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REVIEW

Review on Thyroid Disorders, Epidemiology and Treatment Methods

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ABSTRACT

Thyroid disorders are commonly overwhelming health conditions reported worldwide. The prevalence of thyroid disorders such as hypothyroidism and hyperthyroidism is increasing in developed and developing countries, including India.This is due to change in traditional foods to Besides low / insufficient iodine intake, smoking, ageing, genetic susceptibility, lifestyle, usage of new medicine, endocrine disrupting chemicals and immune status of an individual are the key determinants for thyroid disorders. This review emphasizes the various disorders of the thyroid gland and, its epidemiology and treatment methods.

1. Introduction

The thyroid gland is a bilobed connected through the isthmus and located in the front of the neck. The thyroid hormones such as triiodothyronine (T3 form) and thyroxine (T4 form) are the secretary products of the thyroid gland. The two hormones which regulate the synthesis and release of thyroid hormones are thyroid-stimulating hormone from the anterior pituitary and thyrotrpin-releasing hormone from hypothalamus [1]. The T3 and T4 hormones of the thyroid gland are essentially required for normal growth and oxidative phosphorylation in all the nucleated cells of the body [2]. The primary thyroid diseases notified commonly are hypothyroidism and hy-perthyroidism due to infections in the thyroid gland. The larger parts, widespread among all the endocrine diseases are thyroid disorders and are prevalent in all countries in-cluding India [3].

Table 1. Iodine intake/deficiency and thyroid disorders

S.No.	Daily intake of iodine/ deficiency	Disorders
1	50 mg	Goitre and is endemic
2	25 mg	inborn hypothyroidism
3	All age groups	Goitre, Hypothyroidism, Impaired mind utility, Augmented defenselessness to nuclear radiation
4	Adult group	Goitre and its complications, Iodine induced hyperthyroidism
5	Child and adoles-cent groups	Augmented infant death, Retarded mind and physical growth
6	Neonate group	Augmented neonatal deaths, Perinatal hypothyroidism, Retarded mind and physical growth
7	*In utero*	Abortions, Stillbirths, Neurologic cretinism, Inborn anomalies, Psychomotor defects and Myxedematous cretinism

The major reason for thyroid dysfunctions reduces intake of iodine. Approximately 1/3rd population resides

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in the iodine deficient areas in the world [4]. The amount of iodine intake shows various thyroid disorders and is presented in the Table 1. Literature from developed countries emphasized the prevalence of various thyroid disorders [5]. About 50, 15, 10, 5 and 3.5% of western population are suffering with thyroid disorders like microscopic nodules, palpable goiters, altered levels of thyroid-stimulating hormone, overt hypothyroidism or hyperthyroidism and occult papillary carcinoma [5].Though countries all over the world initiated the control programmes for thyroid disorders (iodine deficiency diseases), but still many parts of the world its occurrence is reported. Creating awareness among communities living in the different parts of the world is most important. Hence, the present review focused on thyroid disorders, its epidemiology and preventive methods.

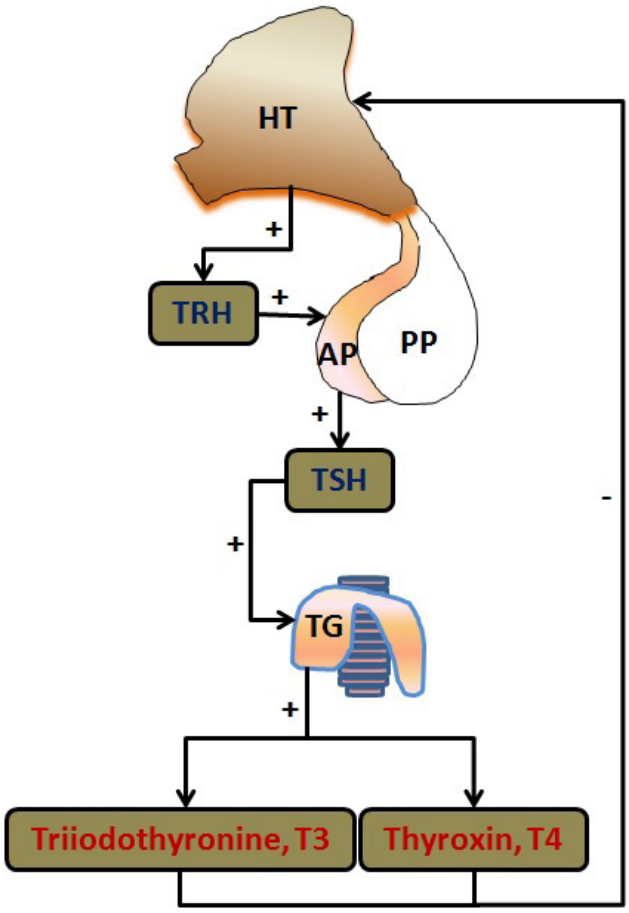

Figure 1. The synthesis and regulation of triiodothyronine and thyroxin from the thyroid gland (TG)

Note:

Where HT: Hypothalamus; AP: Anterior Pituitary; PP: Posterior Pituitary; TRH: Thyrotrophin Releasing Hormone; TSH: Thyroid Stimulating Hormone. '+' and '-' denotes positive and negative regulation respectively.

Table 2. Iodine intake/deficiency and thyroid disorders

S.No.	Thyroid disorder	Diseases associate with
1	Graves' disease	Thyrotoxicosis/ Hyperthyroidism
2	Toxic nodular goitre 1) Toxic adenoma 2) Toxic multinodulargoiter	
3	Thyroiditis	
4	TSH secreting pituitary tumors	
5	hCG induced hyperthyroidism (gestational and trophoblastic disease associated)	
6	Iodine induced hyperthyroidism (iodine and Amiadarone)	
7	Thyrotoxicosis factita	
8	Goitrous hypothyroidism (Hashimoto's thyroiditis, iodine deficiency and lithium)	Hypothyroidism
9	Congenital hypothyroidism	
10	Atrophic hypothyroidism (Hashimoto's thyroiditis and post ablative)	
11	Central hypothyroidism III. Euthyroid i) Diffuse nontoxic (simple) goitre ii) Nodular thyroid disease (solitary nodule and multinodular) iii) Thyroid neoplasia (follicular adenoma and thyroid malignancy)	
12	Thyroiditis (inflammation in thyroid gland) Postpartum thyroiditis (in women after delivery)	

2. Thyroid Gland, Hormones and Disorders

Small, bilobed structures located in the neck are called thyroid gland (20-25 g) secrets minor (7%) triiodothyronine (T3) and major (93%) thyroxin (T4) hormones from its follicles. It is clear from Guyton and Hall [1] that the biological activity of T3 is 3-5 folds higher than T4. Both of these are much similar to each other in holding two tyrosine amino acids. The difference between these two is holding three iodine atoms in T3 and four iodine atoms in T4 [6]. The regulation of synthesis and secretion of T3 and T4 hormornes is under the control of thyrotrophin (also called thyroid stimulating hormone; TSH) released from the anterior pituitary, which is stimulated by tripeptide releasing hormone/thyrotrophin releasing hormone (TRH) from hypothalamus [1] (Figure 1).

Thyroid hormones, virtually affects each and every cell in the body by stimulating the glucose oxidation enzymes, thereby increases body heat and basic metabolism. The high levels of thyroid hormones increase in the basic metabolic reactions 60 to 100% in body cells, which ultimately increases in the rate of energy production from food, transcript thereby translation and protein degradation. Even the thyroid hormones stimulate many other endocrine hormone synthesis and release. According to Elaine and Maieb [6] fetal growth and development is promoted by T3 and T4 thyroid hormones. The variations in the levels of TSH were identified in thyroidal and non-thyroidal disorders. Low levels of T3 and T4 an indication of primary hypothyroidism induces the release of TSH and increases its serum levels. Whereas in hyperthyroidism elevated T3 and T4 levels through a negative feedback mechanism suppresses the serum TSH levels. In children,

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hypothyroidism reduces skeletal growth (dwarfism) and hyperthyroidism excessive bone growth (gigantism) besides mental retardation in throughout their life in both the cases [1]. The dysfunction of the thyroid gland is associated with various diseases and is mentioned in the Table 2.

Moreover the diagnosis of thyroid disorders is difficult. Pretreatment of these disorders is uncommon due to difficulty in diagnosis of its pre and early stages. Though the simple blood test can determine the levels of thyroid hormone, doctors are suggestive to do this due to the symptoms of this disorder are mimicking with many other diseases. In case of subclinical thyroid malfunctioning is found with no or few hypothyroidism symptoms. Mostly the blood levels of 0.5 to 5 mIU/L TSH were found normal and can be called 'euthyroid', beyond these levels considered as malfunctioning of the thyroid gland. A person with a condition of low/over thyroid function determines the hypo/hyperthyroidism or Grave's disease/ Hashimoto's thyroiditis the autoimmune diseases of the thyroid. Thyroid autoimmune disorders play an unpredictable role in thyroid functioning (Table 3). The best method identifies and treat hyper or hypothyroidism is to maintain periodical blood test reports along with the patient's personal and symptoms history and risk factors.

Table 3. Thyroid autoimmune diseases and their symptoms

S.No.	Antibodies of autoimmune thyroid disease	Symptoms	References
1	**Antibodies of thyroid stimulating hormone receptor**	**Identified in Hashimoto's and Grave's cases with 10 and 90 % respectively.**	[7]
2	**Antibodies of thyroid peroxide**	**Identified in Hashimoto's and Grave's cases with 95 and 70 % respectively.**	[8]
3	**Antibodies of thyroglobulin**	**Identified in Hashimoto's and Grave's cases with 80 and 50 to 70 % respectively.** Elevated in thyroid cancer.	[9]

3. Epidemiology of Thyroid Disorders

The rate of prevalence of hyperthyroidism is increasing day by day. Epidemiology says about 1/3rd of the Indian community is suffering by hyperthyroidism and among these 39% are suffering with goiter [10]. Reported 2.7 and 0.23% of UK women and men respectively is identified with hyperthyroidism [11]. The cases with hyper or hypothyroidism became common in the United States of America. About 12% of people are suffering with the disorder of the thyroid and is increasing drastically though the US Government has taken steps to consume iodized salt throughout the country. The increased occurrence of diffuse goiter is reported in women of pre-menopausal stage and its ratios 4:1 in women and men [12]. The occurrence of hypothyroidism was also reported in countries like Norway, Japan, Denmark and Sweden [13-17]. About 2% of women are suffering with hypothyroidism during pregnancy [18,19]. However still many places in the each and every corner of the world need to be surveyed for exact statistics and prevalence of hypothyroidism.

4. Treatment Methods of Thyroid Disorders

The thyroid Hashimoto requires treatment only the patients who are not having autoimmune antibodies against it. In this case, antibodies are produced for longer periods (years) and facilitates the release of sufficient amount of thyroid hormones. However, it is clear from the literature about more than 80 among 100 suffering with hypothyroidism are needed treatment [20]. The condition where over or low production of thyroid hormones produced are need to be corrected by supplementation of thyroid correction medicine. Thyroid replacement medication is required in case of hypothyroidism and controlling medication for hyperthyroidism. The surgery and/or oral iodine supplementation is the alternative methods for hyperthyroidism. The regulation of cholesterol biosynthesis,the rate of degradation and its receptors are under the control of thyroid gland secretions. The risk of cardiovascular diseases in hypothyroidism patients is high along with body weight. High blood pressure due to increased lipid profiles (LDL and cholesterol) along with homocysteine and C-reactive protein an inflammatory marker is common in hypothyroidism [20]. This condition can be rectified by normalizing the thyroid gland secretions by medication and other means [21].

Whatsoever, the balanced diet with medication can correct the thyroid gland secretions. A number of food components are detrimental to thyroid disorders and they correct the thyroid functions. The balanced diet with the help of a physician can set right the body condition and thyroid function. It is not at all suggestible to decide the own diet by the patient suffering with thyroid disorders. The key nutrients must be included in the diet are iodine, vitamin D, vitamin B12 and selenium. Iodine deficiency is the primary cause of thyroid dysfunction worldwide. The supplementation of iodine play pivotal role in maintaining thyroid function, since it is a component of thyroid hormones (http://ods.od.nih.gov/factsheets/Iodine-QuickFacts) [22]. Iodine supplementation through iodized salt is the best way and is used in all kinds of food making.Anyhow the intake of iodine has its own limitations. Low or high intake of iodine leads complications like Hashimoto's disease [23]. Since the low levels of vitamin D identified

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in all most all patients holding Hashimoto's and Grave's disease, it is found to be one of the causes of thyroid disorders [24]. The richest source of vitamin D is morning and evening sunlight, besides this milk, dairy products, fatty fish, eggs and mushrooms are the potential dietary sources. The other vitamin showing effect on thyroid dysfunction is B12. Its deficiency was identified in thyroid disorders. The dietary source for vitamin B12 is salmon fish, muscle meat, liver, mollusks and sardines. The other food component, the heavy metal selenium a component of thyroid hormones biosynthetic enzymes play crucial role in thyroid function [25]. It is clear from the studies that selenium rich foods such as tuna fish, lobster, crab and Brazil nuts are increasing the selenium levels and corrects the thyroid dysfunctions [26].

There are certain foods can be avoided by patients of thyroid dysfunction. For example the foods release goitrogens are capable to worst the thyroid disorders. Most popularly the vegetables belong to Cruciferae like cauliflower, cabbage and broccoli release goitin (IMFNB) [27]. Isoflavone rich foods (soya based diet) are also rich with goitrogens. In most of the studies the role of goitrogenic foods in thyroid dysfunction is still not clear. The diet of gluten free grains and millets reduces the thyroid function even with iodine rich foods [28]. Another good method to control thyroid dysfunction is by doing systematic exercise every day with the suggestion of a physician. The over or abnormal exercise may cause side effects.

5. Conclusion

The change in the weight, cardiovascular risk, fatigue, change in mood and upset of gastrointestinal are the inimitable challenges identified during thyroid dysfunction. The methods of treatment and diet can alter the thyroid malformations. The iodine, selenium, vitamin D and B12 rich diets are helpful to reduce thyroid dysfunction along, avoiding goitrogens rich, millets and gluten free foods. Proper exercise is an alternative to maintain a healthy body and its condition. Here, conclude by suggesting for patients of thyroid disorders doing all together may normalize their health. The medication along with iodine, vitamin D, B12 and selenium rich diet with low goitrogens and proper exercise may normalize thyroid gland secretions and helps maintaining better health.

References

[1] Guyton AC, Hall TE (1996) Text Book of Medical Physiology. (9thedn), W.B. Saunders Company, Philadelphia, pp: 945-946.

[2] Dumont J, et al. (2011) Ontogeny, anatomy, metabolism and physiology of the thyroid. Thyroid Disease Manager. https://www.thyroidmanager.org/chapter/ontogenyanatomy-metabolism-and-physiology-of-the-thyroid

[3] Kochupillai N (2000) Clinical Endocrinology in India. Current Science 8: 1061-7.

[4] Zimmerman MB (2009) Iodine deficiency. Endocr Rev30: 376–408.

[5] Wang C, Crapo LM (1997) The epidemiology of thyroid disease and implications for screening. EndocrinolMetabClin North Am 26(1): 189-218.

[6] Elaine N, Maieb R (1990)The Endocrine System: Human Anatomy and Physiology. (3rdedn), W.B. Saunders Company, Cambridge, UK, pp: 546-583.

[7] Bucci I, Giuliani C, Napolitano G (2017) Thyroid-Stimulating Hormone Receptor Antibodies in Pregnancy. Clinical Relevance. Front Endocrinol (Lausanne) 8: 137.

[8] Kandi S, Rao P (2012) Anti-thyroid peroxidase antibodies: its effect on thyroid gland and breast tissue. Ann Trop Med Public Health 5: 1-2.

[9] Kim ES, Lim DJ, Baek KH, Lee JM, Kim MK, Kwon HS, Song KH, Kang MI, Cha BY, Lee KW, Son HY (2010) Thyroglobulin antibody is associated with increased cancer risk in thyroid nodules. Thyroid 20(8): 885-91.

[10] UshaMenon V, Sundaram KR, Unnikrishnan AG, Jayakumar RV, Nair V, Kumar H (2009) High prevalence of undetected thyroid disorders in an iodine sufficient adult south Indian population. J Indian Med Assoc 107: 72-7.

[11] Vanderpump MPJ, Tunbridge WMG, French JM, et al. (1995) The incidence of thyroid disorders in the community: a twenty-year follow-up of the Whickham survey. ClinEndocrinol (Oxf)43:55–69.

[12] Tunbridge WMG, Evered DC, Hall R, et al. (1977) The spectrum of thyroid disease in the community: the Whickham survey. ClinEndocrinol (Oxf) 7: 481–93.

[13] Konno N, Yuri K, Taguchi H, Miura K, Taguchi S, et al. (1993) Screening for thyroid diseases in an iodine sufficient area with sensitive thyrotrophin assays, and serum thyroid autoantibody and urinary iodide determinations. ClinEndocrinol 38: 273–281.

[14] Berglund J, Ericsson UB, Hallengren B (1996). Increased incidence of thyrotoxicosis in Malmo during the years 1988–1990 as compared to the years 1970–1974. J Intern Med 239: 57–62.

[15] Knudsen N, BuElow I, Jurgensen T, Laurberg P, Ovesen L, et al. (2000) Comparative study of thyroid function and types of thyroid dysfunction in two ar-

  DOI: https://doi.org/10.30564/jer.v1i2.1241

eas in Denmark with slightly different iodine status. Eur J Endocrinol 143: 485–491.

[16] Bjoro T, Holmen J, Kruger O, Midthjell K, Hunstad K, et al. (2000). Prevalence of thyroid disease, thyroid dysfunction and thyroid peroxidase antibodies in a large, unselected population. The Health Study of Nord-Trondelag (HUNT). Eur J Endocrinol 143: 639–647.

[17] Nystrom HF, Jansson S, Berg G (2013) Incidence rate and clinical features of hyperthyroidism in a long-term iodine sufficient area of Sweden (Gothenburg) 2003–2005. ClinEndocrinol 78: 768–776.

[18] Korevaar TIM, Medici M, Visser TJ, Peeters RP (2017) Thyroid disease in pregnancy: new insights in diagnosis and clinical management. Nat Rev Endocrinol 13: 610–622.

[19] Medici M, Korevaar TI, Visser WE, Visser TJ, Peeters RP (2015) Thyroid function in pregnancy: what is normal? ClinChem 61: 704–713.

[20] Biondi B, Klein I (2004) Hypothyroidism as a risk factor for cardiovascular disease. Endocrine 24(1): 1-13.

[21] Duntas LH, BrentaG(2012) The effect of thyroid disorders on lipid levels and metabolism. Med Clin North Am 96(2): 269-281.

[22] Dietary supplement fact sheet iodine (2011) Office of Dietary Supplements website. http://ods.od.nih.gov/factsheets/Iodine-QuickFacts.

[23] Dean S (2008) Medical nutrition therapy for thyroid and related disorders. In: Mahan KL, Escott-Stump S, eds. Krause's Food, Nutrition, & Diet Therapy. (13th edn). Philadelphia, PA: Saunders, 711-724.

[24] Jyotsna VP, Sahoo A, Ksh SA, Sreenivas V, Gupta N (2012) Bone mineral density in patients of Graves disease pre- & post-treatment in a predominantly vitamin D deficient population. Indian J Med Res 135(1): 36-41.

[25] Rayman MP (2012) Selenium and human health. Lancet 379(9822): 1256-1268.

[26] Toulis KA, Anastasilakis AD, Tzellos TG, Goulis DG, Kouvelas D (2010) Selenium supplementation in the treatment of Hashimoto's thyroiditis: a systematic review and a meta-analysis. Thyroid. 1163-1173.

[27] Institute of Medicine Food and Nutrition Board. Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington, DC: National Academies Press; (2001).

[28] Elnour A, Hambraeus L, Eltom M, Dramaix M, Bourdoux P (2000) Endemic goiter with iodine sufficiency: a possible role for the consumption of pearl millet in the etiology of endemic goiter. Am J ClinNutr 71(1): 59-66.

 DOI: https://doi.org/10.30564/jer.v1i2.1241