Review Article

An Overview on Hyperthyroidism, Evaluation and Management Approach in Primary Health Care Centre

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Abstract

Hyperthyroidism is specified by the mass manufacture of thyroid hormone and/or release from the thyroid gland. Thyrotoxicosis is a sickness in which the impacts of an inappropriate enhancement in thyroid hormone on tissues lead to difficulties and systemic symptoms. High thyroid hormone levels cause a hyper metabolic condition that induces multiple systemic manifestations. Managing hyperthyroidism cases is considered a difficult challenge because of the variety of the underlying causes and the variety of the management options. An assessment of publicized literature discussing hyperthyroidism and to equip an adequate analysis of hyperthyroidism, its diagnosis, and management. The following keys were used in the mesh ((“hyperthyroidism"[Mesh]) AND (“management” [Mesh]) OR (“evaluation"[Mesh])). And PubMed database was used for articles selection. Hyperthyroidism is a condition that is commonly overlooked. Maintaining a high suspicion index is important for the primary healthcare physician. Treatment suggestions are based on characteristics diversity, including underlying reason, patient age, disease severity, and comorbidity. The major treatment options are ant thyroid medications (thionamides), surgery, and radioactive iodine. All methods had equal quality of life parameters, but generally, radioiodine was the most cost-effective.

Keywords: Hyperthyroidism, Management, Diagnosis, Evaluation

Introduction

Hyperthyroidism is specified by the mass manufacture of thyroid hormone and/or release from the thyroid gland [1]. Hyperthyroidism is specified by the mass manufacture of thyroid hormone and/or release from the thyroid gland. Thyrotoxicosis is a sickness in which the impacts of an inappropriate enhancement in thyroid hormone on tissues lead to difficulties and systemic symptoms [2].

1.2% of the residents have hyperthyroidism in the U.S, which accounts for 0.5% of overt hyperthyroidism and 0.7% of subclinical hyperthyroidism [2, 3]. In the United Kingdom, thyroid disease is not uncommon, too. The annual incidence of hyperthyroidism is 770 cases per 100,000 for women and 140 per 100,000 cases for men [4, 5].

Thyroid hormones have a great impact on cardiac homeostasis and high levels of thyroid hormone induce a hyper metabolic state that causes several systemic manifestations [6]. Thus, in this article, we intend to analyze the published literature on hyperthyroidism.

Materials and Methods

The following keys were used in the mesh ((“hyperthyroidism"[Mesh]) AND (“management” [Mesh]) OR (“evaluation"[Mesh])). And PubMed database was used for articles selection. In terms of inclusion criteria, articles were chosen based on the encompassing of one of the following subjects: evaluation, management, and pertussis.

Criteria of exclusion were all other articles that did not have one of these subjects as their main finishing point.

Results and Discussion

Two hormones produced by the thyroid gland are Triiodothyronine (T3) and thyroxine (T4). Thyroxine is the most common kind of thyroid hormone, and it functions

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primarily as a prohormone [7]. Hypothalamic thyrotropin-releasing hormone (TRH), which raises the synthesis and secretion of thyroid-stimulating hormone (TSH), controls the concentration of thyroid hormone. It also restrains the production and secretion of thyroid hormone by the thyroid gland. Most T4 is converted to biologically active T3 by iodide deduction by deiodinases.

Yet, there are 3 types of deiodinase: First is responsible for most of the circulating T3; Type 1 activates thyroid hormone by transforming T4 to active T3, and it deactivates thyroid hormone by transforming T4 to inactive inverse T3 (rT3) or T2 [7-9]. Cardiomyocytes lack significant intracellular deiodinase activity. As a result, the heart relies heavily on the function of T3, a hormone that normally enters myocytes. Most T4 and T3 (over 95%) bind to thyroxine-binding globulin and another family of hormone-binding proteins in the bloodstream. Residues of T3 that do not attach to the cell nucleus are carried by a variety of membrane-transfer proteins, where they regulate the expression of specific genes [9, 10].

The most widespread endogenous reasons for hyperthyroidism are painless thyroiditis, Graves 'disease, toxic adenoma, and toxic multinodular goiter [11]. The most regular reason for hyperthyroidism in the United States is Graves 'disease. It is an autoimmune disease in which thyroid-stimulating antibodies cause thyroid hormone production by activating TSH receptors. Female and personal or family history of autoimmune disease are both danger factors for Graves' disease [12, 13]. Toxic multinodular goiter is the second most common reason for hyperthyroidism in the States and the most frequent among the elderly living in iodine-deficient areas [12]. Nodules form over time with the repeated proliferation of clonogenic cells, leading to a somatic activator mutation in TSH receptors [14]. Plummer disease is a single nodule also known as a poisonous adenoma. Unlike these three diseases, painless or temporary (silent) thyroiditis leads to the death of thyroid follicles and the release of pre-made thyroid hormones into the bloodstream due to an autoimmune method [15].

It has the same clinical presentation as other underlying causes. By the use of scintigraphy, its incidence among patients with thyrotoxicosis was 0.5% in a Danish paper. Childbirth (postpartum thyroiditis) or the use of medicines including lithium, interferon-alpha, interleukin-2, and amiodarone can lead to painless thyroiditis [16]. Hyperthyroidism of gestational happens in the first three months of pregnancy because of stimulation of the thyroid gland by placental beta-human chorionic gonadotropin (b-hCG), which has structural similarities with TSH. Hyperthyroidism induced by b-hCG can also be activated by reproductive vomiting and, in rare circumstances, trophoblastic tumors of gestational. TSH-secreting pituitary adenomas, metastatic thyroid follicular cancer, and struma ovarii are also rare reasons for hyperthyroidism [16, 17].

Amiodarone-caused hyperthyroidism can be seen in 12% of patients treated, especially in iodine-deficient regions, and is induced by two methods. Type I hyperthyroidism is caused by the presence of 37% iodine in amiodarone. In the U.S, amiodarone is the most prevalent reason for iodine excess. Type II thyroiditis affects people who have healthy thyroid glands and it can also be caused by medications like interferon and interleukin-2. Thyroxine-induced hyperthyroidism is caused by the intentional or accidental intake of too much thyroid hormone which causes factitial hyperthyroidism. Thyroid medications might be used by certain individuals to help them lose weight [17-19].

Thyroid hormones can influence a wide range of systemic functions, so the clinical manifestations of hyperthyroidism can vary. The binding of T3 to alpha and beta receivers increases thermogenesis and the rate of basal metabolism in the cell. This can lead to loss of weight, fatigue, and heat sensitivity as an effect of the rules. In Graves' disease, skin abnormalities such as warm, moist skin, hair loss, and pretibial myxedema might develop. Accelerated bone resorption, osteoporosis, frailty, and the risen threat of fractures are common symptoms of musculoskeletal disorders. Lymphoma, gynecomastia in males, and oligomenorrhea in women are all possible side effects. Dysphagia, polyphagia, and diarrhea are the expected gastrointestinal symptoms. In individuals with Graves' disease, ophthalmologic signs include lid retraction and infiltrative Graves' ophthalmopathy [1, 2].

All risk factors for Graves' ophthalmopathy are as follows: smoking, agedness, longer duration of symptoms, and female [20]. Hyperthyroidism is related to effective difficulties of cardiovascular that should be adequately identified and treated. Blood pressure and tachycardia are the most common symptoms of cardiovascular hyperthyroidism. Hypertension affects around 10% of the population due to secondary factors such as endocrine etiology, and hypertension may be the initial symptom of a main endocrine disease [1]. Hypertension with hyperthyroidism has a complex etiology. T3's tissue effects are critical for homeostasis under normal circumstances. Excess T3 causes many problems because it enhances cardiac contractility and dilates arterioles. This results in lowering systemic vascular resistance and increasing arterial filling [21].

As a result, renin is released, and the angiotensin-aldosterone axis is activated. Thyroid hormone also affects some ion channels, including calcium/calmodulin-dependent kinase IV, which regulates vascular tone and blood pressure via regulating endothelial nitric oxide synthase activity. As a result, renin production and the angiotensin-aldosterone axis are stimulated. Thyroid hormone also affects some ion channels, notably calcium/calmodulin-dependent kinase IV, which regulates vascular tone and blood pressure via regulating endothelial nitric oxide synthase activity. As a result, thyrotoxicosis is linked to arterial stiffness.

Hypertension, tachycardia, and rising cardiac output will be clinical manifestations similar to advanced adrenergic activity, although catecholamine may be down or normal in hyperthyroidism. Congestive heart failure and atrial fibrillation are other potential difficulties. Atrial fibrillation is associated with higher T4 levels, toxic nodules, male, and agedness. According to preliminary research, heart failure occurs in 6 to 16% of people with hyperthyroidism, but in the presence of cardiovascular illness, a much higher rate is predicted. Nevertheless, after adequate management for hyperthyroidism and restoring euthyroid status, these cardiovascular manifestations could be reversible [1, 21].

Diagnosis

TSH grades are needed only for the first test, in a patient suspected of hyperthyroidism but there is no proof of hypophysis disease. More tests are needed if TSH statuses are abnormal. Hyperthyroidism can be diagnosed by an undetectable TSH level. Anti-thyroid antibodies are elevated in Graves' disease and lymphocytic thyroiditis, although it is usually not necessary to make a diagnosis. Thyroid-stimulating antibody ranks can be used to track the effectiveness of ant thyroid therapy in patients with Graves' disease. Absorption of radionuclide and scan easily distinguish high absorption of Graves' disease from low uptake of thyroiditis. It also provides other useful anatomic information. Nonspecific laboratory abnormalities that can happen with hyperthyroidism are hypercalcemia, granulocytosis, anemia, lymphocytosis, elevated transaminases, and elevated alkaline phosphatase.

A diagnostic strategy for patients presenting with symptoms of hyperthyroidism is briefly explained in **Figure 1** [19].

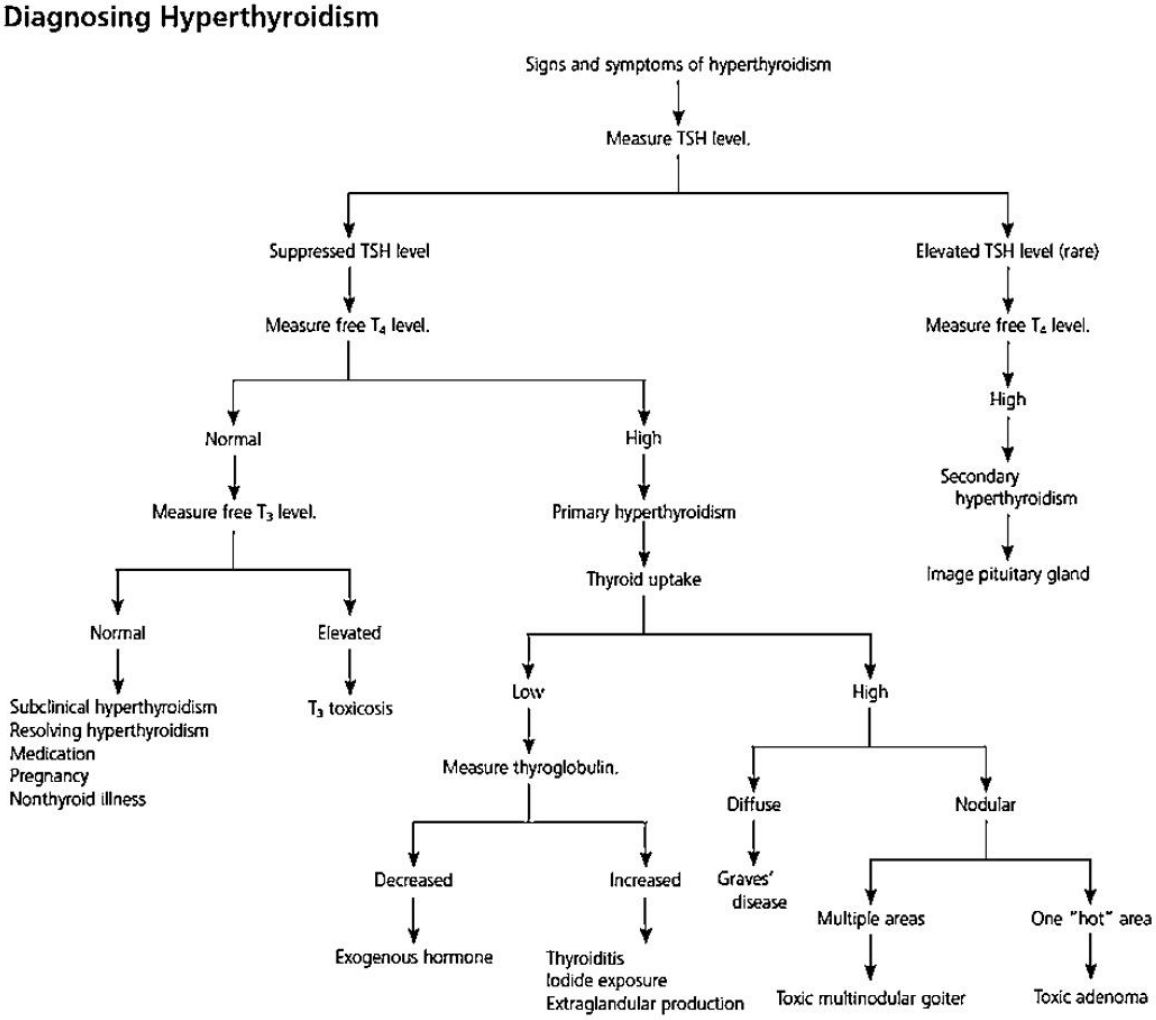

Figure 1. Diagnostic algorithm adopted from Fitzgerald [19]

Extra estrogen from pregnancy or estrogen therapy results in an enhancement in thyroxine-binding globulin levels, which appears to increase total T4 and T3 levels while TSH and T4 ranks remain free. These results do not show hyperthyroidism and do not demand treatment. Exogenous glucocorticoids or dopamine may induce a slight reduction in TSH levels, a

condition that often happens in the section of intensive care. Total levels of T4, total T3, and free T4 remain normal. TSH levels return to normal after discontinuation of these drugs [22].

The reason for hyperthyroidism can be determined with a radioactive iodine uptake test and a thyroid scan. The percentage of an iodine 123 (I-123) tracer dosage taken up by the thyroid gland varies between 15% and 25% after 24 hours. Individuals with thyroiditis have a very low uptake (0 to 2%), but patients with toxic multinodular goiter, graves' illness, or toxic adenoma have very high absorption. The distribution of the radio detector in the thyroid gland can be noticed on a thyroid scan. Graves' illness is characterized by a constant distribution of I-123, but the assemblage of I-123 in one or more areas indicates a toxic multi-nodular goiter or toxic adenoma.

Ultrasonography is sometimes used instead of absorbing and scanning radioactive iodine because it is cheaper and securer. In pregnancy period, lactation, and amiodarone-induced thyrotoxicosis, imaging is predominant [23].

Management

Thyrotoxicosis is a condition that is commonly overlooked. Stress can readily be attributed to initial symptoms like lethargy, palpitations, and anxiety. Maintaining a high suspicion index is important for the primary healthcare physician and this is obtained by carefully looking for goiter and ocular symptoms, checking for tremors, and feeling the hands. They are generally cold and dry in primary anxiety, but warm and moist in thyrotoxicosis. Hyperthyroidism is a symptom, not a diagnosis that is determined by the underlying reason [24]. In older patients, thyrotoxicosis is notably easy to overlook. In this age group, lethargy or weariness, as well as tremor, are typical presenting symptoms. Because most instances are not caused by Graves' illness, typical ocular symptoms will usually be absent. Symptoms may be attributed to other diseases or drug side effects due to comorbidities, and medicines, particularly b-blockers, may disguise some of the symptoms. Taking into consideration probable causal variables like amiodarone medication and prior thyroid surgery might provide vital information. The primary healthcare physician should have sufficient knowledge and expertise in approaching and managing patients with overt hyperthyroidism or they should visit a professional for further management. Ant thyroid drugs e.g. surgery, radioactive iodine, carbimazole, and propylthiouracil, may be used to treat people with the disease. Treatment recommendations are made based on a variety of characteristics, including the underlying cause, the patient's age, the severity of the disease, and the existence of comorbidity. If not contraindicated, a b-blocker, such as propranolol 40 mg three times a day, maybe administered to manage symptoms while waiting for a referral.

If the patient has to wait a long time to visit a professionals, the general practitioner may start taking ant thyroid drugs in discussion with an endocrinologist. The most widely used ant thyroid drug in the UK is Carbimazole, with a standard starting dose of 20 to 40 mg per day for adults. After 6 weeks to 2 months, depending on the response, the dose is reduced to an upkeep dose of 5-15 mg per day. T4 levels should typically be used to guide treatment when monitoring thyroid function; TSH concentrations can take many months to recover to normal. Bone marrow suppression is the most earnest side effect of carbimazole treatment. If there are signs or symptoms of infection, particularly sore throat, a complete blood count (CBC) should be done immediately. Carbimazole should be discontinued promptly if neutropenia is confirmed. It is recommended that a CBC be evaluated at the same time as each subsequent thyroid function test. In uncomplicated Graves' disease, therapy with anti-thyroid medications alone is a feasible choice because 40–60% of patients will achieve permanent remission after 12–18 months of treatment. Remission is uncommon in the severe course of the disease and individuals with a big goiter, and it is extremely unlikely in toxic nodular goiter, including toxic adenoma; in such instances, definitive treatment, either radioiodine or surgery, should be considered as soon as possible. The surgery is extremely necessary for elderly patients with comorbidities [24, 25].

Beta-blocking agents are useful for controlling symptoms, mainly in the elderly and those with cardiovascular illness. Propranolol is usually authorized, but longer-acting agents, such as atenolol, may improve compliance. Oral calcium channel blockers such as diltiazem and verapamil may also be used to decrease the pulse rate of patients that cannot endure beta-blockers.

The major treatment choices are operation, antithyroid medicines (thionamides), and radioactive iodine. Treatment should assess the size of the goiter, the patient's preference, the timing of future pregnancies, and the presence of significant comorbidities. All methods had equal quality of life parameters, but generally, radioiodine was the most cost-effective [26, 27].

The primary healthcare physician should have appropriate knowledge about the various management options (even surgical plans) and their follow-up challenges in detail as such patients will be frequently encountered in the primary healthcare facilities.

Iodine of Radioactive

In the U.S, most individuals with Graves' disease and toxic nodular goiter are treated with radioactive iodine. It is the treatment of choice because it is low-cost, extremely effective, simple to use, and safe. Because of the possible danger of thyroid cancer, leukemia, or genetic harm in future kids, there is a lack of enthusiasm in using radioactive iodine in women of childbearing age. However, these matters have not been confirmed by the long-term follow-up of the patient. Though the hyperthyroidism treatment in children is still

debated, radioactive iodine is becoming more favored in this age group [28, 29].

Graves' illness is a chronic disease that affects people of any age. With the use of radioactive iodine, Graves's ophthalmopathy can arise or worsen in up to 15% of people. In two-thirds of patients, prednisone (40 to 80 mg daily, reduced for at least three months) can stop or treat serious eye damage. Low-dose radioactive iodine is periodically used in people with ophthalmopathy because hypothyroidism after treatment is associated with worsening eye conditions. The use of tobacco is associated with the progression of Graves' ophthalmopathy. Most radioactive iodine is excreted in the urine, saliva, and feces within 48 hours. However, for several weeks, frequent toilet flushing and regular hand washing are recommended. Avoid close contact with others, especially young people and pregnant women, for 1 to 3 days [30, 31].

Surgery

The advantages of hyperthyroidism surgery contain an almost 100% recovery rate, early detection of malignancies, and rapid thyroid diseases or hypothyroidism. Risks of thyroid operation before and after surgery include bleeding, recurrent laryngeal nerve damage, hypocalcemia, and the need for a lifelong thyroid hormone substitute. If compressive symptoms, concurrent hyperparathyroidism, large or sub sternal goiters, low radioactive iodine uptake, pregnancy planning, pregnancy in the second trimester, Graves' disease with Graves' ophthalmopathy, or the indicated quick definitive therapy in thyrotoxicosis present, surgery should be considered. Surgery is the most cost-effective final therapy for Graves' disease, toxic adenomas, and toxic multinodular goiters, with failure rates of less than 1% when performed by a high-volume surgeon. For Graves' disease and toxic multinodular goiters, a total thyroidectomy is indicated. Toxic adenomas, on the other hand, should have their thyroid glands evaluated with ultrasonography. For isolated toxic adenomas, a lobectomy may be performed if it is possible. This has the added benefit of allowing you to avoid hormone replacement treatment. To achieve euthyroid status, patients should take ant thyroid medicines before surgery. The beta-blockade should be started in high-risk patients, such as the elderly, those with severe thyrotoxicosis, and those who have a history of cardiovascular disease. It is recommended calcium carbonate and activated vitamin D treatment before and after surgery to reduce the risk of postoperative hypocalcemia. TSH should be tested 6–8 weeks after complete thyroidectomy or lobectomy to determine residual thyroid function or the dosage of thyroid hormone replacement. Before they reach euthyroid, around 75% of individuals will require dosage changes in the future [1, 25, 27].

Conclusion

Hyperthyroidism is a disease that is often neglected. Maintaining a high suspicion index is important for the immediate care physician. Treatment recommendations are based on a mixture of elements, such as patient age, comorbidity, disease severity, underlying cause, and underlying cause. The main treatment choices are operation, ant thyroid medicines (thionamides), and radioactive iodine. All processes had equal life parameters quality, but in general, radioactive iodine was the most cost-effective.

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