

Review Article

Different Treatment Approaches Used in Autoimmune Thyroid Disease: A Comprehensive Review

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Abstract

Autoimmune thyroid disease triggers because of T cells attack in immune system (in thyroid gland). AITD mainly comprises of two types, 'Hashimoto's thyroiditis' (hypothyroidism) and 'Grave's Disease' (thyrotoxicosis). India have '42 million' people groups experiencing thyroid disease, likewise it has perceived that in contrast with men 80% women's affect more from immune system thyroid disease. The attentiveness of autoimmune hyperthyroidism accounts from 3.0% to 7.5%, hypothyroidism accounts from 0.9%. Genetic Susceptibility, Sex Steroids, Stress, Pregnancy, Radiation Exposure, 'Thyroid Peroxidase Antibodies'(TPO), 'Thyroglobulin Antibodies' (TG), B&T- Cell Responses exist as prime reasons of AITD. 'Cytokine interferon-gamma' also shows dynamic part in origin of HD disease. Radionuclide scan, physical inspection, fine needle aspiration, lymphocytic infiltrate, TPO along with TG detection (by RIA) is significant diagnosis parameter for 'AITD'. Levothyroxin treatment intended for HT and anti thyroid drugs are meant for GD. Observing of regular dosing is precarious for both therapies. Side effects of hormonal therapy could be avoided by using herbal approach (*Bacopa monnieri*, *Melissa officinalis*, *Withaniasomnifera*, *Embllica officinalis*) to improve thyroid dysfunction. There is also a possibility for novel clinical investigation of the plants to further verify their effectiveness in thyroid dysfunction.

Abbreviations

AITD: Autoimmune Thyroid Disease; HT: Hashimoto's Thyroiditis; GD: Grave's Disease; TPO: Thyroid Peroxidase; TG: Thyroglobuline; Ab: Antibody

Thyroid Hormone

After bound to high-affinity thyroid receptors, 'Triiodothyronine' (T_3) naturally controls 'gene expression' with recognizing specific response in T_3 -target genes. In reaction to hormones it prevents or activates transcription [1]. Thyroid Hormone (T_3) is pivotal for endochondral and intramembranous growth of bones. Linear development and Bone mass maintenance are significantly done by this hormone. Deficiency of T_3 hormone in Childs causes growth deficiency, impaired skeletal development. Excess quantity of T_3 boost formation of bone and enhances the growth. In adult thyrotoxicosis, amended bone remodeling triggered by a difference between resorption and bone formation, effects in remaining bone damage and improved risk for postoporotic fracture [2].

Autoimmune Thyroid Disease

Autoimmune diseases arise when immune system negatively activated in body, continued attacking healthy cells. About 80% women affected more by 'autoimmune diseases' instead of men. The particular cause is not known but some recent researches show that variation in certain genes in women's could make this huge difference [3].

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Autoimmune thyroid disease is a well known class in 'autoimmune diseases'. It also have high occurrence in women than men.

Leading types

- Hashimoto's autoimmune thyroiditis-It exists in a form of primary myxedema. 'Cytotoxic T cell' surface molecule undoubtedly has important part in AIT. Consumption of high iodine and Smoking both causes increased incidences of AIT.
- Graves' disease autoimmune thyroiditis.
- Rarer autoimmune thyroiditis is mute and postpartum thyroiditis and the iatrogenic thyroiditis.
- Immunological processes triggered by Thyroperoxidase (TPO) antibody, showing antibody status. Increased 'TPO' and 'Thyroglobulin antibodies' occurs with increasing age. But existence of TPO antibodies is higher than 'TG antibodies' in every age group. Significantly Hypothyroidism accompanied with TPO antibodies, not with TG antibodies. Hypothyroidism individuals found negative for anti-TG antibodies [4].

TH have significant part in skeletal improvement, it manages bone mass and additionally direct linear development. Short height, growth disturbances, epiphyseal dysgenesis and lower bone age are portrayin Childhood hypothyroidism. Advanced bone age, augmented growth, cranial sutures, growth plate and premature closure (in severe cases) are portraying in Child thyrotoxicosis [5].

Brief Account of Autoimmunity

A 46 year prior, the original evidence of autoimmunity of thyroid was first discovered in a rabbits immunized with thyroglobulin, after that description of antibodies and thyroglobulins in Hashimoto's thyroiditis patient's serum have distinguished. Also the anus mirabilis and the abnormal thyroid stimulator cause Graves' disease. The affiliations of environmental, genetic, endogenous factors are needed in the right concentration to initiate autoimmunity for thyroid [6].

Pathogenesis of AITD (Figure 1)

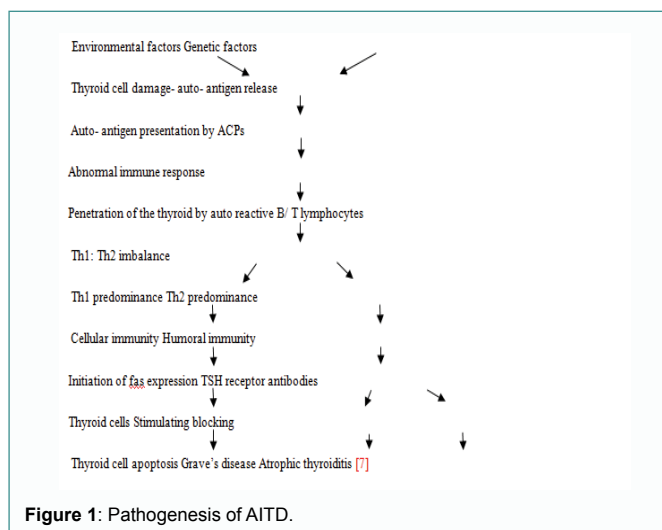
A hypothesis came that the disbalance between helper and suppressor T-lymphocytes is occurred by decreased suppressor T-lymphocyte function which cause autoimmune thyroid disease's probable pathogenetic mechanism [8].

Prevalence of AITD

- In worldwide the prevalence of 'AITD' in women is 2% to 4%, in men is 1%. With growing age its rate always increases.
- Frequent rate is approximately 0.8/100 in 'autoimmune hypothyroidism' and 95% of them are women [7].

The prevalence of hypothyroidism, among adult female ranges from 3.0% to 7.5%. It is more regularly in aged females. It is additionally discovered that after the age of 60 a higher TSH in 11.6% of 683 women present, and unevenly half of the women holds confirmation of thyroid autoimmunity. Hypothyroidism is more predominant in Iceland (high iodine intake areas), having 18% in elderly women.

The frequency of hyperthyroidism in the ordinary female population have range from 0.9%. It is assessed that 79% of the reasons to develop AITD can be credited to genetics, hormonal and environmental risk factors that are involved [9].



Signs and Symptoms

Hyperthyroidism

Signs: Fine hair, Muscle weakness, thin skin, Low cholesterol, Tachycardia, Glucose intolerance, Tremor, Widened pulse pressure, reflexes Stare, rapid deep tendon, Lid lag.

Symptoms: Weight Loss, Menstrual irregularities, Fatigue, Increased Sweating, Heat intolerance, Nervousness, Hyper defecation, Restlessness.

Hypothyroidism

Signs: Depression, Slow reflex, Constipation, Cold, Hair Loss, skin Lethargy, intolerance, Weight gain, Easy fatigue.

Symptoms: Bradycardia, Relaxation, Myxedema, High cholesterol, Dry course, deep hoarse voice, Growth retardation [10].

Triggering aspects for grave's disease

Genetic susceptibility: There are particular epidemiologic

confirmations demonstrating hereditary of Grave's hyperthyroidism and chronic autoimmune thyroiditis. The diseases associate in families and females are inclined for this disease [11].

Stress: Suppression in immunity could prompted by stress, perhaps mediated by movement of cortisol on the immunity cells. Improved immunologic hyperactivity can connected with Suppression of stress. This response could impulsive in 'autoimmune thyroid disease' with genetically prone subjects [12].

Infection: Treatment with Hepatitis C infection by therapy of interferons is well-known factor [13].

Smoking: Risk feature for Grave's hyperthyroidism and moreover a stronger risk feature for Grave's ophthalmopathy.

Sex steroids: Adequate quantities of estrogen improves he immunological reactivity regarding self- antigens, this has proven with many evidences.

Drugs: In prone individuals, drugs like amiodarone, iodine, iodine having drugs takes part in reoccurrence or origination of 'Grave's disease'. They could release thyroid antigens from immune system with directly destruct thyroid cells [14].

Pregnancy: Hyperthyroidism connects with moderate fertility. So, there are few chances to occur Grave's disease throughout pregnancy time. Moreover, immune system suppresses (both B-cell and T-cell functions are lessened) in pregnancy so disease's complications improves as pregnancy progresses. But the progress of 'postpartum thyroid disease' may associated by immunosuppression. The presence of fetal cells in 'maternal tissues' (fetal microchimerism) might play role in postpartum autoimmune thyroiditis development [15].

Triggering aspects for hashimoto's thyroiditis

Genetic susceptibility: Hashimoto's Thyroiditis have family clustering, sometimes alone either with Graves' disease [16]. The sibling reappearance risk is approximately >20.

Stress: Numerous stress inter related to Hashimoto's thyroiditis. The mechanisms consist of immune suppression initiation by non antigen-specific mechanism, because of the properties of cortisol or corticotropin releasing hormone on immune cells, followed by immune hyperactivity prominent to 'autoimmune thyroid disease'.

Sex steroids and pregnancy: Women's are prone for Hashimoto's thyroiditis in comparison to men, it shows sex steroids character. Other than that, older women's are more susceptible for Hashimoto's thyroiditis than younger. Throughout pregnancy, both B and T cells have diminished functions, as the appropriate elevation in CD4+, CD25+ and regulatory T cells. These immunosuppressions have contribution in the 'postpartum thyroiditis' development. In later years, the conventional Hashimoto's disease progress in nearly 20% patients with having postpartum thyroiditis [17].

Iodine intake: Prevalence of Hashimoto's disease (Hypothyroidism) starts with slight iodine insufficiency, while higher incidence inter connected with extreme intake of iodine.

Radiation exposure: children's exposure in the fate of tragic Chernobyl nuclear has established thyroid auto- antibodies of higher frequency. Many evidences are there, which recommend that, risk for elevation in thyroid dysfunction increases positive thyroid antibodies. In comparison to females subjects without radiation exposure, radiation exposed females have 'autoimmune thyroid disease'

(anti-TPO antibodies level >200 IU/mL and hypoechogenicity on ultrasound). People with <5 year exposure to ionization radiation are at high risk [18].

Fetal microchimerism: 'Auto immune thyroid disease' individuals have maternal thyroid glands which identifies the existence of fetal cells. With relation of thyroid gland these cells may introduce host versus graft reactions and have critical role in the introduction of 'Hashimoto's thyroiditis'.

Auto antibodies: TPO Ab's detectable level typically shows the progress of a higher TSH and consequently show risk feature for hypothyroidism.

Thyroid peroxidase (TPO) antibodies: The crucial thyroid enzyme 'Thyroid Peroxidase' (TPO) anti- bodies catalyze fabrication of 'thyroid hormone' (coupling and iodination reaction). Patients of 'Grave's disease' and 'autoimmune hypothyroidism', 90% of 'Anti-TPO auto antibodies' are found. In 'autoimmune hypothyroidism', 'Thyroglobulin (TG) antibodies' gathered with predominant antibodies. Anti-TPO antibodies are primarily from IgG4 sub classes IgG class I and in excess.

Thyroglobulin (TG) antibodies: When thyroid cells get stimulated by TSH, TG is hydrolyzed and endocytosed in lysosome then release T3 and T4. The particular location is unclear for T- and B-cell epitopes. Less than 60% patients found with lymphocytic thyroiditis and thyroglobulin auto antibodies and 30% patients found with Grave's disease.

Thyroid Stimulating Hormone Receptor (TSH-R) antibodies: 'Atrophic thyroiditis' and 'Grave's disease' instigated by prime autoantigen 'Thyroid Stimulating Hormone Receptor (TSH-R)'; Situated on thyroid follicular cell's basal surface. In Grave's disease, thyroid cell get stimulated by complexing of Thyroid Stimulating Antibodies (TSABs) to receptors, and produce extreme number of thyroid hormones which causes hyperthyroidism. In 'atrophic thyroiditis', inhibited thyroid cells stimulation occurred by some antibody that binds to the TSH and receptors. Classes of TRAb-

- 'Thyroid Stimulating Auto Antibodies' (TSAb) cause Grave's hyperthyroidism
- 'Thyroid Stimulation-Blocking Antibodies' (TBAb) block receptor complex with TSH

Mechanism of thyroid cell injury: In AITD several antibodies and cell-facilitated mechanisms are accountable for thyroid injury. Compare to normal counterparts, the expressions of CD 95 Lin (death receptor) and CD 95 (death receptor) ligands are higher thyroid tissues in 'Hashimoto's thyroiditis'. Here highly increased manifestation in Grave's disease of 'modulators of apoptosis' (Bcl-2, cFLIP and Bcl-XL). Role of inhibitory apoptosis mechanism supports by this. Even though, there is major expression of CD95/Fas and its ligand in all cases, but Thyrocytes go through apoptosis individual in Hashimoto's thyroiditis.

By enhancing caspases expression the 'cytokine interferon-gamma' likely to have important role in pathology of TH1 disease (Hashimoto's thyroiditis), although FAS facilitated apoptosis alert the cells.

B-Cell responses: Individual's with 'Hashimoto's thyroiditis' and 'primary myxedema', antibodies like 'TG' and 'TPO' present in higher concentration. In 'Grave's disease', these antibodies are frequent but

not common. In 'postpartum thyroiditis', 'TPO antibody' is more recurrent than TG antibodies. For entering in antigen and become pathogenic, 'TPO antibodies' required cell-mediated injuries.

T-Cell responses: Within thyroid lymphocytic infiltrate, mostly 'CD4+' cells occurs in the 'CD4+' and 'CD8+'. Lymphocyte produces interferon gamma, Cytokines including IL-2, IL-4, IL-6, IL-10, IL-12, IL-13 and IL-15. These might be varies in concentration depends on patients. Metabolic characters of thyroid cells injured by complement attack are originated by alternate or classical pathways. This damage shows secrete Prostaglandins, oxygen metabolites, IL-6, IL-1. They all are accountable for enhancing autoimmunity [19,20].

Other antibodies Na+/I- symporter: NIS is first verified cultured dog thyroid cells, it is a chief thyroid autoantigen. Antibodies which disturb iodide uptake mediated by NIS; presents 15% in sera of Hashimoto's, 1/3rd in sera of 'Grave's disease'. 'AITD' patients contain 10% to 25 % antibodies for thyroid hormones. Although, some patients also have tubulin, cytoskeletal proteins and some auto antibodies (non-specific) against DNA [9,21].

Diagnosis of Autoimmune Thyroid Disease

Measurement of circulating antibodies for 'TPO' and 'TG' can detect AITD. Around 98% of patients detect positive for both antibodies. For detecting autoimmune hypothyroidism in comparison to TG Ab, 'TPO Ab' is more sensitive and precise. In long-lasting 'Hashimoto's thyroiditis' diagnosis, increased level of TSH with 'TPO antibodies' are best precise standard.

Autoimmune thyroid disease and neoplasms

1/3rd of patients which suffers from thyroid cancer have also established 'thyroid antibodies' and Thyroiditis. For initiation of non-Hodgkin's thyroid lymphoma, Hashimoto's disease (pre-existing) is key risk factor. Several studies also show that 'breast cancer' could also originate by enhanced regularity of autoimmune thyroiditis [10].

Autoimmune thyroid disease in kidney disease

Thyroid dysfunction shows major variations in tubular and glomerular functions and also in electrolyte and water homeostasis. From clinical practice observation point, it is noticeable that both diseases followed by remarkable alterations in water and electrolyte metabolism, beside cardio vascular function (Table 1).

Important test method for thyroid hormone detection

'Chemiluminescence' method used for identification of 'Thyroid Auto antibodies' (Thyroid Peroxidase and Thyroglobulin Ab), T3, T4,

Table 1: Concentration of different thyroid hormones [20].

Thyroid Hormones	Abbreviations	Concentration
'Serum Thyrox' level	'T4'	4.6-12 ug/dl
'Serum Triiodothyronine' level	'T3'	~80-180 ng/dl
'Serum thyroglobulin' level	'Tg'	~0-30 ng/m
'Thyroglobulin antibody level	(TgAb)	>20 IU/mL.
'Thyroid peroxidase antibody' level	(TPOAb)	>35 IU/mL.
Free 'Thyroxine' level	(FT4)	0.7-1.9 ng/dl
'TRH stimulation test Peak' level	(TSH)	9-30 IU/ml in 20 min to 30 min
Free 'Thyroxine index' level	(FT4I)	~4-11
'Thyroid hormone binding' ratio	(THBR)	Up to 0.9-1.1
Serum 'thyrotropin' level	(TSH)	~0.5-6 uU/ml
Free 'T ₃ Index' level	(FT3I)	<80-180
'Radioactive iodine' uptake level	(RAIU)	~10-30%
'Thyroxine-binding globulin' level	(TBG)	~12-20 ug/dl T4+1.8 ugm

TSH & FT3, FT4, TSH3G UL and 'RIA' is used for detection of TSH Receptors Ab [22].

Study Data of Autoimmune Thyroidism

In UK genetic preference analyzing study was organized for 'autoimmune thyroid disorders'. For 'Grave's disease', patients group comprises 2791 Caucasian (white) volunteers (2717 female/474 male) and for 'Hashimoto's thyroiditis' 495 Caucasian (white) volunteers with (427 females/ 68 males) have chosen, from the diverse 'specialist referral center and thyroid clinic's in Bournemouth, Birmingham, Cardiff, Cambridge, Leeds, Exeter, New castle, United Kingdom (UK) and Sheffield they all were recruited.

Presence of biochemical hyperthyroidism indicate some signs of 'Grave's disease'

- Radionuclide scan illustrate distributed uptake/ Ultra sound scan diffuse goiter.
- Grave's ophthalmopathy (NOSPESC score ≥ 2)
- TSH receptor's positive auto antibodies, positive antibodies for TG and TPO.
- On physical inspection diffuse goiter shows.
- After thyroid histology lymphocytic infiltration confirmed.

Presence of biochemical hypothyroidism demonstrate some signs of 'Hashimoto's disease'

- Positive TG and TPO antibody.
- On physical inspection diffuse goiter present.
- In fine needle aspirate lymphocytic infiltrate present.

In 'grave's disease', there are 9.77% cases of further autoimmune disorders and in 'Hashimoto's thyroiditis' and 14.3% cases of further immune disorders. In 'Grave's disease' 3.15% patients (more possibilities in male) have rheumatoid arthritis, 4.25% of patients acquire rheumatoid arthritis associated with 'Hashimoto's disease'. Therefore, this is most common simultaneous autoimmune disorder in both cases. In 'Grave's' and 'Hashimoto's thyroiditis' here are threats of almost all autoimmune diseases as like type1 diabetes, Addison's disease, systemic lupus erythematosus, pernicious anemia, vertigo, celiac disease and multiple sclerosis probably marked increasing %. There was virtual assembling of parental hypothyroidism with 'Grave's disease' and 'Hashimoto's thyroiditis' [23].

Autoimmune thyroiditis prevalence in India

In India thyroid is very frequent, affecting ~42 million peoples and increasing considerably. In India study shows thyroid diseases affecting commonly are-Hyperthyroidism, Hypothyroidism, Goiter & Iodine insufficiency disorder, Thyroid cancer and Hashimoto's Thyroiditis.

A population built study revealed that % of adult volunteers affected subject with 'anti-thyroid peroxidase antibodies' are 16.7 %, for 'anti-thyroglobulin antibodies' are 12%. The occurrence of 'anti-TPO' and 'anti-TG antibodies' are 9.5%, 8.5 %, if patients with irregular thyroid function were omitted.

In India, for Hashimoto's thyroiditis revolutionary study, 6283 schoolgirls were selected. Between them, they found Goiter in 1810 school girls. 'Juvenile autoimmune thyroiditis' constitute in 58 schoolgirls (7.5%), in between 764 fine needle aspirate cytology

definite girls. Subclinical hypothyroidism (15%) and evident hypothyroidism (6.5%) also indicated from confirmed instance of fine needle aspiration cytology [24].

Treatment of Diseases

Allopathic treatment

Hashimoto's Thyroiditis:

Synthetic hormones: 'Thyroid hormone' deficiency would accelerate 'Hashimoto's disease'; it needed 'thyroid hormone' replacement therapy. Normally this replacement occurred by regular oral intake of thyroid's hormone (synthetic) levothyroxine (Synthroid, Levoxy, thyronom etc). This hormone is naturally formed by thyroid gland. As per 'American Thyroid Association', this treatment may give rise to little weight reduction causing fat loss and muscle protein, (>10% of body weight).

This medication helps to restores sufficient concentration of hormone and all the hypothyroidism symptoms get reversed through this medicine. Although, levothyroxine medication is commonly for lifetime but dosage amount always wary according 'TSH' level in blood (every 12 months) with proper monitoring and guidance of doctor every year [5,25].

Grave's disease:

Anti-thyroid medicines: These drugs prevent thyroid gland by synthesizing more hormones. Ex- Propylthiouracil and Methimazole (Tapazole).

Radioactive iodine: Above 60 years for hypothyroidism, its oral consumption is beneficial. Thyroid gland absorbs radio activated iodine, got shrinked.

Surgery: Sometimes, Thyroidectomy is suggested for the treatment of 'Grave's disease'.

Beta blockers: Suggested for anxiety, sweating increased heart rates in 'Grave's disease' [26].

Observation of the dosage: In 'Hashimoto's thyroiditis', TSH in blood always be checked monthly later treatment started, for initially regulating the correct dosage of externally provided thyroid hormone. Doctor starts treating with initial dosage of medication and thereby steadily raises dosage (Skipping of dose will cause returning of symptoms). Excess amount of 'thyroid hormone' can regulate worse osteoporosis (bone loss) and also induce arrhythmias. Increasing hormone substitution permits heart to control the increased metabolism. Satisfactorily if used in appropriate dose, Levothyroxine drug has certainly not any side effects; also it is inexpensive [25]. In 'Grave's disease' ophthalmopathy needed as additional medications like steroid, unusual eye droplets to decrease symptoms [26].

Ayurvedic treatment

In Ayurveda, the balanced state of Agni (enzymatic activity), dhatu (metal), tridoshas (bodily humors) and mala (impurity) are the base so that body can achieve samavastha (homeostasis) [26,27]. Herbal plant extracts and some formulations are used to balance bodily humors and tridoshas [27].

Herbal formulations

- Tab. Thycet 1 bd
- Tab. punarnavamandur 1 bd

- Tab. kanchnarguggul 1 bd
- Tab. Chandraprabhavati 1 bd
- Tab. Bramhivati 1 bd Cap.optilife 1 bd
- HanspatyadiKashay 3 - 4 tsp bd
- Ashwagandharishta- 3 - 4 tsp with water.
- VarunadiKashay 3 - 4 tsp bd [28]

Herbs

Hashimoto's thyroiditis

- *Bacopa monnieri* (Scrophulariaceae) commonly known as 'Brahmi' whole plant raised both T3 and T4, reduce oxidative stress, improves memory, concentration (200 mg/kg) [27,29,30]
- *Cucumis melo* (Cucurbitaceae) 'Musk melon' fruit peel raised thyroid hormone levels [27].
- *Withaniasomnifera* (Solanaceae) 'Ashwagandha' root lowered cortisol, raise thyroid hormones levels, lower oxidative stress [27,29,30].
- *Commiphoramukul* (Burseraceae), thyroid stimulatory function [29,30,31].
- *Mangiferaindica* (Anacardiaceae), Showed thyroid stimulatory and anti-peroxidase roles [29,33].
- *Bauhiniapurpurea*L. (Caesalpiniaceae), thyroid hormone regulating act [29].
- *Costuspictus*D. Don, a rhizomatous extract has been explored for anti-hypothyroid efficacy and could significantly restore the normal level of thyroid hormones [30].
- *Fucusvesiculosus*var. *divaricatus* (Fucaceae) marine alga rich in iodine [29,32,33].

Grave's disease

1. *Melissa officinalis* L. (Lamiaceae), lemon balm hinders the TSH binding to the receptor by binding to the receptor. It also constrains formation of cyclic AMP stimulated by TSH receptor antibodies [29,32].
2. *Leonuruscardiaca* L. (Lamiaceae) It reduces the inflammation and also inhibits the enzyme 5-deiodanase [29,34].
3. *Convolvulus pluricaulis* (Convolvulaceae) It powerfully show action on liver enzymes which helps in enhancing the indications of hyperthyroidism [29,33].
4. *Rauwolfiaserpentina*L. (Apocynaceae) The root extract helps in reduction of the serum T3 and T4 concentrations [29,33].
5. *Embllica officinalis* Gaertn. (Phyllanthaceae) Its fruit extract reduces T3, T4 levels in serum. The decreased level of T3 was by obstructing peripheral conversion of T4 to T3 in extra-thyroid tissues [29,34,35].
6. *Moringa oleifera* auct. nonlam Family: Moringaceae [30] leaf extract reduces serum T3 concentration and increase serum T4 concentration, also the plant extract inhibit the peripheral change of T4 to T3 [29].

7. *Ocimum sanctum* L. (Lamiaceae) The leaf extract significantly inhibits only T4 concentration [29,36].

Conclusion

This review focused on types, symptoms, prevalence, affecting factors and cure of AITDs. We saw the incidence of AITDs increasing readily year by year. It is desirable for the treatment, that they get identified early. The symptoms which shows in patients suffering 'Grave's disease' and 'Hashimoto's disease' are identically general but not really easy to discriminate from other 'autoimmune diseases' and simple 'Thyroid disease'. So, the awareness for 'Autoimmune Diseases' must spread around. This has also realized that probably increased 'TPO' levels (thyroid peroxidase), TG (Thyroglobulin) and T& B cell are key source of 'AITD' followed by interaction of genetic or environmental factors. One thing also we perceived that, imbalance between helper and suppressor T-lymphocytes instigated by decreased suppressor T-lymphocyte function, could leads to 'Autoimmune Thyroid Diseases'. In allopathic approach consistent oral intake of thyroid's hormone (levothyroxine) for Hashimoto's Thyroiditis and Propylthiouracil, Methimazole Radioactive iodine and Surgery for Grave's Disease is used. The herbal approach to thyroid dysfunction it is necessary to avoid side effects of hormonal therapy. Some herbs like *Bacopa monnieri*, *Withaniasomnifera*, *Cucumis melo*, *Melissa officinalis*, *Rauwolfia serpentina*, *Embllica officinalis* are efficacious in normalizing thyroid dysfunction. This will provide much more options to treat thyroid dysfunction. Long-term effectiveness studies of the substances are needed to perform to optimize the length of treatment.

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