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A Review on Thyroid and its Management

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Abstract: ***The thyroid hormones control the metabolism of cells, which is their speed of activity. Thyroid hormones regulate the rate of oxygen consumption. Although Thyroid hormones have a similar effect and influence the proper working of all body cells, their action is particularly evident in certain tissues and for certain functions. This review focuses on introduction of thyroid, symptoms, causes of hypothyroidism, hyperthyroidism and laboratory evaluation and last but not the least treatment and evaluation of thyroid.***

Keywords: Thyroid, Symptoms, Causes, Treatment and Management

1. Introduction

The thyroid is able to compensate, continue to produce a normal amount of thyroid hormone despite disruption, in response to some of these agents by increasing serum TSH. The success of this compensation can be assessed in the adult based on markers of thyroid hormone action, but is much more difficult to determine during fetal development and in infants and children. Brain development is the best characterized pathway that is thyroid hormone dependent and vulnerable to thyroid hormone disruption .Local thyroid hormone activation and timing of availability of tri-iodothyronine (T3) is critical in brain and sensory development Agents that interfere with thyroid hormone signalling during this period are the most difficult to detect and quantitate. A significant focus in clinical thyroid disease is to detect and evaluate thyroid disease at the earliest stages .Recent efforts in assessing the impact of environmental agents that disrupt thyroid function have focused on identifying the earliest and subtle effects [1].The sensitive thyroid stimulating hormone (TSH or thyrotropin) assay has become the single best screening test for hyperthyroidism and hypothyroidism, and in most outpatient clinical situations, the serum TSH is the most sensitive test for detecting mild thyroid hormone excess or deficiency. Therapeutic options for patients with Graves' disease include thyroidectomy (rarely used now in the United States), anti thyroid drugs (frequently associated with relapses), and radioactive iodine (currently the treatment of choice). In clinical hypothyroidism, the standard treatment is levothyroxine replacement, which must be tailored to the individual patient. Awareness of subclinical thyroid disease, which often remains undiagnosed, is emphasized, as is a system of care that incorporates regular follow-up surveillance by one physician as well as education and involvement of the patient [2].

2. Symptoms

2.1 Hyperthyroidism

The following list illustrates the spectrum of possible signs and symptoms associated with the various causes of hyperthyroidism-

- Nervousness and irritability
- Palpitations and tachycardia
- Heat intolerance or increased sweating
- Tremor
- Weight loss or gain
- Alterations in appetite
- Frequent bowel movements or diarrhea
- Dependent lower-extremity edema
- Sudden paralysis
- Exertional intolerance and dyspnea
- Mental disturbances
- Impaired fertility
- Changes in vision, photophobia, eye irritation, diplopia, or exophthalmos
- Sleep disturbances (including insomnia)
- Fatigue and muscle weakness
- Pretibial myxedema (in patients with Graves' disease)

2.1.1 Hypothyroidism

Hypothyroidism is a disorder that occurs when the thyroid gland does not make enough thyroid hormone to meet the body's needs. Thyroid hormone regulates metabolism the way the body uses energy and affects nearly every organ in the body. Without enough thyroid hormone, many of the body's functions slow down. About 4.6 percent of the U.S. population age 12 and older has hypothyroidism

The signs and symptoms of hypothyroidism can include one or more of the following [3]:

- Fatigue
- Weight gain from fluid retention
- Dry skin and cold intolerance
- Yellow skin
- Coarseness or loss of hair
- Hoarseness
- Goiter
- Reflex delay, relaxation phase
- Ataxia
- Constipation
- Memory and mental impairment
- Decreased concentration
- Depression
- Irregular or heavy menses and infertility
- Myalgias
- Hyperlipidemia
- Bradycardia and hypothermia
- Myxedema fluid infiltration of tissues

Although most physicians can diagnose and treat hypothyroidism, in certain situations a clinical

endocrinologist experienced in the spectrum of thyroid disease would be most likely to recognize the more subtle manifestations of hypothyroidism and most skilled in the physical examination of the thyroid gland. Consultation with an endocrinologist is recommended in the following situations:

- Patients of age 18 years or less
- Patients unresponsive to therapy
- Pregnant patients
- Cardiac patients
- Presence of goiter, nodule, or other structural changes in the thyroid gland
- Presence of other endocrine disease

Not all patients with chronic thyroiditis have hypothyroidism, and if it is present, it may not persist. Rarely Patients with chronic thyroiditis have a change from a hypothyroid to an on suppressible euthyroid state or even to a hyperthyroid state because of the development of Stimulating TSH receptor auto antibodies (TSI or TRAb) of Graves' disease.[4]

2.1.2 Causes of Hyperthyroidism

Hyperthyroidism is the consequence of excessive thyroid hormone action. The causes of hyperthyroidism include the following [5]:

- Toxic diffuse goiter (Graves' disease)
- Toxic adenoma
- Toxic multinodular goiter (Plummer's disease)
- Painful subacute thyroiditis
- Silent thyroiditis, including lymphocytic and post partum variations
- Iodine-induced hyperthyroidism (for example, related to amiodarone therapy)
- Excessive pituitary TSH or trophoblastic disease
- Excessive ingestion of thyroid hormone.

Causes of Hypothyroidism

Hypothyroidism results from under secretion of thyroid hormone from the thyroid gland. In the United States, the most common cause of primary hypothyroidism is chronic autoimmune thyroiditis (Hashimoto's disease). Other causes are surgical removal of the thyroid gland, thyroid gland ablation with radioactive iodine, external irradiation, a radio synthetic defect in iodine organification, replacement of the thyroid gland by tumor (lymphoma), and drugs such as lithium or interferon. Secondary causes of hypothyroidism include pituitary and hypothalamic disease. Patients should undergo assessment for the cause of their hypothyroidism [6].

2.2 Diagnosis

A comprehensive history should be elicited, and a thorough physical examination should be performed including the following:

- Weight and blood pressure
- Pulse rate and cardiac rhythm
- Thyroid palpation and auscultation (to determine thyroid size, nodularity, and vascularity)
- Neuromuscular examination
- Eye examination (to detect evidence of exophthalmos or ophthalmopathy)
- Dermatologic examination
- Cardiovascular examination
- Lymphatic examination (nodes and spleen)

2.3 Laboratory Evaluation

The sensitive TSH assay is the single best screening test for Hyperthyroidism, and in most outpatient clinical situations, the serum TSH is the most sensitive test for detecting mild (subclinical) thyroid hormone excess or deficiency. Other laboratory and isotope tests may include the following:

- T 4 or free T 4
- Triiodothyronine (T4) radioimmunoassay (RIA) or free T3 abnormal results of T 4 or T measurements are often due to binding protein abnormalities rather than abnormal thyroid function. Therefore, total T 3 4 or T must be determined in conjunction with some measure of their thyroid hormone binding such as T resin uptake or assay of thyroidbinding globulin to yield a "free thyroid hormone estimate." Commercial laboratories often call these methods free T 4 or free T3 even though they do not measure free hormone directly.
- Thyroid autoantibodies, including TSH receptor antibodies (TRAb) or thyroid stimulating immunoglobulins (TSI) These studies are not routinely necessary but may be helpful in elected cases, such as in patients with hyperthyroidism during pregnancy.
- Radioactive iodine uptake
- Thyroid scan with either 123 I (preferably) or Tc pertechnetate. Such a scan is not a thyroid function test but is done to help determine the cause of the hyperthyroidism. The scan may also be useful in assessing the functional status of any palpable thyroid irregularities or nodules associated with a toxic goiter .Reverse T testing is seldom, if ever, helpful in clinical practice.

2.4 Treatment and Management

Three types of therapy are available for Graves' disease:

1) Surgical intervention,
2) Anti thyroid drugs,
3) Radioactive iodine.

Surgical Intervention

Some physicians prefer surgical treatment of pediatric patients with Graves' disease or patients with very large or nodular goiters. Potential complications associated with surgical management of Graves' disease include hyperparathyroidism and vocal cord paralysis in a small proportion of patients. Surgeons trained and experienced in thyroid surgical procedures should perform this operation.

Anti-Thyroid Drugs

Antithyroid drugs, methimazole and propylthiouracil, have been used since the 1940s and are prescribed in an attempt to achieve a remission. The remission rates are variable, and relapses are frequent. The patients in whom remission is most likely to be achieved are those with mild hyperthyroidism and small goiters.

Radioactive Iodine

Radioactive iodine therapy is safe, but most treated patients become hypothyroid and require lifelong thyroid

replacement therapy. Some clinical endocrinologists are hesitant to use radioactive iodine to treat patients of childbearing age, but no evidence has suggested that such therapy has any adverse effects. Specifically, studies have found no effect on fertility, no increased incidence of congenital malformations, and no increased risk of cancer in patients treated with radioactive iodine or in their offspring. After administration of a dose of radioactive iodine, thyroid replacement therapy should be carefully initiated during the time the patient's thyroid function passes through the normal range into the hypothyroid range. The final thyroid replacement dose must be individualized. This approach promptly resolves the hyperthyroidism with a minimum of hypothyroid morbidity [7].

Patient Care in Thyroid Disorder

Once the diagnosis of Grave's disease with hyperthyroidism has been established, the patient should be given a complete explanation of the illness and options for treatment. The goal is to involve the patient as a partner in the medical decision-making process and care, rather than Have the endocrinologist dictate the choice of therapy. Patients who elect to receive, radioactive iodine should be given an explanation of the treatment, and a consent form for such therapy should be signed. After receiving radioactive iodine, patients should be given an instruction sheet that itemizes appropriate precautions and explains follow-up Management .[8]The radioactive iodine uptake should be assessed before treatment to ensure adequate uptake at the time of therapy, to rule out the presence of a variant of thyroiditis or iodine contamination, and to help determine the dose of radioactive iodine. A thyroid scan is also useful in distinguishing toxic nodular goiter and toxic adenoma from Graves' disease. Typically, toxic nodular goiter is more resistant to radioactive iodine and frequently necessitates use of a larger dose. ßAdrenergic antagonists provide symptomatic relief and can be administered before radioactive iodine is given. Because patients with hyperthyroidism may be relatively resistant to the effects of ßadrenergic blocking agents, larger and more frequent doses may be necessary. The dose of these drugs can be tapered and discontinued once the patient no longer has hyperthyroidism. In addition, in severe thyrotoxic states, adjuvant treatment can include organic or inorganic iodides and anti thyroid drugs after radioactive iodine therapy. After treatment with radioactive iodine, patients should have follow-up examinations at frequent intervals (varying from 4 to 6 weeks, but individualized for each case) until they are euthyroid and their condition is stable. Most patients will require full thyroid hormone replacement therapy. Patients usually become hypothyroid within 3 months and could begin receiving partial replacement doses of levothyroxine approximately 2 months after receiving radioactive iodine. This schedule is determined by laboratory testing and clinical evaluation. At this time, the patient's thyroid status is quickly changing from euthyroid to hypothyroid, and the TSH level may not be a good indicator of function because it fails to increase quickly. From 2 weeks to several months may elapse before TSH responsiveness is recovered, and free thyroid hormone estimate tests are more accurate than TSH values during this interval. When the condition of patients has stabilized, the frequency of visits and re-evaluations can be extended. A common schedule for follow-up consultations is at 3 months, at 6 months, and then annually, but this can be modified on the basis of the physician's judgment [9].

3. Conclusion

The pathology of the thyroid gland presents the pathologist with a particular set of diagnostic problems. If best practice and the minimum data set guidelines are adhered to, the correct diagnosis should be reached in most cases. Newer techniques such as immunocytochemistry can certainly be helpful in more difficult cases but, as in all areas of pathology, histological features take precedence and good communication with the relevant clinical colleagues is paramount.

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