EUROPEAN JOURNAL OF BIOMEDICAL AND PHARMACEUTICAL SCIENCES
http://www.ejbps.com

ISSN 2349-8870
Volume: 4
Issue: 11
271-277
Year: 2017

AN OBSERVATIONAL STUDY ON THE EFFECT OF THYROID DISORDERS ON VARIOUS ORGAN SYSTEMS

D.Geetha Rani[1], R.Deepthi[1]*, Dr.P.Kishore[1], Dr.B.Sridhar[2]

[1]Department of Pharmacy Practice, Care College of Pharmacy, Oglapur(V), Damera(M), Warangal(Rural)-506006.
[2]Endocrinologist, Samraksha Superspeciality Hospital, Hanumakonda.

***Corresponding Author: R. Deepthi**
Department of Pharmacy Practice, Care College of Pharmacy, Oglapur(V), Damera(M), Warangal(Rural)-506006.

Article Received on 01/09/2017 **Article Revised on 22/09/2017** **Article Accepted on 12/10/2017**

ABSTRACT
Thyroid diseases are the most common endocrine disorders world-wide. India is no exception. India is in the transition phase from iodine deficiency to iodine sufficiency, and this is expected to change the thyroid status of the population. Thyroid disorders for the most part are controllable. However, uncontrolled thyroid diseases can produce serious effects on other parts of the body. The present study was aimed to elucidate the effect of thyroid disorders on various organ systems of the body. It is an symptom based observational study performed for 6months period in randomly selected 204 subjects among whom, the majority of the patients were suffering with hypothyroidism (77.9%) compared to hyperthyroidism (22.05%). Majorly effected organ system is reproductive system (39.09%) followed by Digestive system (21.3%), CNS (13.18%), Haemopoietic system (10.9%), Hepatic system (6.81%), CVS (6.36%), Skeletal system(2.27%). Effect of thyroid disorders on reproductive system (P=0.0109**), central nervous system (P=0.0005**), cardiovascular system (P=0.0001**), hepatic system (P=0.0426**) was found to be statistically significant. Diagnosing the thyroid disorders in early stages can minimize their deletorious effects on various organ systems of the body. Therefore it is very important for a clinical pharmacist to educate the patient regarding the importance of symptoms awareness and medication adherence to the prescribed regimen in thyroid disorders which will go a long way in preventing reproductive, cardiovascular and central nervous system complications in future.

KEYWORDS: *Thyroid disorders, Thyroid effect, CVS, CNS, Medication adherence.*

INTRODUCTION
The thyroid gland is situated low down at the front of neck weighing about 25g.[16] The gland has two lobes, each pear shaped hugging anterolateral aspects of cervical trachea from oblique line of thyroid cartilage to 5th or 6th tracheal ring. The principal hormones secreted by the thyroid are thyroxine (T_4) and triiodothyronine (T_3). Thyroid hormones enter cells and T_3 binds to thyroid receptors in the nucleus. T_4 can also bind, but not as avidly. The hormone-receptor complex then binds to DNA via Zinc fingers and alters the expression of a variety of different genes that code for enzymes which regulate cell function.[7]

Table 1: Physiologic effects of thyroid hormones[7]

Target tissue	Effect	Mechanism
Heart	Chronotropic, Inotropic	Increase number of beta-adrenergic receptors. Enhance response to circulating catecholamines. Increase proportion of a myosin heavy chain
Adipose tissue	Catabolic	Stimulate lipolysis
Muscle	Catabolic	Increase protein breakdown
Bone	Developmental	Promote normal growth and skeletal development
Nervous system	Developmental	Promote normal brain development
Gut	Metabolic	Increase rate of carbohydrate absorption
Lipoprotein	Metabolic	Stimulate formation of LDL receptors
Other	Calorigenic	Stimulate oxygen consumption by metabolically active tissues (except testes, uterus, lymphnodes, spleen, anterior pituitary). Increase metabolic rate.

Thyroid disorder is a worldwide public health problem. It has taken an epidemic form, only after diabetes mellitus. It is a spectrum of disorders which manifests either as hyperthyroidism or hypothyroidism. Based on thyroid hormone levels and presence or absence of symptoms they may again be sub classified as overt or subclinical.[5]

Table 2: Vivid conditions of thyroidism with their respective TSH levels.

Condition	TSH	Thyroid hormones
Overt hyperthyroidism	<0.1 mIU/L	Elevated T4 or T3
Overt hypothyroidism	>4.5mIU/L	Low T4
Subclinical hyperthyroidism	TSH<0.1 mIU/L	Normal T4 and T3
	<0.1mIU/L<=TSH<0.4mIU/L	Normal T4 and T3
Subclinical hypothyroidism	4.5mIU/L<TSH=10mIU/L	Normal T4
	TSH>=10mIU/L	Normal T4

An overall estimated 42 million Indians are suffering from thyroid disorders.[8] Thyroid hormone is responsible for the regulating the metabolic rate of every cell in the body Since every cell in the body depends on its metabolism to perform its function, when the thyroid system is malfunctioning it can affect every system of the body. Seven main organ systems which are affected by thyroid disorders are Reproductive System, Digestive system, Central Nervous system, Cardiovascular System, Hepatic system, Haemopoietic System and Skeletal system.

Hypothyroidism beginning before puberty causes a delay in onset of puberty followed by an ovulatory cycle in women.[20] In women, hypothyroidism is associated with delay in the onset of puberty, anovulation, amenorrhea, polymenorrhea, menstrual irregularities, infertility and increased frequency of spontaneous abortions. It was suggested that these alterations may be caused by decrease in gonadotropin secretion, due to hyper prolactinemia. Similar to hypothyroidism, hyperthyroidism may also result in menstrual abnormalities in adult women. The more common manifestations are polymenorrhoea and oligomenorrhoea. Moreover hyperthyroidism in women has been linked to reduced fertility. Reported studies indicate that menstrual disturbances in hyperthyroidism are two times more frequent than in normal population.[20]

There have been reports of disorders of motility and transport functions in the digestive system resulting from hypothyroidism.[12] A reduction in the motor activity of stomach, small intestine and colon has been reported in previous studies. Delayed intestinal transit time has been reported for hypothyroid patients, although normal gastric emptying time and normal intestinal transit time have also been reported for sufferers of thyroid disorders.[12] Digestive symptoms or signs may also reveal clues to thyroid disease and when ignored or underestimated, diagnosis may be delayed and serious consequences may occur.[15]

Disorders of the thyroid gland are frequently associated with severe mental disturbances. This intimate association between the thyroid system and behaviour has been the impetus for exploring the effects of thyroid hormones in modulating affective illness and the role of the hypothalamic-pituitary thyroid (HPT) axis in the pathophysiology of mood disorders.[11] Impaired memory, slowed mental processing, depression, nerve entrapment syndromes, ataxia, muscle weakness and muscle cramps are the most common neurological symptoms which may be caused by hypothyroidism.[9] Disorders of the thyroid gland are among the most common endocrine maladies. Hypothyroidism is the most prevalent form of thyroid disease and symptoms may include memory and learning impairment, depression, psychotic behaviour, retarded locomotor ability, somnolence, progressive intellectual deterioration and in extreme cases, coma.[9] Since the thyroid hormones dramatically affect the maturation of specific neuronal populations, the absence of these hormones during the period of active neurogenesis leads to irreversible mental retardation and is accompanied by multiple morphological alterations in the brain.[17]

Heart is an organ sensitive to the action of thyroid hormone and measurable changes in cardiac performance are detected with small variations in thyroid hormone serum concentrations. Increased or reduced action of thyroid hormone on certain molecular pathways in the heart and vasculature causes relevant cardiovascular derangements. In hyperthyroidism, the preload is increased leading to high cardiac output with increased heart rate, reduced peripheral vascular resistance and hyperdynamic circulation. The reduction in systemic vascular resistance is responsible for the decrease in renal perfusion pressure and for activation of the Renin-Angiotensin-Aldosterone System [RAAS], with the resulting increase in sodium absorption and blood volume. The increased risk of cardiac mortality could be a consequence of the increased risk of arrhythmias, especially Atrial Fibrillation and for the presence of heart failure. In presence of hypothyroidism, changes occur in cardiac structure and function, and is characterized by low cardiac output with decreased heart rate, stroke volume and reduction in systolic and diastolic functions. There is also a decline in cardiac preload and blood volume, as well as drop in renal perfusion with impaired free water clearance and hyponatremia.[2]

Thyroxine and tri-iodothyronine are essential for normal liver growth, development and function. These hormones regulate the basal metabolic rate of hepatocytes and modulate hepatic function. Increased thyroid hormone decreases the concentrations of cholesterol, phospholipids and triglycerides in the plasma, even though it increases the free fatty acids. Conversely, decreased thyroid secretion greatly increases the plasma concentrations of cholesterol, phospholipids and triglycerides. The large increase in circulating plasma cholesterol in prolonged hypothyroidism is often associated with severe atherosclerosis.[14]

Anemia is often the first sign of hypothyroidism. Hypothyroidism can cause a wide variety of anemic disorders. Numerous mechanisms are involved in the pathogenesis of these anaemias that can be microcytic, macrocytic and normocytic. Severity of anemia is associated with the degree of hypothyroidism. The most frequently encountered type of anemia is normochromic normocytic anemia. Reason could be the bone marrow repression due to thyroid hormone deficiency as well as lack of erythropoietin production arising from the reduction in need of O_2. Erythrocyte life cycle in hypothyroidism is normal and there is hypoproliferative erythropoiesis. Thyroid hormones also increase 2-3 DPG (diphosphoglycerate) levels assisting in the transmission of oxygen into the tissues.[6] Thyroid dysfunction could seemingly influence fracture risk by increasing the risk of falling or by weakening the bone strength. Hypothyroidism is a risk factor for falls in elderly individuals[3] and hyperthyroidism has for more than a century been known as a risk factor for osteoporosis.[21] Both overt hypothyroidism and hyperthyroidism have been associated with increased risk of fractures in previous studies. Other studies have reported that fractures are more common in persons with subclinical thyroid dysfunction and some have found fracture incidence to increase by decreasing thyroid-stimulating hormone (TSH) within the reference range.[1]

METHODOLOGY

A Descriptive observational study was done for a period of 6 months in an endocrinology clinic. Institutional research board approval was obtained prior to commencement of the study.

Study population: All the patients visiting clinic were screened for study after obtaining consent from the patients. Patients who are diagnoised with thyroid disorders of all age groups and willing to participate in the study were selected. Pregnant women and cognitive impaired patients, thyroid patients with other co-morbid conditions such as Diabetes mellitus, Hypertension were excluded from the study as they could interfere with our observations. Socio-demographics and relevant clinical data of participants such as age, gender, occupational class whereas clinical data includes symptoms, past history of thyroid disorders and past medication history, Laboratory parameters includes Thyroid function tests, liver function tests, complete blood picture, lipid profile, Serum creatinine, Serum uric acid, urine analysis and radiological results were obtained through direct communication with patients, their care takers and by reviewing patient records and laboratory data.

Statistical Analysis: All the results were analyzed using Graph pad prism software version 7.01 and chi-square test.

RESULTS

During the study period a total of 204 subjects were reviewed. Females are predominant (160) compared to males (44). Of 204 patients, majority of the patients were found to be in between 31-40 years, which constitute 39.21% followed by the age group 21-30 (30.8%), 10-20 (15.2%), 41-50 (10.78%), 51-60 (2.45%) and the least number of patients were in age group of 61-70 (1.47%). In present study majority of the patients are suffering with hypothyroidism than hyperthyroidism.

Reproductive system (39.09%) was found to be majorly effected organ system by thyroid disorders compared to other organ systems such as Digestive system (21.3%), CNS (13.18%), Haemopoietic system (10.9%), Hepatic system (6.81%), CVS (6.36%) and Skeletal system (2.27%).

Reproductive system is majorly affected organ systems due to thyroid disorders and menstrual cycle was highly disturbed. Oligomenorrhagia was mostly found (42%) followed by polymenorrhagia (13%), amenorrhoea (4%). Primary infertility (12.25%) was frequently observed compared to secondary infertility (4%). Abortions (2.94%) were observed.

Digestive system is mostly affected by hypothyroidism compared to hyperthyroidism and thus constipation (10.29%) is more common in hypothyroid patients followed by indigestion (6.86%), abdominal distension (5.39%) and diarrhoea (3.43%) in hyperthyroid patients.

Among CNS, numbness and tingling (6.86%) were observed followed by anxiety (5.38%), insomnia (3.43%) and depression (0.49%). Among CVS, Hyperthyroid Patients presented with symptoms such as palpitations (5.38%) and Tachycardia (3.92%).

In skeletal system malaise (9.80%) is presented by more patients followed by weight gain (6.37%), Fractures (0.98%) and pain in both knee joints (0.98%).

Table 3: Effect of Hypothyroidism and Hyperthyroidism on organ systems.

ORGAN SYSTEM	Hypothyroidism	Hyperthyroidism	RESULTS
Reproductive system	78 (45.34%)	8 (16.6%)	X^2=6.428 D.F.=1 P=0.0109* Odds ratio=0.3675
Digestive system	39 (22.67%)	8 (16.6%)	X^2=0.5376 D.F.=1 P=0.4634 Odds ratio=0.735
Central Nervous system	14 (8.13%)	15 (31.25%)	X^2=12.13 D.F.=1 P=0.0005* Odds ratio=3.839
Cardiovascular system	1 (0.5%)	13 (27.08%)	X^2=34.47 D.F.=1 P=0.0001* Odds ratio=4.658
Hepatic system	15 (8.72%)	0	X2=4.113 D.F.=1 P=0.0426* Odds ratio=0
Haemopoietic system	20 (11.62%)	4 (8.3%)	X2=0.3424 D.F.=1 P=0.5584 Odds ratio=0.7167
Skeletal system	5 (2.9%)	0	X2=1.387 D.F.=1 P=0.2390
Total	172 (100%)	48 (100%)	

*** P<0.05 is considered significant. X2, Chi square; D.F, Degree of freedom; CI, confidence interval**

Effect of thyroid disorders on reproductive system, central nervous system, cardiovascular system, hepatic system was found to be significant. Whereas, effect of thyroid disorders on digestive system, haemopoietic system and skeletal system was found to be insignificant.

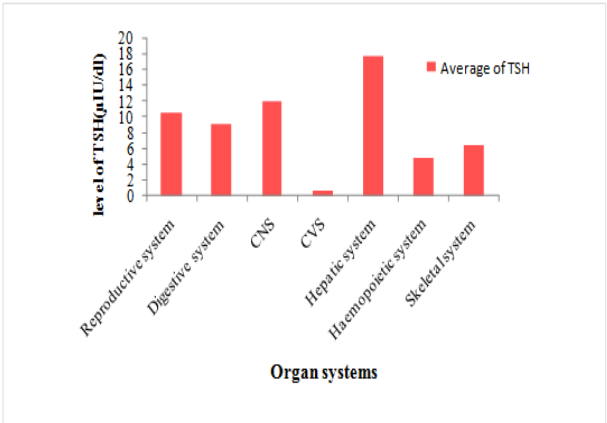

Figure 1: Average of TSH in Thyroid disorders showing its effect on various organ systems.

DISCUSSION

India is in the transition phase from iodine deficiency to iodine sufficiency and this is expected to change the thyroid status of the population. During the study period of 6months a total of 204 patients data was collected and assessed.

In the present study, among 204 patients, females were predominant (78.4%) compared to males(21.6%) similar to study conducted by Nagarkar, et al.[13] As women are more susceptible to different hormonal leaps, they are sensitive to hormonal changes compared to males. American Thyroid Association estimates that more than half of thyroid conditions remain undiagnosed. Women often overlook their symptoms or mistake them for symptoms of other conditions. Women are also at high risk for developing thyroid disorders after childbirth. Symptoms such as fatigue and depression are common during this period, but these are also symptoms of thyroid disease. Among 204 patients, majority were found to be in between 31-40 years, which constitute of 39.21% followed by the age group in between 21-30(30.8%), 10 20(15.2%), 41-50(10.78%), 51-60(2.45%) and the least number of patients are in between age group of 61-70(1.47%). Similar results were found in the study conducted by Nagakar, et.al,[13] out of 128 patients, 44 (34.4%) patients were 31-50 years, 49 (38.3%) patients were 51-70 years.

In the present study majority of the patients were suffering with hypothyroidism (77.9%) compared to

hyperthyroidism (22.05%) which is similar to study conducted by Deepthi Govindan Kutty, et.al.[4] Female gender were found to have significant association with hypothyroidism. Prevalence of hypothyroidism is more compared to hyperthyroidism because the most common cause of hypothyroidism is an autoimmune disease called Hashimoto's disease, a condition where in immune system makes antibodies killing thyroid cells leading to decreased production of TH. Diet with too much or too little iodine can also seriously affect thyroid hormone production. Thyroid gland requires the dietary element iodine to synthesize T_3 and T_4 hormones. Too much iodine can cause hypothyroidism; too little can cause hyperthyroidism. Iodine deficiencies are rare as consuming too much iodine is commonly seen than consuming too little.

In our study effect of thyroid disorders on reproductive system was found to be significant (P=0.0109). Among 204 cases, effect on reproductive system was found to be in 8 (16.6%) and 78 (45.34%) in hyperthyroidism and hypothyroidism patients respectively. Effect was evaluated based on menstrual irregularities, infertility, abortions. In our study among menstrual irregularities the prevalence of Oligomenorrhagia (42) is predominant followed by polymenorrhagia (13), amenorrhoea (4). Similar to study conducted by T.Veeresh, et.al.[20] The connection between thyroid hormone levels and the menstrual cycle is mainly mediated by thyrotropin-releasing hormone (TRH), which has a direct effect on the ovary. Additionally, abnormal thyroid function can alter levels of sex hormone-binding globulin, prolactin, and gonadotropin-releasing hormone, contributing to menstrual dysfunction. For example, increased levels of TRH may raise prolactin levels, contributing to the amenorrhea associated with hypothyroidism. We found that 29 patients had infertility problem and 6 patients had miscarriage. Similarly Hiraoka, et.al,[19] found that of 203 infertile women who first visited infertility treatment division including 13 hypothyroid patients with elevated TSH above 4.5 mIU/l (elevated-TSH patients), 11 of whom were diagnosed as SH(Suclinical Hypothyroidism) and 190 patients with normal TSH (normo-TSH patients). They evaluated the subjects according to reproductive outcome. Multivariate analysis showed significant influence of elevated TSH on clinical pregnancy, although miscarriage and live birth were not affected. In addition, they revealed that the rate of decreased ovarian reserve and unexplained infertility was increased in patients with elevated TSH levels. In our study patients with infertility are reinforced the usefulness of TSH screening and results showed positive to thyroid disorders. This is because severe hypothyroidism is commonly associated with failure of ovulation. Ovulation and conception can occur in mild hypothyroidism. However, these pregnancies are often associated with abortions, stillbirths, or prematurity. SH may be of greater clinical importance in infertile women with "unexplained" infertility, especially thyroid disturbances and female fertility when the luteal phase is inadequate and such patients should be investigated in depth for thyroid dysfunction.

Effect of thyroid disorders on digestive system was found to be insignificant (P=0.4634). Effect on digestive system was found to be in 8 (16.6%) hyperthyroid, 39 (22.67%) hypothyroid patients. Effect was evaluated based on constipation (10.28%), indigestion (6.86%), abdominal discomfort (5.39%) and diarrhea (3.43%). Study conducted by Olga Yaylali, et.al.,[12] on 30 females with primary hypothyroidism and 10 healthy females. All cases underwent esophagogastric endoscopy and found the mean esophageal transit time and gastric emptying time were markedly increased in cases of hypothyroidism and concluded that Hypothyroidism prominently reduces oesophageal and gastric motor activity and can cause gastrointestinal dysfunction.

Hyperthyroidism in the body is linked more heavily in causing diarrhea. The reasons for this diarrhea includes: Fat malabsorption, Intestinal hyper motility, Hyper secretion of bile and increased intestinal transit time leading digestive system to run on high process resulting in wasted food and loose stools. In contrast, constipation is typically found in conjunction with hypothyroidism. This is thought to be due to slower intestinal peristalsis. Hypothyroidism slows the action of the digestive tract causing constipation. This is because hypothyroidism can weaken the contraction of the muscles lining the small and large intestine causing the stools to move too slowly.

In our study, among 204 patients, the effect of thyroid disorders on CNS was found to be in 29 (13.18%). Effect was found to be significant (P=0.0005). Effect on CNS in hyperthyroidism and hypothyroidism was found to be 15 (31.25%) and 14 (8.13%) respectively. Effect was evaluated based on numbness and tingling (6.86%), anxiety (5.38%), insomnia (3.43%) and depression (0.49%). Mauro Giovanni Carta, et.al,[10] conducted a study on 222 subjects. All subjects underwent a complete thyroid evaluation and found that 16.6% of the overall sample had an anti-TPO value above the normal range. Subjects with at least one diagnosis of anxiety disorders or mood disorders were positive for serum anti-TPO more frequently than subjects without mood or anxiety disorders. A statistically significant association with anti-TPO+ was found in Anxiety Disorder and Depressive Disorder and concluded that individuals in the community with thyroid autoimmunity may be at high risk for mood and anxiety disorders, similar findings are observed in our study. Thyroid hormones affect myelination, therefore increased levels lead to oxidative damage to the myelin membrane and/or the oligodendroglial cell. In hypothyroidism, muscle contraction and relaxation are slowed down while duration is prolonged. The amount of myosin ATPase decreases. Slowing of release and reaccumulation of calcium in the endoplasmic reticulum may decrease relaxation. In peripheral nerves, segmental demyelination

has been observed with decreased nerve conduction velocities. Patients develop polyneuropathy with loss of reflexes and weakness, decreases in vibration, and touch-pressure sensations are observed. This is the reason why most of the thyroid patients in our study presented with severe numbness and tingling and delayed jerks have been observed.

Effect of thyroid disorders on CVS 14 (6.36%) in our study was found to be significant (X^2=34.47, D.F.=1, P=0.0001, Odds ratio=4.658). Effect on CVS in hyperthyroid and hypothyroid patients was found to be 13 (27.08%) and 1 (0.5%) respectively. Both hyperthyroidism and hypothyroidism produce changes in cardiac contractility, myocardial oxygen consumption, cardiac output, blood pressure, and systemic vascular resistance (SVR). It is well known that hyperthyroidism can produce atrial fibrillation and less well recognized that hypothyroidism can predispose to ventricular dysrhythmias. In almost all cases these cardiovascular changes are reversible when the underlying thyroid disorder is recognized and treated.

The effect of thyroid disorders on hepatic system was found to be significant in our study (X^2=4.113, D.F.=1, P=0.0426, Odds ratio=0). In our study effect of hypothyroidism on hepatic system 15 (8.72%) was found to be predominant. Effect on hepatic cells was evaluated based on increased LDL and altered TG levels. R. Malik, et.al.,[14] conducted a study to evaluate the relationship between the thyroid gland and the liver and found that thyroid hormones increase the expression of LDL receptors on the hepatocytes and increase the activity of lipid-lowering liver enzymes, resulting in a reduction in low-density lipoprotein levels. Thyroid hormones also increase the expression of apolipoprotein A1, a major component of highdensity lipoprotein. Similar observation was found in our study. These fatty changes in the hepatocytes leads to minimal enlargement of liver.

The effect of thyroid disorders on haemopoietic (X^2=0.3424, D.F.=1, P=0.5584, Odds ratio=0.7167) and skeletal system(X^2=1.387, D.F.=1, P=0.2390) was found to be insignificant. In hyperthyroid and hypothyroid patients affect on haemopoietic system was found to be 4 (8.3%), 20(11.62%) respectively. TSH could affect hematopoiesis by binding to a functional thyrotropin receptor (TSHR), which is found in both erythrocytes and some extrathyroidal tissues. T_3 is involved in the control of growth and apoptosis of hematopoietic cells and bone marrow tissue by potentiating the erythroid burst-forming unit (BFU-E) proliferation. T_4 has been shown to exert a direct, β2-adrenergic receptor-mediated stimulation of red cell precursors. The effects of thyroid hormones on erythropoiesis seem to be mediated at the molecular level by the T_3 binding to specific nuclear receptors (the endogenous receptor alpha, c-erbA/TRα) and the closely related retinoic acid receptor α (RARα) involved in the regulation of normal erythroid differentiation. In hypothyroid conditions, reduced EPO levels might also account for anaemia,[18] thus patients complains of Malaise.

The prevalence of effect of hypothyroidism 5(2.9%) on skeletal system was found to be predominant than hyperthyroidism. The effect was evaluated based on occurrence of osteoporosis and immediate fractures of bones. TH play a fundamental role in endochondral ossification, in skeletal development and growth and in the maintenance of bone mass, predominantly through the action of TRα1. The thyroid gland follicles contain a population of parafollicular cells, which synthesize and secrete the hormone calcitonin. As there is thyroid dysfunction, calcitonin deficiency was observed leading to decrease in absorption of calcium by the skeletal system and stimulates bone resorption by osteoclasts, increasing the porosity of cortical bone and reducing the volume of trabecular bone. Thus Net effect was osteoporosis and increased risk of fractures were observed.

CONCLUSION

Thyroid hormone is a critical regulator of growth, development and metabolism in virtually all tissues, and altered thyroid status affects many organs and organ systems. Diagnosing the thyroid disorders in early stages and providing appropriate therapeutic regimen can minimize the effect on other organ systems of the body. Non-adherence to medication worsens thyroid disorders leading to deterious effects on other organ systems of the body. The most affected organ system by thyroid dysfunction is reproductive system. In both hypo and hyperthyroidism menstrual irregularities, infertility and miscarriages in females and oligospermia in males are observed, indicating that the thyroid hormones play an important role in normal reproductive functions. Digestive diseases related to thyroid hormone abnormalities must be recognized in early stages and treatment should be initiated. A bowel movement less than once daily could be a useful pointer for the diagnosis of hypothyroidism.

Early detection of Cardiovascular effects like palpitations, bradycardia and tachycardia caused by thyroid disorders can further prevent Cardio vascular complications like atrial fibrillation leading to heart failure. Similarly effects on Central nervous system, hepatic, haemopoietic and skeletal systems may lead to serious complications. Therefore it is very important that a clinical pharmacist should educate the patient regarding the importance of regular clinic visits, thyroid profile monitoring, adhering to medication regimen and create awareness about physical and functional alterations which occur in different organ systems so that appropriate therapeutic changes may be done to prevent further damage to organ systems and hence provide better health to thyroid disorder patients.

ACKNOWLEDGEMENT

The authors wish to acknowledge the assistance rendered by the department of Pharmacy Practice, Care College of Pharmacy and Samraksha superspeciality hospital, Telangana, India.

REFERENCES

1. Anders Svare, Tom Ivar Lund Nilsen, Bjørn Olav Asvold, Siri Forsmo, Berit Schei, Trine Bjøro and Arnulf Langhammer. Does thyroid function influence fracture risk? Prospective data from the HUNT2 study, Norway. European Journal of Endocrinology. 2013; 169: 845–852.
2. Bahaa M. Fadel, M.D., Samerellahham, M.D., Matthew D. Ringel, M.D., Josephlindsay, JR., M.D., Leonardwartofsky, M.D. Kenneth D. Burman, M.D. Hyperthyroid Heart Disease. Clin. Cardiol. 2000; 23: 402–408.
3. Barrett-Connor E, Weiss TW, McHorney CA, Miller PD & Siris ES. Predictors of falls among postmenopausal women: results from the National Osteoporosis Risk Assessment (NORA). Osteoporosis International. 2009; 20: 715–722.
4. Deepthi Govindan Kutty, Dhanya.C. S, C. I. Sajeeth. Prevalence of Thyroid Disorders in Patients in Two Tertiary Care Hospitals at Palakkad. IOSR Journal of Pharmacy and Biological Sciences. Nov – Dec 2013; 8(3): 33-37.
5. Dr. Firdushi Begum. A Hospital based study on Thyroid Dysfunction based on estimation of TSH & Thyroid Hormones. Sch. J. App. Med. Sci., 2015; 3(8): 3096-3102.
6. Erdogan Mehmet, Kosenli Aybike, Sencer Ganıdagli and Kulaksizoglu Mustafa. Characteristics of anemia in subclinical and overt hypothyroid Patients. Endocrine Journal, 2012; 59(3): 213-220.
7. Ganong WF. The thyroid gland. In: Review of medical physiology, 22nd edition, New York; McGraw-Hill, 2005; 317-332.
8. Gopalakrishnan AU, Menon UV; Thyroid disorders in India: An epidemiological perspective. Indian J of Endocrinol Metab. 2011; 15(2): S78-S81.
9. Hande Turker, Cuneyt Turker and Nilgun Cengiz. Neurological Complications of Hypothyroidism. Hypothyroidism - Influences and Treatments, Dr. Drahomira Springer (Ed.), 2012.
10. Mauro Giovanni Carta, Andrea Loviselli, Maria Carolina Hardoy, Sergio Massa, Mariangela Cadeddu, Claudia Sardu, Bernardo Carpiniello, Liliana Dell'Osso and Stefano Mariotti. The link between thyroid autoimmunity (antithyroid peroxidise autoantibodies) with anxiety and mood disorders in the community: a field of interest for public health in the future. BMC Psychiatry. 2004; 4(25).
11. M Bauer, A Heinz and PC Whybrow. Thyroid hormones, serotonin and mood: of synergy and significance in the adult brain. Molecular Psychiatry. 2007; 140–156.
12. Olga Yaylali, Suna Kirac, Mustafa Yilmaz, Fulya Akin, Dogangun Yuksel, Nese Demirkan and Beyza Akdag. Does Hypothyroidism Affect Gastrointestinal Motility? Gastroenterology Research and Practice. 2009.
13. Rajnish Nagarkar, Shirsendu Roy, Mohammad Akheel, Vijay Palwe, Nayana Kulkarni, Prakash Pandit. Incidence of Thyroid Disorders in India: An Institutional Retrospective Analysis. International Journal of Dental and Medical Specialty. 2015; 2(2): 19-23.
14. R. Malik and H. Hodgson. The relationship between the thyroid gland and the liver. Q J Med, 2002; 95: 559–569.
15. Ronald Daher, Thierry Yazbeck, Joe Bou Jaoude, Bassam Abboud. Consequences of dysthyroidism on the digestive tract and viscera. World Journal of Gastroenterology. 2009; 15(23): 2834-2838.
16. Sinnatamby C S. Thyroid gland. In: Last's Anatomy, Regional and applied10th edition, London; Churchill Living stone: 1999; 330-332.
17. Smith JW, Evans AT, Costal B, Smythe JW. Thyroid hormones, brain function and cognition: a brief review. Neuroscience and Biobehavioral Reviews, 2002; 45-60.
18. Tanveer Hassan Banday, Sadaf Bashir Bhat, Sabreen Bashir Bhat, Naveed Shah, Shahnawz Bashir. To study prevalence of incipient iron deficiency in primary hypothyroidism. Int J Res Med Sci, 2014; 2(2): 472-475.
19. Takehiro Hiraoka, Osamu WadaHiraike, Yasushi Hi rota, Tetsuya Hirata, Kaori Koga, Yutaka Osuga, To moyuki Fujii. The impact of elevated thyroid stimulating hormone on female subfertility. Japan Society for Reproductive Medicine, 2016; 15(2): 121–126.
20. T.Veeresh, D.Moulali, Dr. D.V.H. S Sarma. A Study on Serum FSH, LH and Prolactin Levels in Women with Thyroid Disorders. International Journal of Scientific and Research Publications. 2015; 5(3).
21. Von Recklinghausen FD. Die fibro¨se oder deformierende Ostitis, die Osteomalazie und die osteoplastische Karzinose in ihren gegenseitigen Beziehungen. Festschrift R. Virchow.pp 1–89. Berlin: Georg Reimer Verlag, 1891.