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Hyperthyroidism and Thyrotoxicosis Etiologies

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Abstract:

Hyperthyroidism is a multifactorial etiology thyroid complaint, and the right combination of genetic, ecological predispose leading to immunological broad-mindedness, become distinct as low or reduced TSH levels with elevate T3 levels and , or raised T4 levels. The etiologically related risk issues to the growth of hyperthyroidism are burning, excess iodine, stress, contagion by some pathogens, genetic predilection. The most common cause of hyperthyroidism is Graves disease results from loss of resistant tolerance to thyroid antigens particularly the TSH receptor antigen and is strong by thyrotoxicosis and toxic multinodular goiter which is another disease of hyperthyroidism is most commonly found in elder people. The increased hormones of the thyroid gland lead to an uneven function as it is associated with long-term complications that are severe. Thus hyperthyroidism is associated with Considerable morbidity. Therefore early case findings of it and adequate teaching of right treatment are important, the researches of pathoimmunologic processes as well as genetic background of deseases can help to give the precise, and personalized therapeutic approach to.

Keywords: Thyroid immune disease, hypothyriodism graves diseas, toxic multinodular goiter

Thyroid gland:

The thyroid gland is an endocrine gland in the inferior neck that theatres a main role in the switch of basal metabolic rate and endorses somatic and mental development (Khan & Farhana, 2022). It is subdivided into lobules by septae that project down from the pill.The thyroid lobules are made up of several types of functional units called thyroid follicles (Rykova, et al ; 2019) . The thyroid sacs are the physical and useful units of a thyroid gland, the Follicular cells are endodermal derivatives that secrete Thyroid hormone(Armstrong, et al; 2023) , the socializing form of this hormone is thyroxine, tetraiodothyronine (T4) lengthways with a minor amount of triiodothyronine (T3) (Kaplan, et al ; 2015)

Not only do thyroid disorders increase and will increase in the future years (Yoo and Chung 2016) they also are health problems in Iraq (AL- Ramahi 2011 ;Jwaid 2011 ;AL-Janabi 2013 ; Mansoor, 2017) reported that the rate of disorders prevalence was higher, in female than in male.

Thyroid Hormones

Hypothalamic Neurons secrete thyrotropin releasing hormone (TRH) in response to low circulating thyroid hormone levels, thereby stimulating the forward pituitary to announcement thyroidal inspiring hormone . (Khan and Farhana., 2022) .released TSH binds to thyroid stimulating hormone receptors (TSHRs) inducing the catalyst thyroid peroxidase (TPO) to react to thyroglobulin (Tg) TSH causes results in iodinisation thus forming thyroxin (T4). Thyroxin is secreted by the thyroid, and it is converted into triiodothyronine (T3), the active form, by deiodination in the target tissues (Muhammad , et al ; 2023 ; Armstrong, et al; 2023) with the major part of T3 nonthyroidal most derived outside the thyroid a small amount being locally produced in the thyroid from T4 because it is still present in the gland. Thyroxine is the main circulating form of the hormone and has a lengthier half-life than T3, Thyroxin is the chief secretory creation of the thyroid gland.. (Kohrle and Fradrich 2022) .

Hyperthyroidism and Thyrotoxicosis :

Hyperthyroidism is a common disorder of the thyroid and is defined as a syndrome associated with excessive activities related to the manufacture of thyroid hormone. It is, as a matter of fact, one of the very common misconceptions that the term thyrotoxicosis and the term hyperthyroidism are used interchangeably. Thyrotoxicosis is actually defined as exposure to an excessive amount of thyroid hormone by the tissues. (Ross, et al; 2016) While the presence of hyperthyroidism does lead to thyrotoxicosis hence technically making the two terms interchangeable in usage. There is however a distinct difference to be made between them. (Mathew , et al; 2023) Thyrotoxicosis is inappropriately high function of thyroid hormone action resulting in expression of the diseases that cause clinically significant morbidity and mortality (Bayraktar , et al; 2023) The commonest manifestation of overt hyperthyroidism back home is low or suppressed TSH levels with high T3 and/or high T4 levels. if only T3 is raised and there is low/repressed TSH and normal T4 (“T3 toxicosis”), it is subclinical hyperthyroidism is low or repressed TSH with normal T3 and T4 heights. In overt or subclinical hyperthyroidism the rate amount of glitches are (Mathew , et al; 2023; Biondi and Cooper . 2018)

Main cases are directly induced by inherent pathology of the thyroid gland. Whereas subordinate hyperthyroidism is not, it accrues from chanced circulation of increased TSH which over-stimulates the thyroid. When the gland is overactive the body’s activities increase and an individual may feel nervousness, anxiety, fast heart rate, hand tremor, excessive sweating, weight loss with increased appetite, sleep difficulties, and other symptoms that may be caused by various disorders. Hyperthyroidism can also result from toxic multinodular goiter, toxic adenomas, and a variety of other rare syndromes. (Kahaly , 2020)

Causes of Hyperthyroidism:

A- Gravse Disease

More prevalent than toxic nodular goiter, the most shared form of hyperthyroidism encountered in scientific practice is an organ-specific autoimmune complaint of the thyroid called Graves’ disease. The attendance of circulating anti-thyroid-stimulating hormone receptor (TSH-R) stimulating autoantibodies reasons hyperthyroidism. It is established by thyrotoxicosis..

The principal pathogenic mechanism for inducing thyroid growth ("hyperplasia" and "hypertrophy"), TRAb both at cells of the thyroid and at orbital fibroblast, is (Smith and Hegedus, 2016; Rattanamusik et al; 2023). The risk for GD is about 3% for women and 0.5% for men between the age of 30-60 years and the most common cause of hyperthyroidism (Antonelli et al; 2020), it is said to bind the TSHR which takes up TSHR-binding inhibitory immunoglobulins in case of TSHR antibodies and TRAb is generally acronymed for any kind of antibody specifically interacting with the receptor (TSHR) when assessed by competitive binding assay., (Kahaly and Diana 2017).

B- Toxic Multinodular Goitre (TMG) :

Toxic Multinodular Goitre is a rare thyroid state that causes hyperthyroidism in elderly females. It comprises the autonomous functioning nodules, which secrete extreme thyroid hormone Growth of the thyroid depends on TSH, which is a pituitary hormone and when thyroid gland increases in size is referred to as goiter. There may be TSH receptor agonists or antibodies which stimulate the receptors forming a diffusely goiter. (Rattanamusik et al 2023), the goiter might be diffusely enlarged, nodular type and may be non-toxic in euthyroid as well. The common precipitating factors include increased age, low iodine intake and previous external irradiation. Multinodular goiters generally grow steadily; however, in some cases, such as when there is substernal extension, compression appears from the trachea and esophagus and results in dyspnea and dysphagia (Fanning et al 2018); usually, means higher ratio among the female population compared to men. (Sujeethra , et al ; 2020).

C - **Thyroid gland carcinoma:**

These are the types of hyperthyroidism that develop into thyroid gland carcinoma given excessive stimulation of the thyroid hormone: follicular thyroid cancer and metastatic differentiated thyroid carcinoma (Takedani, et al; 2021). Follicular thyroid cancer, or functioning thyroid cancer, occasionally causes hyperthyroidism by producing thyroid hormones on its own. In this type of cancer, the tumor starts in small sacs known as follicles in the thyroid. The greatest shared reason of hyperthyroidism is when the cancer cells start to produce the thyroid hormones thyroxine and triiodothyronine (Kahaly, 2020). His family members had exposure to radiation, iodine uptake, diabetes, obesity, Hashimoto thyroiditis, exogenous estrogen use and dietary choices.. (Liu ,et al; 2017 ; Ashorobi and Lopez , 2023)

D - Human chorionic gonadotrophin (HCG) -Induced Hyperthyroidis :

HCG is a hormone secreted by embryos throughout pregnancy. Primarily responsible for maintaining high progesterone level in the ovary, its structure resembles TSH, allow it to bind to thyroid follicle cell receptors? may analysis of hyperthyroidism be wasted if the hypermetabolic indications are just credited to the trophoblastic disease?. (Pereira and Lim 2021 ; Ethan, et al; 2023)

E – medication :

Amiodarone is a very common medication used to treat atrial fibrillation. Since it is iodine-based, it can lead to hyperthyroidism especially in those individuals who are predisposed to this disease, for instance, patients having thyroid nodules. This is referred to as Type I amiodarone-induced hyperthyroidism. (Takedani , et al ; 2021**).**

F- **Iodine :**

Iodine compositely donates to the production of thyroxine and triiodothyronine by the thyroid. It is usually derived from dietary sources, but high increases in the intake, as with supplements, can have an influence on the production of the thyroid hormones leading to hyperthyroidism (Kahaly, 2020). The question is open for iodine if its excess or deprivation is responsible for thyroid cancer(Aceves, et al; 2021).

E- Vitamin D deficiency:

Indeed, selenium and Vitamin D reserve a dangerous home among the front nutritional factors as has been bare by the latest investigation on the environment triggers for AITD. An insufficiency of vitamin D is not only one of the driving factors but because it is associated with deftness in AITD. The most recent meta-analysis has proven that patients with GD are more likely shown to have lack of Vitamin D, the serum vitamin D levels may impact the prognosis of patients with GD. Vitamin D deficiency is a risk for development of the autoimmune thyroid diseases or a consequence.. (Leko , et al ; 2023).

Environmental factors:

1) A-Molecular Mimicry:

One model rpecr that a ppetide epitope in an environmental antigen was identified having the same amino acid sequence as that present in TSH receptor, TPO, or TG initiated molecular imitation heghtened immune reactivity to component of the body and had produced the damage. (Yoo, and Chung , 2016) Some mimickry that proteins in common gut bateria also mimic Reoviral antigens and body tisue molecular imitation of virus, viral damage to the that entere via another mechanism sych as secretion of cytokines (Benvenga and Guarneri, 20150)

It may represent another environmental agent that influences the progression of autoimmune thyroid diseases. An infection by H. pylori in the gastric mucosa contributes to the development of clinical manifestations associated with gastritis, and this bacterium infects the individual (Al-Mofarji, 2011; Mansoor, 2017). Benvenga and Guarneri, and bioinformatics data induced are Borrelia, Yersinia, Clostridium botulinum, Rickettsia prowazekii and Helicobacter pylori; likely pathogenic importance is in Toxoplasma gondii, approximately Bifidobacteria and Lactobacilli,Candida albicans Treponema pallidum and hepatitis C virus for autoimmune thyroid illness., (Benvenga and Guarneri 2016).

B- Stress :

Non–antigen-specific mechanisms might play a role in the induction of immune tolerance associated with emotional or psychological stress in nonthyroidal autoimmune diseases, presumably effects of cortisol on immune cells by increasing immune reactivity which leads to autoimmunity against thyroid (Iddah & Macharia; 2013). Stressful life events, such as separation or tragedy, can trigger the onset of Graves' disease in those with genetic susceptibility. It results in the release of Cortisol and corticotrophin hormones that cause immune suppression and overcompensation, just like postpartum periods stress-related mediators such as glucocorticoids and regulators of homoeostasis such as Thyoid hormones. (Navarro. 2020).

E-Smoking

Smoking is slight in Graves' risk but common of antithyroid antibodies, varies by race, rises with age and falls with smoking. (Ragusa , et al ; 2019) burning upsurges risk for GD around 2 fold , while liquor use and bodily action seem unconnected to it. (Leslie and DeGroot,2015).

F- Other factors

Thyroid trauma, ethanol injections, and radio-iodine treatment created risk factors for the subsequent development of Graves disease and ophthalmopathy because each of these procedures causes a release of thyroid antigens, setting off an autoimmune response against thyroid-stimulating hormone (TSHR) Leslie and DeGroot(2015) . Three decades ago, it was recognized by Leslie and DeGroot (2015) that energy to the thyroid in new people was shadowed by a higher occurrence of positive thyroid antibody tests. Dangerous iodide treatment for poisonous multinodular goiter and Ethanol inoculation for treatment of poisonous thyroid nodes have both been shadowed by the development of autoimmune Tombs' illness. (Yoo, and Chung, 2016.) .

Global warming and sudden weather variations could play an important role in increasing the incidence of autoimmune thyroid diseases other than AITD. In addition to this Rapid industrialization and environmental toxin exposure is blamed for AITD. (Yoo, and Chung , 2016)

Susceptibility genes :

These are immune response control genes which in turn control AITD development . The major loci involved AITD have been identified as CTLA4,CD40,CD25(foxP3),protein tyrosine phosphatase ,non receptor type 22 and several cytokinerelgulation genes Hasham and Tomer. Major histocompatibility complex (MHC) antigens associated with thyrotoxicosis in indo-Chinese have been well reviewed and are that this represents a disease of heredofamilial predisposition. (Messaaoui et al.,2012;Ghazi et al ,2015)

Previous Studies On Tumor Necrosis Factor (TNF) Superfamily Immune Regulatory Gene That Is Expressed On Thyroid Follicular Cells And CD40 C/T1 Polymorphism Had Been Associated With GD. Dashdamirova et al; 2022).

Pathogensis :

Other common features are tachycardia increased pp but wt loss signs are increased perspiration and goiter occasionally exophthalmos. physical findings fine skin and hair tremulousness hyperactive heart Plummer's nails muscle weakness may coexist in a patient with Graves' disease. There maybe some eye signs al cardiac murmurs. An algorithm keeps you while rewriting the data and does it right.

thyroid hormones are key regulators of metabolism since it is the case that they have preeminent belongings on both cholesterol and fatty acid metabolism development and growth. This results in reduced levels of LDL cholesterol, HDL cholesterol, and TGs in the serum. Not fair any levels but many of them compared to when a being doesn't have Hyperthyroidism.

Thyroid Hormones effect a large panel of genetic factor related to lipogenesis. Amongst the lipid's specific receptor is a ligand-dependent transcription factor Hormone action causes a cascade of processes initiated at binding sites contingent on this premise if special is indeed special it is only evident complicated organization of cellular activities would be result from the pathway.

The two major isoforms of receptors are α (THRα) most prevalent in heart and bone tissue, and β (THRβ) most plentiful in the liver. In the absence of their ligands, THRs would be bound to TREs and act as transcriptional repressors for target genes, by recruitment of a co-repressor complex. It can so be hypothesized that in the presence of ligands there will be conformational changes in THRs preceded accompanying the recruitment of a co-activator complex which will now activate target genes.

One of the hormone's effects on bone is via. inspiration of osteoblasts and osteoclasts which results in faster bone remodeling, extra hormone can accelerate the process of natural bone turnover therefore leads to osteoporosis. (Braun and Schweizer 2018).

How the synergy of excess thyroid hormone with the sympathetic nervous system in human turns out to be positive is not settled. Thyroid hormone directly stimulates heart metabolism and sympathetic effects in other organs. Irregular thyroid hormone causes uneven sex hormones in women and fertility is reduced.. (Magtooph,2015 ; Ethan , et al ; 2023).

Epidemiology :

Hyperthyroidism Prevalence Varies By Ethnic Group (Mathew , et al ; 2023). The prevalence of GD is aproximatrly 1% to 1.5% of the general population with an incidence of 20 to 30 new cases /100 000/year.

Some of the meta-analyses of studies have projected the disease to affect about 1% of the population. In contrast, Graves' is stated four to five times more common in females than in males. The lately reported incidence for England is 1-2/1000 population per year, which is much higher than previously measured rates of about 0.3/1000 in this country. Incidence in females is much advanced compared to in males. Some other meta-analyses of studies have estimated the disease to touch about 1% of the population. In difference, Graves' is reported four to five times more common in females than in males. The lately reported incidence for England is 1–2 per 1,000 population per year, which is much higher than before measured rates of about 0.3 per 1,000 in this country. Incidence in females is much more as likened to in males.

The estimated occurrence of hyperthyroidism in the general population is about 1.3% as revealed by the study. Mathew et al reported a number from their meta-analysis for Europe of 0.75% 0% for explicit hyperthyroidism among the Chinese was another discovery with a similar figure estimated at 0.78 (Mathew et al; 2023) The risk percentage struck women aged between 30 to 60 years is that of 3% Whereas men from that same age collection

have a risk percentage of 0.5% it is the greatest common etiology of hyperthyroidism in West countries.Antonelli, et al; 2020)

Conclusion

Hyperthyroidism is of multifactorial etiology, the right mixture of genetic, environmental, and endogenous issues is obligatory for the start of the disease process. So, early detection of this disorder is very significant.

Conflicts Of Interest

The authors declare no conflicts of interest.

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