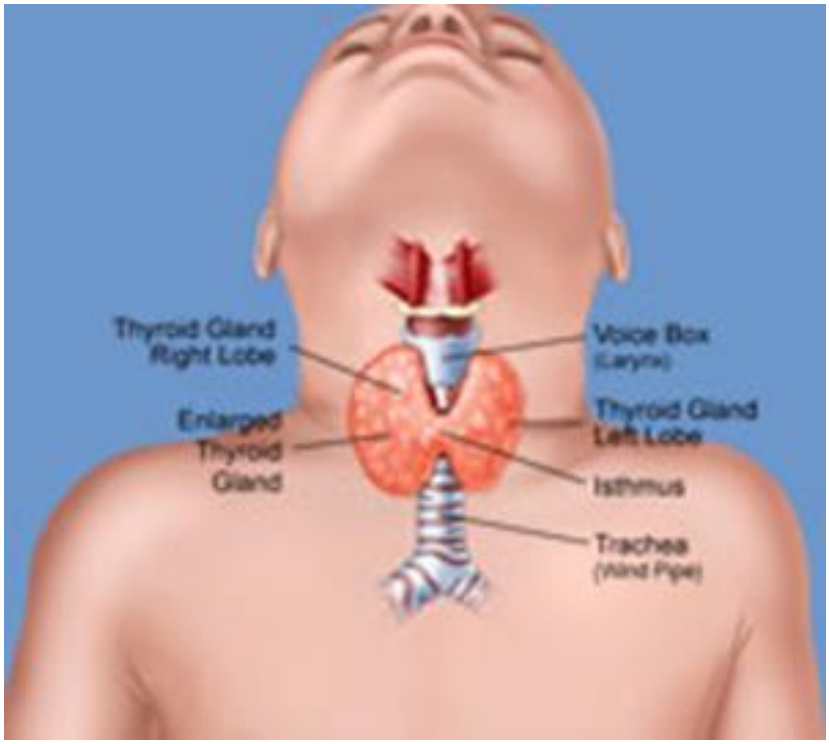

Fig 4: Hypothyroidism - Enlarged Thyroid

Enlarged thyroid gland due to an autoimmune disorder called chronic lymphocytic thyroiditis (CLT) or Hashimotoâs thyroiditis.

- Physical exam. Children with CLT will often have an enlarged thyroid (called a goiter), caused by the immune cells invading and damaging the thyroid tissue, and resulting in decreased levels of thyroid hormones. Thyroid hormones below the normal levels lead to an increase production of thyroid-stimulating hormone (TSH).
- Laboratory studies to test for antibodies that develop against proteins found in normal thyroid cells: anti-thyroperoxidase (anti-TPO) and anti-thyroglobulin (TgAb).
- Ultrasound. In children with CLT, the thyroid tissue will have a very irregular appearance and there may be areas that look like a thyroid nodule. In general, thyroid nodules are more common in patients with CLT; however, it's not clear whether there is an increased risk of thyroid cancer in patients with CLT.

Iatrogenic hypothyroidism

Iatrogenic hypothyroidism is a form of acquired hypothyroidism that occurs in people who have had their thyroid gland medically ablated (destroyed) or surgically removed. By removing the thyroid gland, the body no longer produces thyroid hormone, leading to iatrogenic hypothyroidism.

Etiology

- Autoimmune disease. People who develop a particular inflammatory disorder known as Hashimoto's thyroiditis suffer from the most common cause of hypothyroidism. Autoimmune disorders occur when your immune system produces antibodies that attack your own tissues. Sometimes this process involves your thyroid gland. Scientists aren't sure why the body produces antibodies against itself. Some think a virus or bacterium might trigger the response, while others believe a genetic flaw may be involved. Most likely, autoimmune diseases result from more than one factor. But however it happens, these antibodies affect the thyroid's ability to produce hormones.
- Treatment for hyperthyroidism. People who produce too much thyroid hormone (hyperthyroidism) are often treated with radioactive iodine or anti-thyroid medications to reduce and normalize their thyroid function. However, in some cases, treatment of hyperthyroidism can result in permanent hypothyroidism.
- Thyroid surgery. Removing all or a large portion of your thyroid gland can diminish or halt hormone production. In that case, you'll need to take thyroid hormone for life.
- Radiation therapy. Radiation used to treat cancers of the head and neck can affect your thyroid gland and may lead to hypothyroidism.
- Medications. A number of medications can contribute to hypothyroidism. One such medication is lithium, which is used to treat certain psychiatric disorders. If you're taking medication, ask your doctor about its effect on your thyroid gland.

Less often, hypothyroidism may result from one of the following:

- Congenital disease. Some babies are born with a defective thyroid gland or no thyroid gland. In most cases, the thyroid gland didn't develop normally for unknown reasons, but some children have an inherited form of the disorder. Often, infants with congenital hypothyroidism appear normal at birth. That's one reason why most states now require newborn thyroid screening.
- Pituitary disorder. A relatively rare cause of hypothyroidism is the failure of the pituitary gland to produce enough thyroid-stimulating hormone (TSH) —

usually because of a benign tumor of the pituitary gland.

- Pregnancy. Some women develop hypothyroidism during or after pregnancy (postpartum hypothyroidism), often because they produce antibodies to their own thyroid gland. Left untreated, hypothyroidism increases the risk of miscarriage, premature delivery and preeclampsia — a condition that causes a significant rise in a woman's blood pressure during the last three months of pregnancy. It can also seriously affect the developing fetus.
- Iodine deficiency. The trace mineral iodine — found primarily in seafood, seaweed, plants grown in iodine-rich soil and iodized salt — is essential for the production of thyroid hormones. In some parts of the world, iodine deficiency is common, but the addition of iodine to table salt has virtually eliminated this problem in the United States. Conversely, taking in too much iodine can cause hypothyroidism.

Hypothyroidism and pregnancy

- During the first few months of pregnancy, the fetus relies on the mother for thyroid hormone. Thyroid hormone plays an essential part in normal brain development. Maternal hypothyroidism can cause irreversible harm to the fetus. Early studies found that children born to mothers with hypothyroidism during pregnancy had lower IQ and impaired psychomotor (mental and motor) development. If properly controlled, often by increasing the amount of thyroid hormone, women with hypothyroidism can have healthy, unaffected babies.
- Current recommendations include asking all women at the initial prenatal visit if they have any history of thyroid dysfunction or thyroid hormone medication. Laboratory screening of thyroid functions and/or thyroid antibodies should be considered for women at high risk of hypothyroidism. Detection and treatment of Maternal hypothyroidism early in pregnancy may prevent the harmful effects of maternal hypothyroidism on the fetus. For women on thyroid hormone prior to conception, thyroid function testing should be Performed regularly throughout pregnancy as it is very likely that the thyroid hormone dose will need to be increased. Women are encouraged to ask their primary care providers for further information and clarification on this important topic.

Pathophysiology

Thyroxine, the primary secretory product of the thyroid, is relatively inactive. It is converted to the active hormone, triiodothyronine, by the enzyme thyroxine 5'-deiodinase. Triiodothyronine acts on a family of nuclear receptors that bind to regulatory regions of genes and modify their expression. For example, triiodothyronine increases transcription of the genes for calcium ATPase in the sarcoplasmic reticulum of skeletal and cardiac muscle. Calcium ATPase mediates clearance of calcium from the cytoplasm after muscle contraction, so hypothyroidism results in prolonged muscle relaxation.As another example, thyroid hormones affect expression of myelin genes, and hypothyroidism causes a variety of structural and functional changes in the hippocampus, especially during development. Most of the genes targeted by thyroid hormones in the brain remain to be identified.

The genes that code triiodothyronine receptors are differentially expressed in various tissues, and the effects of triiodothyronine depend on the specific isoform of the receptor. The receptors are expressed in a developmentally specific pattern, especially in the brain. Many of the specific genes regulated by triiodothyronine in various tissues have not been identified, but the general metabolic effects of thyroid hormones are to stimulate oxygen consumption in almost all metabolically active tissues, increase absorption of carbohydrates from the intestine, and modulate lipid metabolism. Thyroid hormones mobilize mucopolysaccharides and prevent their deposition in skin and connective tissues. Thyroid hormones also interact with catecholamines. The cognitive and affective manifestations of hypothyroidism are not fully understood. A variety of factors may be involved, including reduced responsiveness of the reticular activating system to catecholamines, impaired fluid and electrolyte regulation, and altered blood flow.

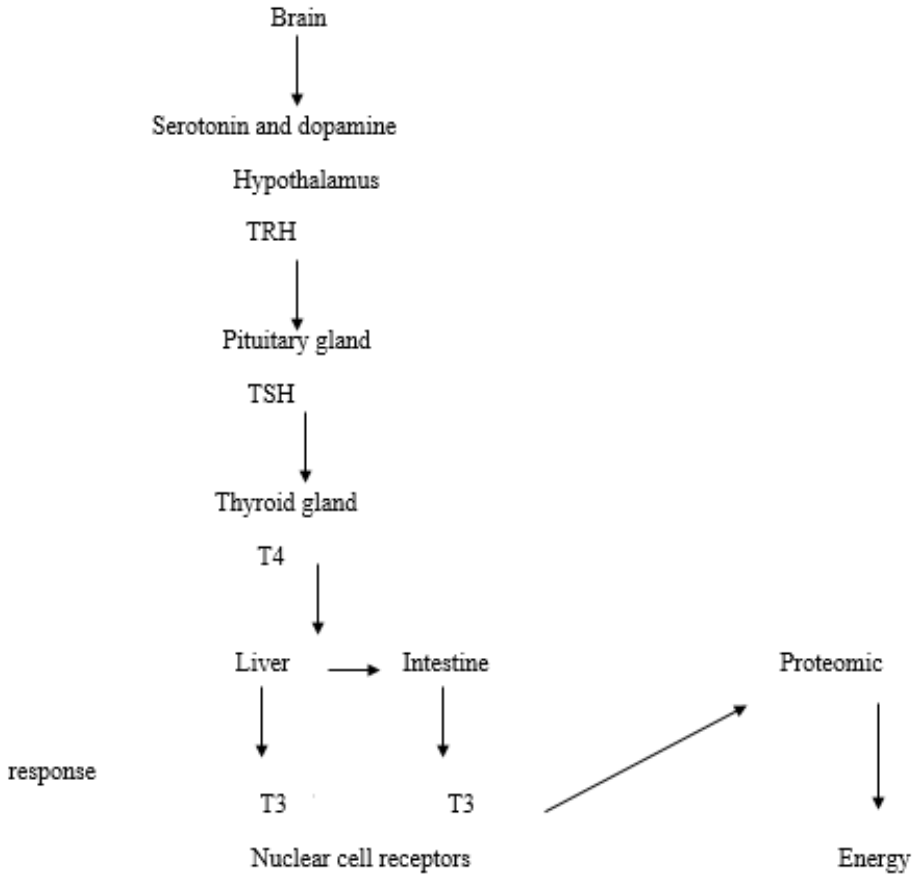

Fig 5

Symptoms of hypothyroidism

Hypothyroidism is a common condition which can go undetected if symptoms are mild. Symptoms of hypothyroidism are usually very subtle and gradual, and may include:

- Fatigue and/or exercise intolerance
- Slower reaction time (an important issue for drivers)
- Weight gain
- Constipation
- Sparse, coarse and dry hair
- Coarse, dry and thickened skin
- Slow pulse
- Cold intolerance
- Muscle cramps
- Sides of eyebrows thin or fall out
- Dull facial expression
- Hoarse voice
- Slow speech
- Droopy eyelids
- Puffy and swollen face
- Increased menstrual flow and cramping in girls and women

The symptoms of hypothyroidism may resemble those of other conditions or medical problems. Always consult your physician for a diagnosis.

Diagnostic Studies

- *Radioactive iodine* **(***RAI***)** *uptake test:* High in Graves' disease and toxic nodular goiter; low in thyroiditis.
- *Serum T_4 and T_3:* Increased in hyperthyroidism. Normal T_4 with elevated T_3 indicates thyrotoxicosis.
- *Thyroid-stimulating hormone* **(***TSH***):** Suppressed (except when etiology is a TSH-secreting pituitary tumor or pituitary resistant to thyroid hormone). Does not respond to thyrotropin-releasing hormone (TRH).
- *Thymoglobulin:* Increased.
- *TRH stimulation:* Hyperthyroidism is indicated if TSH fails to rise after administration of TRH.
- *Thyroid T_3 uptake:* Normal to high.
- *Protein-bound iodine:* Increased.
- *Serum glucose:* Elevated (related to adrenal involvement).
- *Plasma cortisol:* Low levels (less adrenal reserve).
- *Alkaline phosphatase and serum calcium:* Increased.
- *Liver function tests:* Abnormal.
- *Electrolytes:* Hyponatremia may reflect adrenal response or dilutional effect in fluid replacement therapy. Hypokalemia occurs because of GI losses and diuresis.
- *Serum catecholamines:* Decreased.
- *Urine creatinine:* Increased.
- *ECG:* Atrial fibrillations; shorter systole time; cardiomegaly, heart enlarged with fibrosis and necrosis (late signs or in elderly with masked hyperthyroidism).
- *Needle or open biopsy:* May be done to determine cause of hyperthyroidism, differentiate cysts or tumors, diagnose enlargement of thyroid gland.
- *Thyroid scan:* Differentiates between Graves' disease and Plummer's disease, both of which result in hyperthyroidism.

Medical Management

In congenital hypothyroidism, treatment should be initiated

as soon as the diagnosis is suggested, immediately after obtaining blood for confirmatory tests. Delaying treatment after 6 weeks of life is associated with a substantial risk of delayed cognitive development. Newborns with elevated TSH should be treated empirically with thyroid hormone replacement until they are aged 2 years to eliminate any possibility of permanent cognitive deficits caused by hypothyroidism.

- Once treatment is initiated for congenital hypothyroidism, serum total T4 and TSH concentrations should be assessed monthly until the total or free T4 levels normalize, and then every 3 months until the patient is aged 3 years. Thereafter, total T4 and TSH should be measured every 6 months.
- In patients with thyroid agenesis, serum TSH levels may remain slightly elevated (15-25 mcIU/mL) despite adequate thyroid hormone replacement, as indicated by serum total or serum free T4 levels and clinical assessment.
- This phenomenon has been termed a reset thyrostat and reflects initial transient unresponsiveness of the hypothalamic-pituitary axis (hypertrophied thyrotrophs) to thyroid hormone replacement. The higher the initial serum TSH, the more likely one is to observe persistent mild elevation despite adequate replacement. If appropriate thyroid hormone replacement therapy is given, the thyrostat typically resets to a normal value within a few months.
- Initial evaluation and follow-up can be conducted on an outpatient basis.
- Therapeutic goals are normalization of thyroid function test results and elimination of all signs and symptoms of hypothyroidism.
- Therapy should correct growth, pseudo precocious puberty, and galactorrhea. Goiter may be reduced; however, replacement therapy often does not result in complete normalization of size.
- When indicated by an elevated serum TSH, dosage adjustments of 0.0125 mg levothyroxine are usually sufficient. Because the half-life of T4 in the serum is about 6 days, approximately 3.5 weeks are required for serum T4 values to reach a new steady state. Depending on the degree of hypothyroidism and the time spent in the hypothyroid state, suppression of elevated TSH levels may take longer; therefore, repeat measurements of total T4 and TSH should be obtained no sooner than 1 month after any dosage adjustment or change in brand of thyroid hormone.
- Levothyroxine tablets are easily crushed and can be given in a spoon with a small amount of water, formula, or cereal. Suspensions are not commercially available and are not recommended because maintaining a consistent concentration of levothyroxine in solution is difficult.
- Levothyroxine is the preferred form of thyroid hormone replacement in all patients with hypothyroidism. Rarely, patients with congenital hypothyroidism display a "reset thyrostat" (ie, the serum TSH is not suppressed to reference range even with supraphysiologic replacement of levothyroxine). The primary therapeutic goal in patients with congenital hypothyroidism is to maintain the free serum T4 level within the high end of the reference range without resulting in symptoms of hyperthyroidism.
- Thyroid hormones only should be used as replacement therapy in children with hypothyroidism. In active form, thyroid hormone influences growth and maturation of tissues, metabolism, and development. It does not enhance final adult height in euthyroid children.
- Approximately 20% of children with CLT recover to the euthyroid state and do not require lifelong thyroid hormone replacement. After treatment beyond the completion of puberty, a 6-month trial off thyroid hormone replacement therapy should be considered, with monitoring of serum TSH and total T4 levels every 3 months. If serum TSH levels rise above the reference range, levothyroxine treatment should be resumed and continued for life. Patients with CLT should undergo at least yearly monitoring of thyroid function with serum total T4 and TSH assessment to assure adequate treatment and maintenance of euthyroidism.
- In the case of concomitant hypopituitarism with corticotropin deficiency or any other causes of suspected adrenal insufficiency, glucocorticoid replacement should always precede thyroid hormone replacement. This reduces the risk of adrenal crisis resulting from increased demands from enhanced metabolism from thyroid hormone replacement.
- Once euthyroid, infants with congenital hypothyroidism should be observed every 3 months until they are aged 3 years. Thereafter, these children can be evaluated every 6 months.

Surgical Management

Rarely, a massive goiter may require surgical resection for cosmetic indications. Generally, surgical therapy has no role in the treatment of hypothyroidism.

Nursing management

Nursing diagnosis

1. **Risk for decreased cardiac output related to impaired cardiac contractility.**
   - Monitor BP lying, sitting, and standing, if able. Note widened pulse pressure.
   - Monitor central venous pressure (CVP), if available.
   - Assess pulse/heart rate while patient is sleeping.
   - Administer IV fluids as indicated.
   - Provide supplemental O_2 as indicated.
   - Administer medications as indicated:[beta]-blockers, e.g., propranolol (Inderal), atenolol (Tenormin), nadolol (Corgard), pindolol (Visken);Thyroid hormone antagonists, e.g., propylthiouracil(PTU), methimazole (Tapazole);
2. **Fatigue related to hyper metabolic state with increased energy requirements as evidenced by Impaired ability to concentrate/ decreased performance**

- Monitor vital signs, noting pulse rate at rest and when active.
- Note development of tachypnea, dyspnea, pallor, and cyanosis.
- Provide for quiet environment; cool room, decreased sensory stimuli, soothing colors, quiet music.
- Encourage patient to restrict activity and rest in bed as much as possible.

- Provide comfort measures, e.g., judicious touch/massage, cool showers.
- Provide for diversional activities that are calming, e.g., reading, radio, television.
- Avoid topics that irritate or upset patient. Discuss ways to respond to these feelings.

3. Disturbed thought process related to increased CNS stimulation / Altered sleep pattern

- Assess thinking processes, e.g., memory, attention span, orientation to person/place/time.
- Provide quiet environment; decreased stimuli, cool room, dim lights. Limit procedures/personnel.
- Reorient to person/place/time as indicated.
- Provide clock, calendar, room with outside window; alter level of lighting to simulate day/night.
- Provide safety measures, e.g., padded side rails, close supervision, or soft restraints as last resort as necessary.
- Administer medication as indicated, e.g., sedatives/anti-anxiety agents/antipsychotic drugs.

4. Imbalanced nutritional status less than the body requirement

- Monitor daily food intake. Weigh daily and report losses.
- Encourage patient to eat and increase number of meals and snacks, using high-calorie foods that are easily digested.
- Avoid foods that increase peristalsis (e.g., tea, coffee, fibrous and highly seasoned foods) and fluids that cause diarrhea (e.g., apple/prune juice).
- Consult with dietitian to provide diet high in calories, protein, carbohydrates, and vitamins.
- Administer medications as indicated: Glucose, vitamin B complex; Insulin (small doses).

5. Knowledge, deficient [Learning Need] regarding condition, prognosis, treatment, self-care, and discharge needs as evidenced by Questions, request for information, statement of misconception

- Review disease process and future expectations.
- Provide information appropriate to individual situation.
- Identify stressors and discuss precipitators to thyroid crises, e.g., personal/social and job concerns, infection, pregnancy.
- Provide information about signs/symptoms of hypothyroidism and the need for continuing follow-up care.
- Discuss drug therapy, including need for adhering to regimen, and expected therapeutic and side effects.
- Identify signs/symptoms requiring medical evaluation, e.g., fever, sore throat, and skin eruptions.

Research Abstract

Awat Feizi et. al. conducted a prospective cohort study to investigate the growth status of CH, generate specialized growth charts of CH infants, and compare them with their counterparts of regional normal infants. In this study 760 (345 girls and 415 boys) neonates born in 2002–2009 diagnosed by neonatal CH screening program in Isfahan were followed up from the time of diagnosis. 552 healthy children were recruited as a control group. The empirical 3rd, 15th, 50th, 85th, and 97th percentiles for height, weight, and head circumference of both sexes were determined and compared with their counterpart values of the control group. The relative frequency of patients with impaired growth for each studied variable was determined. Also, specialized growth charts of CH patients were generated. The percentiles of weight, height, and head circumference of studied patients are significantly different from regional healthy children ($P < 0.001$). The relative frequency of impaired head circumference was decreased to less than 3% at the 3rd year of age and for height it reached gradually 3% and 9% at the 5th year of age for boys and girls, respectively ($P < 0.05$); however for weight still it was statistically more than 3% in both sexes. CH patients had impaired growth development which was improved during follow up, but the catch-up time was earlier for head circumference and later for weight.

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