Section: General Medicine

Original Research Article

A STUDY OF LIPID ABNORMALITIES IN PATIENTS WITH SUBCLINICAL HYPOTHYROIDISM

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Abstract

Background: To assess whether subclinical hypothyroidism is associated with abnormal lipid profile and compare it with euthyroid group. **Materials and Methods:** This was a hospital based cross sectional study conducted in 2 years. All adult patients who fit the biochemical criteria for subclinical hypothyroidism were included in the study. **Result:** Incidence of subclinical hypothyroidism is more in females. Being a hospital based study almost all of our patients are symptomatic. Most common symptom found is excessive tiredness and most common sign is goitre. 38(76%) of patients have TSH between 10 and 20 uIU/mL and all these patients are at high risk for progressing to overt hypothyroidism (TSH>10 uIU/mL). Total cholesterol and LDL cholesterol were significantly high in the SCH group when compared to euthyroid controls. There was no much difference between levels of triglycerides and HDL cholesterol in both the groups. High total cholesterol (64%) was the most common finding followed by high LDL (52%) in the SCH group. **Conclusion:** Considering the dyslipidemia, one of the potential CVS risk factors, patients if eligible as per the current recommendations to be started on thyroxine therapy and hypolipidaemic drugs.

INTRODUCTION

Subclinical hypothyroidism (SCH) refers to biochemical evidence of thyroid hormone deficiency in patients who have few or no apparent clinical features. The term subclinical hypothyroidism was originally used to describe slightly elevated serum TSH level (>5 mU/L) in a patient with a normal fT4. Other terms for this condition are mild hypothyroidism, early thyroid failure, preclinical hypothyroidism and decreased thyroid reserve.[1,2]

Although considered an asymptomatic disorder, some patients may present with non-specific symptoms like fatigue, muscle weakness, weight gain, which can be suggestive of hypothyroidism. The prevalence of SCH in the population is relatively high, and it varies from between 4% and 20%. Furthermore, it depends on gender and age, usually occurring more frequently over the age of 60, with a prevalence of around 15% and 8% for women and men, respectively. Its diagnosis should be based on an understanding of geographic and demographic differences in biochemical criteria versus a global reference range for TSH that is based on the 95% confidence interval of a healthy population.[3,4]

The etiologies of subclinical and overt hypothyroidism are identical. Chronic autoimmune thyroiditis (Hashimoto's disease) accounts for the majority of cases. Approximately 54% of patients with subclinical hypothyroidism have Hashimoto's disease with high serum concentrations of antithyroid microsomal or antithyroid peroxidase antibodies.

Thyroid dysfunction has significant public health consequences. Overt thyroid disorder has been widely recognized as being a cardiovascular risk factor, as it is associated with dyslipidaemia, insulin resistance, hypertension, inflammation, oxidative stress, endothelial dysfunction, coagulation disorders and, thus, atherosclerosis. Recent studies suggest that this may also be true for SCH. In fact, a growing number of studies have associated SCH with an increased number of cardiovascular risk factors, including hypertension, weight gain, insulin resistance, dyslipidaemia and ischemic heart disease.[3]

SCH refers to raised serum TSH levels with normal serum-free thyroxine (FT4) levels, which can be mild (TSH <10 mIU/L) or severe (TSH ≥10 mIU/L). SCH patients often present lipid abnormalities, especially elevated total cholesterol (TC) and low- density lipoprotein cholesterol (LDL-C). SCH potentially contributes to a pro- atherogenic lipid profile, with effects being greater at higher TSH levels.[4]

A large epidemiological study found not only raised TC and LDL-C concentrations but also raised high-density lipoprotein cholesterol (HDL-C) and triglyceride (TG) concentrations in SCH subjects

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compared to euthyroid controls. Furthermore, a recent meta-analysis documented that SCH is related to an increased risk of coronary artery disease (CAD) events and mortality. Thus, the control of cholesterol, an independent risk factor of CAD, may benefit SCH patients.[5]

In pregnancy, SCH incidence is more common than overt hypothyroidism, ranging from 15% to 28% in iodine-sufficient regions. Studies have shown an association between SCH in pregnancy and hypertensive disorders of pregnancy, preterm labour and impaired cognitive development of infants, but evidence linking SCH to adverse pregnancy outcomes is still inconsistent and conflicting. Benefits of treatment with thyroxine are uncertain and associated with but a higher risk of premature delivery, diabetes and high blood pressure during pregnancy.

Another important concern is the progression of subclinical hypothyroidism to overt hypothyroidism during its natural history. Risk is high if the TSH is more than 10 uIU/mL or thyroid peroxidase antibody is positive. In the Whickham survey the annual risk of women developing hypothyroidism was 4.3% per year if both an elevated serum TSH and anti-thyroid antibodies were found, 2.6% with elevated TSH alone, and 2.1% per year with positive anti-thyroid antibodies alone. This study mainly focuses on the clinical profile (symptoms and signs), and lipid abnormalities of patients with subclinical hypothyroidism in our setting.

MATERIALS AND METHODS

This was a hospital based cross sectional study conducted at GGH Nagarkurnool October 2021 to September 2023 . All adult patients who fit the biochemical criteria for subclinical hypothyroidism were included in the study. None of the patients were part of a routine screening programme. Newly diagnosed cases of subclinical hypothyroidism patients who fulfilled the inclusion and exclusion criteria were included in the study.

Inclusion Criteria

All patients with age >12 yrs, with subclinical hypothyroidism (normal T3, T4 and with raised TSH >5 mIU/L).[6]

Exclusion Criteria

Hypothyroid patients who are already on thyroxine, Hyperthyroid patients on treatment, Patients with any serious non thyroidal illness, chronic liver disease, chronic renal disease, Pregnancy, comorbidities like diabetes mellitus and hypertension and drugs like beta blockers, diuretics, steroids, OCPs, hypolipidaemic drugs.

Selection of Controls: Controls were taken for comparing the lipid profile of the cases. Euthyroid (normal T3, T4, TSH) population were taken as control

Exclusion Criteria for Controls: Age less than 12 years, Diabetes mellitus, Chronic renal failure, Chronic liver disease, Pregnancy and Who were on drugs like beta blockers, diuretics, steroids, OCP & lipid lowering drugs.

Method of study: The patients in the study group were evaluated with a detailed clinical history, thorough clinical examination and relevant laboratory investigations. The diagnosis of subclinical hypothyroidism was made according to the diagnostic criteria mentioned above. The evaluation aimed to look for the symptoms and signs & lipid abnormalities.

Clinical data comprised of symptom analysis, thorough examination to identify signs, history of past medical illness and surgery, history of drug intake and type of salt used. Laboratory data consisted of complete blood picture, blood sugar, blood urea, serum creatinine, T3, T4, TSH, TPO antibody where required and fasting lipid profile. Blood urea, sugar and serum creatinine were estimated using ERBA XL300 automated analyzer. T3 and T4 was measured by Competitive Chemi Luminescent Immuno Assay and TSH by Ultra Sensitive Sandwich Chemi Luminescent Immuno Assay. Anti TPO antibody estimation was done only in few cases by electrochemiluminescence assay. A value more than 34 IU/L is taken as positive. Fasting lipid profile was done with the ERBA XL300 autoanalyzer. Total cholesterol, Triglycerides and HDL were estimated and LDL was calculated by the Friedwald's equation:

LDL Cholesterol = Total cholesterol - Triglycerides - HDL-C. 5

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using.

Epidemiological Information Package (EPI 2002).

Using this software, range, frequencies, percentages, means, standard deviations, chi square and 'p' values were calculated. Kruskul Wallis chi-square test was used to test the significance of difference between quantitative variables. A 'p' value less than 0.05 is taken to denote significant relationship.

RESULTS

In the present study among cases of 50 patients, the mean age was 41.23 years with standard deviation of 8.52 years, range being 20 to 60 years. Majority of the patients (46%) were in the agegroup 41-50 years. 47 (94%) of the patient's observed were female in cases group and all the patients in control group were female,100%.8.2% of the patients were having normal BMI, followed by 22.7% patients were having underweight and 9.1% of the patients were having overweight. [Table 1]

We found that 64% of the patients among the cases had tiredness, 60% of the patients had weight gain, 50% of the patients had musculoskeletal symptoms, 38% of the patents had cold intolerance, 36% of the patients had constipation, 32% of the patients presented with neck swelling, 25% of the patients had

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symptoms of voice change and very few had other symptoms like depression, menorrhagia and infertility, on examination it was found that 54% of the patients had goiter, 40% of the patients had Dry skin, 20% of the patients had puffy eyes. [Table 2]

Table 1: Demographic distribution compared in both groups

Age group	Cases		Controls	
	No.	%	No.	%
Upto 20 years	2	4	3	6
21-30	8	16	6	12
31-40	7	14	6	12
41-50	23	46	20	40
Above 50	10	20	15	30
Total	50	100	50	100
Mean	41.23		43.6	
SD	8.52		11.3	
Range	20-60		21-66	
P-value	0.517			
Gender distribution				
Male	3	6	0	0
Female	47	94	50	100
p-value	0.078			
BMI				
Underweight (<20)	11	22.7	18	35
Normal (20 - 25)	34	68.2	30	60
Overweight &Obese (> 25)	5	10	2	4
Total	50	100	50	100
Mean	23.3	22.9		
SD	1.52	1.58		
Range	19-26	20-27		
P-value	0.2001			

Table 2: Symptoms and signs in cases

Symptoms	Present	
	No	%
Weight gain	30	60
Tiredness	32	64
Musculoskeletal	25	50
Neck swelling	16	32
Cold intolerance	19	38
Constipation	18	36
Voice changes	13	26
Infertility	3	6
Menorrhagia	9	18
Asymptomatic	0	0
Signs on clinical examination		
Pulse Normal	50	100
Goitre	27	54
Dry Skin	20	40
Puffy Eyes	10	20
Delayed relaxation of ankle jerk	7	14

Table 3: TSH distribution

TSH (uIU/mL)	No	%
< 10	9	18
10-20	38	76
> 20	3	6

Most of our patients had baseline TSH 10-20 uIU/mL.

Table 4: Total cholesterol distribution

Total Cholesterol	Cases		Controls	
	No.	%	No.	%
Normal (< 200)	18	36	48	96
Borderline (201-239)	14	28	2	4
High (> 240)	18	36	0	0
Total	50	100	50	100
Mean	214.3		176.36	
SD	45.2		15.6	
Range	111-346		142-214	
P-value	0.001			

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The mean cholesterol in cases was 214.30 with standard deviation 45.2 and that of in control it was 176.36 with standard deviation of 15.6, so this difference between the groups was statistically significant (p-value = 0.001).

Table 5: LDL distribution

LDL	Cases		Controls	
	No.	%	No.	%
Normal (< 130)	24	48	48	96
Borderline (131-159)	2	4	2	4
High (> 160)	24	48	0	0
Total	50	100	50	100
Mean	141.3		105.6	
SD	32.2		17.2	
Range	53-245		88-175	
P-value	0.001			

The mean LDL cholesterol in cases was 141.3 with standard deviation 32.2 and that of in controls it was 105.6 with standard deviation of 17.2, so this difference between the groups was statistically significant (p-value = 0.001).

Table 6: TGL distribution

TGL	Cases		Controls	
	No.	%	No.	%
Normal (< 150)	36	72	48	96
Borderline (151-199)	8	16	1	2
High (> 200)	6	12	1	2
Total	50	100	50	100
Mean	156		136.24	
SD	74		24.2	
Range	72-412		99-216	
P-value	0.0758			

Mean TGL in cases was 156 with standard deviation 74 and that of in controls it was 136.24 with standard deviation of 24.2, so this difference between the groups was not statistically significant (p-value = 0.07)

Table 7: HDL distribution

HDL	Cases		Controls	
	No.	%	No.	%
Normal (> 40)	34	68	38	76
Low(< 40)	16	32	12	24
Total	50	100	50	100
Mean	47.2		48.93	
SD	12.44		13.21	
Range	22-85		32-75	
P-value	0.5018			

Mean HDL in cases was 47.2 with standard deviation 12.44 and that of controls was 48.93 with standard deviation of 13.21, so this difference between the groups was not statistically significant (p-value = 0.501).

DISCUSSION

Subclinical hypothyroidism is defined as an elevated serum TSH with normal free T4. It is more common condition than overt hypothyroidism. Many studies have shown that patients with subclinical hypothyroidism are not entirely asymptomatic, rather they do have many of the symptoms and signs of overt hypothyroidism like weight gain, fatigue etc. This study was done to find out the lipid abnormalities in patients with subclinical hypothyroidism.

In the present study among cases of 50 patients, the mean age was 41.23 years with standard deviation of 8.52 years, range being 20 to 60 years. Majority of the patients (46%) were in the agegroup 41-50 years. Studies have shown that the incidence of subclinical hypothyroidism increases with age. We had only 10 patients (20%) above the age group50 years. This difference may be due to the fact that those studies were community based screening studies done on large population. So screening in elderly population may be needed to detect more cases as larger studies have shown that majority of the patients are asymptomatic. Among the controls, the mean age was 43.6 years with standard deviation of 11.3 years, range being 21 to 66 years. Majority of the patients (40%) were in the agegroup 41 – 50 years. We had 15 patients (30%) above the age group50 years. Study conducted by N. Karthick et al found that there was no significant difference between the age groups. It was similar to our study results.[7] The Colorado Thyroid Disease Prevalence Study had clearly demonstrated the increase in serum TSH with age. In

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our study also we found that subclinical hypothyroidism increases with increase in age.[8]

In our study it was observed that proportion of female patients was more in cases as well as in controls. 47 (94%) of the patient's observed were female in cases group and all the patients in control group were female,100%. Only 3 patients were males which was only (6%). In many studies it was observed that number of female patients was more. Study conducted by Hiregoudar MB et al observed similar results with our study. In this study also they found that more number of females in both the groups.[9] A study on dyslipidemia in subclinical hypothyroidism by Bandyopadhay and co-workers also reported that 78% of their cases were females. One more study conducted by S. Ashok Kumar et al found more number of female cases, they found that 91.7% of the patients were females and only 8.3% of the patients were males.[10] In our study also 94% of the patients were females and only 6% of the patients were males.

In our study it was observed in cases, 68.2% of the patients were having normal BMI, followed by 22.7% patients were having underweight and 9.1% of the patients were having overweight. Mean BMI was 23.56 with standard deviation of 1.45. Study conducted by N. Karthick et al found that, there was no significant difference observed in BMI between the groups (p-value = 0.199).[7] In a study by Asranna et al,[11] mean BMI among cases was 21.48 ± 2.80 kg/m2 and in the control group it was 21.36 ± 1.53 kg/m2. There was no significant difference in the mean BMI of the two groups (P value: 0.776). S. Ashok Kumar et al,[10] found that majority of the cases around 61% were having normal BMI ranging from 20 to 25 kg/m2.

In our study we found that 64% of the patients among the cases had tiredness, 60% of the patients had weight gain, 50% of the patients had musculoskeletal symptoms, 38% of the patents had cold intolerance, 36% of the patients had constipation, 32% of the patients presented with neck swelling, 25% of the patients had symptoms of voice change and very few had other symptoms like depression, menorrhagia and infertility. In our study, on examination it was found that 54% of the patients had goiter, 40% of the patients had Dry skin, 20% of the patients had puffy eyes. There were no asymptomatic patients. In study conducted by Hiregoudar MB et al,[9] found that 54% of the patients had genialized weakness, followed by weight gain (41%), Constipation (34%), Cold tolerance (33%) as the most common symptoms. The Colorado Thyroid Prevalence Study also revealed that subclinical hypothyroidism patients have symptoms similar to hypothyroidism.[6] In their study of 2,336 subjects who were identified to have mild thyroid failure, significantly reported more often having dry skin (28%; $P < 0.001$), poor memory (24%; $P < 0.001$), slow thinking (22%; $P < 0.001$), muscle weakness (22%; $P < 0.001$), fatigue 18%; $P < 0.01$), muscle cramps (17%; $P < 0.001$), cold intolerance (15%; $P < 0.01$), puffy eyes (12%; $P < 0.05$), constipation (8%;$P < 0.05$), and hoarseness (7%; $P < 0.05$) than did the euthyroid subjects. It is important to note that, where as euthyroid subjects experienced a mean of 12.1% of all the listed symptoms, overtly hypothyroid subjects had 16.6% of these symptoms ($P < 0.05$ vs. euthyroid group), and subjects with mild thyroid failure reported an intermediate 13.7% of the symptoms ($P < 0.05$ vs. euthyroid group). In a study by Haggerty et al,[12] the lifetime frequency of depression was significantly higher in the subjects who met the criteria for subclinical hypothyroidism (56%), than in those who did not (20%), suggesting that subclinical hypothyroidism may lower the threshold for the occurrence of depression. In another study by Sampath et al,[13] 109 patients with depression were tested for thyroid dysfunction. 42.2% of them had subclinical hypothyroidism and 3.6% had overt hypothyroidism. So all patients with depression should have a thyroid function test.

Our study tried to evaluate the causes for subclinical hypothyroidism through history. Most possible important aetiology thought to be was autoimmune thyroiditis. Other causes like post thyroidectomy was not seen and drug induced like SCH secondary to antiarrhythmics like Amiodarone was seen in one patient. Definite cause could not be found. Autoimmune thyroidits is usually diagnosed by doing anti TPO antibody assay. Only 1 patient got the test done had positive result. This result was similar to the study by Shruti Mohanty and team where 45 out of 61 subclinical hypothyroid cases had TPO antibodies suggesting autoimmune thyroiditis as the cause. Published literature states that the most common cause of subclinical hypothyroidism is autoimmune thyroiditis (Hashimoto's disease).[14] Progression to overt hypothyroidismis reported to vary from 3 to 20%, the risks being greater in those patients with TSH more than 10 uIU/mL or thyroid antibodies (or both). Hence it is recommended that anti- TPO measurement should be an integral part of the investigation in subclinical hypothyroidism.

In a study by Ian Louis Ross about thyroid dysfunction in patients on amiodarone, he found that hypothyroidism was the common abnormality and subclinical hypothyroidism was present in 13% of cases.[15] Some studies indicate that the incidence of thyroid dysfunction with amiodarone varies with the dietary iodine intake in the population. Amiodarone Induced thyrotoxicosis occurs more frequently in geographical areas with low iodine intake, whereas Amiodarone induced hypothyroidism is more frequent in iodine-replete areas. Our study population was from an iodine-replete area and amiodarone was indeed not found to be a cause for subclinical hypothyroidism.

In our study we found that, the mean cholesterol in cases was 214.30 with standard deviation 45.2 and that of in control it was 176.36 with standard deviation of 15.6, so this difference between the groups was statistically significant (p-value = 0.001). The mean LDL cholesterol in cases was 141.3 with standard deviation 32.2 and that of in controls it was

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105.6 with standard deviation of 17.2, so this difference between the groups was statistically significant (p-value = 0.001). Mean TGL in cases was 156 with standard deviation 74 and that of in controls it was 136.24 with standard deviation of 24.2, so this difference between the groups was not statistically significant (p-value = 0.07) and mean HDL in cases was 47.2 with standard deviation 12.44 and that of controls was 48.93 with standard deviation of 13.21, so this difference between the groups was not statistically significant (p-value = 0.501).

Study conducted by Asranna, et al,[11] found that the mean total cholesterol levels were significantly higher in patients with SH as compared to controls (173.72 mg/dl vs.150.77 mg/dl, P value = 0.004)69. The mean LDL levels were significantly higher among cases compared to controls (106.07 mg/dl vs. 80 mg/dl, P value < 0.001). The mean HDL was lower in patients with SCH (38.63 mg/dl) as compared to controls (42 mg/dl). However, it was not statistically significant.

Another study conducted by S Ashok Kumar et al,[10] observed that Hypercholesterolemia was found in 40.6% of patients. Among 96% of patients, 39 cases had high total cholesterol values. Borderline high values were found in 18.8 % of patients. Mean cholesterol value of 213 mg/dl ranging from a minimum of 119 to 310 mg/dl. LDL was elevated in 42.7% of cases. Among 96 cases, 41 patients had elevated LDL levels of more than 160 mg/dl. Borderline high LDL values were found in 14.6%. The mean LDL value was 139 mg/dl ranging from a minimum of 76 to 290 mg/dl. Triglyceride was elevated in only 27.1%. Among 96 cases, only 26 cases had high triglyceride values. Mean triglyceride value of 121.3 mg/dl ranging from a minimum of 76 to 222 mg/dl. HDL values were found to be normal in 66.7% of cases. The mean HDL was 51.9 mg/dl ranging from a minimum of 33 to 70 mg/dl.

This result was similar to the observations made in The Colorado Thyroid Prevalence Study where they found statistically significant elevation in total cholesterol and LDL in subclinical hypothyroid cases compared with euthyroid controls. Another study done in India by Guptha and Sinha,[16] also demonstrated a significant elevation in serum cholesterol in subclinical hypothyroid cases when compared with euthyroid controls. Same results were obtained in the study by Walsh J P & colleagues.[17]

A recent meta-analysis of the effect of treatment with thyroxine on lipid profile in mild thyroid failure cases by Mark D. Danese and colleagues,[18] has demonstrated a mean reduction in the total cholesterol level of 7.9 mg per deciliter (0.2 mmol per liter) and inthe LDL cholesterol level of 10 mg per deciliter (0.26 mmol per liter). Changes in high-density lipoprotein (HDL) cholesterol wereheterogeneous among the studies and were not statistically significant.

The results of the present study were similar to the study conducted by N.Karthick et al,[7] where the study group of females with SCH had high total cholesterol and LDL levels which were statistically significant. The study also had highly significant triglyceride levels in the study group. However in the present study the difference between the triglycerides of both the groups was not statistically significant.

Studies conducted by Z Efstathiadou et al and MIshal Ejaz et al,[19] also concluded that SCH group was found to have high total cholesterol and high LDL cholesterol similar to the present study with (TC, $p<0.05$ and LDL, $p<0.01$) and $p<0.00001$ respectively .In the follow up Mishal Ejaz et al found that the high cholesterol and LDL levels got reduced significantly with treatment using thyroxine where as study by Z Efstathiadou et al,[19] showed no improvement with thyroxine supplementation 50,71. Study by Nadia Caraccio et al,[20] showed specific and reversible increase of LDL cholesterol ($p=0.01$) in SCH patients similar to our study. But with levothyroxine supplementation there was a significant decrease in both Total cholesterol ($p<0.01$) and LDL ($p = 0.01$).

Present study found significant incidence of goitre and subclinical hypothyroidism in a population where all the study group was on iodised salt. Iodine is essential for thyroid function. Thyroid disorders related to iodine deficiency have decreased progressively with iodine prophylaxis and the increased overall iodine intake. Acute excess iodine ingestion has long been known to result in a transient decrease in iodine organification, termed the acute Wolff-Chaikoff effect. With sustained excess iodine exposure, however, most individuals' thyroid glands escape from this acute Wolff-Chaikoff effect despite the exposure and resume synthesis of normal amounts of T4 andT3. The mechanism responsible for this escape or adaptation to the iodine load probably involves a decrease in the Na+/I- symporter protein, resulting in a decrease in thyroid iodide content. In some individuals this escape phenomenon does not occur, and those patients develop iodine-induced hypothyroidism. Such hypothyroidism generally is reversible when the source of excess iodine exposure is removed.

Another adverse effect resulting from iodine prophylaxis may bethe induction of thyroid autoimmunity. Iodine and iodine containing drugs can precipitate autoimmune thyroiditis in susceptible populations.[2] Most common cause of subclinical hypothyroidism in our study was also thought to be autoimmune thyroiditis. A cross- sectional survey of 102 Peace Corps volunteers in Nigeria, West Africa, in 1998 had demonstrated a high rate of thyroid dysfunction and goitre attributable to excess iodine from their water filters. The study showed that during prolonged excess iodine exposure, there was a marked increase in serum total iodine concentrations and the prevalence of goitre, elevated serum TSH values, and elevated serum thyroid peroxidase antibody values also increased. The prevalence of all abnormalities decreased after removal of excess iodine from the drinking water system. Thyroid autoimmune diseases are complex, polygenic

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afflictions, the penetrance of which is heavily dependent on various environmental influences. In general, iodine deficiency attenuates, while iodine excess accelerates autoimmune thyroiditis in autoimmune prone individuals.

In the present scenario of the post–iodination status in India, the high prevalence of subclinical hypothyroidism is significant as similar findings were reported from other countries, stating subclinical hypothyroidism is more prevalent and marked in subjects consuming excessive amounts of iodine. Excessive iodine intake should be considered an etiology of subclinical hypothyroidism in addition to chronic thyroiditis in these areas.

Limitations

1. The main limitations of this study are the limited number of study participants, both the cases and the controls.
2. All the participants were from 1 center rather than multiple centres.
3. Due to financial constraints TPO antibody could not be assessed in all the cases.
4. Only cases who presented to GGH Nagarkurnool were studied. Screening of the general population was not done.
5. Follow-up study of SCH patients to assess the progression to overt hypothyroidism was not possible.

CONCLUSION

Subclinical hypothyroidism is defined as an elevated serum TSH level associated with normal total or free T4 and T3 levels. This is a much more common disorder than overt hypothyroidism. Subclinical hypothyroidism was more common in females in our study. Being a hospital based study majority of our patients were having symptoms. We tend to miss those patients who are entirely asymptomatic. Screening of high risk populations may help to detect those cases. The most common symptom was excessive tiredness, weight gain and musculoskeletal complaints like myalgia and arthralgia. Goitre was the most common sign on physical examination. Most of our patients had risk factor for progression to overt hypothyroidism like baseline TSH > 10 uIU/mL. The observation of increased incidence of SCH in an iodine sufficient population is supporting the hypothesis that thyroid autoimmunity can be initiated by iodine in susceptible individuals. Depression and infertility may be associated with subclinical hypothyroidism and screening of such individuals may be useful. Compared with euthyroid controls, cases with subclinical hypothyroidism had significantly elevated total cholesterol and LDL-C levels. Considering the dyslipidemia, one of the potential CVS risk factors, patients if eligible as per the current recommendations to be started on thyroxine therapy and hypolipidaemic drugs.

It is very important to follow up both the untreated and the treated cases of SCH for evidence of progression to overt hypothyroidism and for assessing the benefit of thyroxine on different aspects of subclinical hypothyroidism namely the effect on symptoms, reduction in goitre size, improvement in lipid abnormalities and also to avoid over treatment respectively.

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