Original Research Article

Incidence of sub clinical thyroid dysfunction among asymptomatic adult population

Suhail Bin Ahmed[1], Ather Akhtar Pasha[1*], Yogita Singh Thakur[1]

[1]Department of Medicine, Deccan College of Medical Sciences, Hyderabad, Telangana, India
[*]Corresponding author email: **atharpasha@yahoo.com**

International Archives of Integrated Medicine, Vol. 4, Issue 11, November, 2017.

Available online at http://iaimjournal.com/
ISSN: 2394-0026 (P) **ISSN: 2394-0034 (O)**
Received on: 26-10-2017 **Accepted on:**04-11-2017
Source of support: Nil **Conflict of interest:** None declared.
How to cite this article: Suhail Bin Ahmed, Ather Akhtar Pasha, Yogita Singh Thakur. Incidence of sub clinical thyroid dysfunction among asymptomatic adult population. IAIM, 2017; 4(11): 264-268.

Abstract

Background: Patients with subclinical thyroid dysfunction are universally encountered in routine clinical practice. Advanced diagnostic techniques have created new categories of thyroid disorders such as subclinical hypo-and-hyperthyroidism. The management of subclinical thyroid dysfunction is controversial. Patients with subclinical thyroid dysfunction may have vague, nonspecific symptoms that do not aid the clinical apperception. This study aimed to screen the normal adult population for the incidence of subclinical thyroid dysfunction and discuss the optimal management strategy.

Materials and methods: Four hundred subjects with no clinical evidence of thyroid dysfunction were included in the present study. Elaborate history in the form of a symptom questionnaire was obtained and clinical examination was performed. Laboratory analysis of thyroid function was done by electro-chemiluminescence immunoassay (ECLIA). Patients with normal free thyroxine (FT4) and tri-iodothyronine levels (T3) were further classified into subclinical hypo/hyperthyroid based on the serum thyroid-stimulating hormone (TSH) levels. The incidence of subclinical thyroid disorder in the sample population was detected and optimal management strategies were followed as per the European thyroid association (ETA) guidelines.

Results: The normal TSH value by ECLIA was 0.27 - 4.2µIU/ml. Seventeen (4.25%) out of four hundred subjects included in the present study were found to have subclinical thyroid dysfunction. The Ratio of subclinical hypothyroid cases to subclinical hyperthyroid cases was found to be 12:5. Clustering of the cases was found around the age of 60 years and was significantly more common among females in comparison to males. Cases with subclinical thyroid dysfunction were managed by follow up after a thorough evaluation and treatment of other comorbid conditions.

Conclusions: The study provides valuable insight towards understanding the epidemiology and management of subclinical thyroid disorders in the present scenario. Screening is recommended for a

high-risk population since there is good evidence that subclinical thyroid dysfunctions may be associated with progression to overt disease in up to 5% of the population.

Key words

Subclinical, Hypothyroidism, Hyperthyroidism, Management.

Introduction

Subclinical thyroid dysfunction is defined as an abnormal level of serum thyroid-stimulating hormone (TSH) with normal free thyroxine (FT4) and triiodothyronine levels (T3) [1]. A study published by the American Academy of family physicians showed the prevalence of subclinical hypothyroidism as 4 to 8.5 percent and may be as high as 20 percent in women older than 60 years. Subclinical hyperthyroidism is found in approximately 2 percent of the population [2]. In the Indian scenario there is paucity of data regarding the prevalence, therapeutic management and screening guidelines of the condition [3].

Etiology of subclinical thyroid dysfunctions can be assessed by careful clinical evaluation. The causes for low serum TSH concentration include pregnancy, euthyroid sick syndrome or medications such as dopamine, glucocorticoids and dobutamine [4]. Serum TSH may be elevated in patients with nonfunctioning pituitary adenomas, central hypothyroidism, adrenal insufficiency and primary subclinical hypothyroidism [5-7].

The consequences of subclinical hypothyroidism include progression to overt hypothyroidism, atherosclerosis, coronary heart disease (CHD) and mortality by heart failure [8]. Some studies have recorded cognitive impairment in subjects with subclinical hypothyroidism that normalized after thyroxine replacement [9]. Subclinical hyperthyroidism may progress to overt hyperthyroidism, atrial fibrillation and reduced bone mineral density [2].

Most of the management guidelines recommend rechecking serum TSH, FT4, T3 and thyroid peroxidase antibodies within 3–6 months [3, 10]. This therapeutic stake is not to improve the clinical status of patients but to prevent the progression towards adversities.

Materials and methods

Present study was a cross sectional observational study that was conducted in the department of medicine of a tertiary care hospital between January 2016 and February 2017 in Hyderabad, Telangana. Four hundred asymptomatic subjects of age ≥ 18 years, with no clinical history of thyroid dysfunction were included in the study. Patients with known family history of thyroid dysfunctions, overt thyroid dysfunction on medication and newly diagnosed cases of obvious thyroid dysfunction were excluded from the study. Written informed consent was obtained from all subjects included in our study.

Detailed history was taken in the form of a symptom questionnaire. Complete clinical evaluation by physical examination was conducted to look for the thyroid enlargement, skin texture, weight loss/gain, pedal edema and ocular manifestations of thyroid dysfunctions. Electrocardiogram (ECG), plasma glucose, total cholesterol (TCHOL), high-density lipoprotein cholesterol (HDL-C), triglyceride levels and Mean Arterial blood Pressure (MAP) were recorded. Fasting venous blood sample was drawn from all subjects with aseptic precautions and serum/plasma samples were used to study the thyroid function tests (T3, FT4 and TSH). Thyroid function test, (TST) was based on the principle of electro-chemiluminescence.

Statistical analysis of the data was performed by SPSS statistical software (version 22) and statistical significance was calculated by percentage analysis and p-value interpretation. *P* values > 0.05 was considered insignificant and

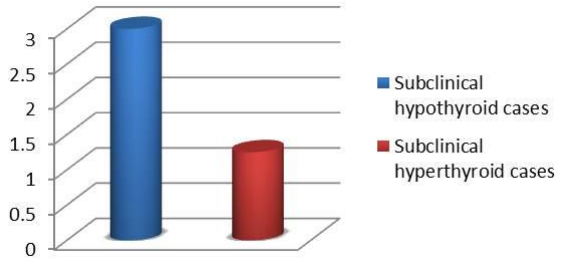
values ≤ 0.05 was regarded as significant.

Results

Four hundred healthy subjects with no clinical history of thyroid dysfunction of age ≥ 18 were included in this study. Seventeen (4.25%) out of four hundred subjects included in the present study were found to have subclinical thyroid dysfunction.

Normal TSH values ranged from 0.27 - 4.2µIU/ml. Those cases with TSH > 4.2µIU but <10µIU/ml with normal T3, FT4 were considered subclinical hypothyroid subjects and those with TSH < 0.27µIU/ml with normal T3, FT4 were taken as subclinical hyperthyroid subjects. Twelve (3%) of the subjects had subclinical hypothyroidism with TSH > 4.2µIU/ml but < 10 µIU/ml (P value < 0.05) and five (1.25%) out of four hundred subjects had subclinical hyperthyroidism (**Figure - 1**).

Figure - 1: Distribution subclinical thyroid dysfunction cases.



Demographic data analysis showed an increased clustering of the cases around the age of 60 years as depicted in **Figure - 2**. Among the Seventeen cases of subclinical thyroid dysfunction eleven (64.70%) were females and six (35.29%) were males. Out of the Seventeen cases eight (47.05%) were absolutely asymptomatic (p value <0.05). The remaining cases presented with co-morbid clinical manifestations such as altered blood glucose profile, lipid profile, mean arterial pressure, pedal edema and ECG changes as depicted in **Figure - 3**. Medical treatment was initiated for hypertensive, diabetic patients, patients with altered lipid profile, ECG changes and pedal edema.

Figure - 2: Age distribution of the cases.

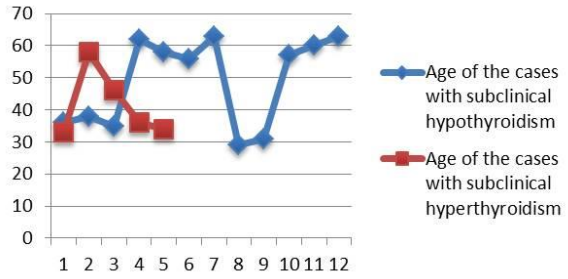
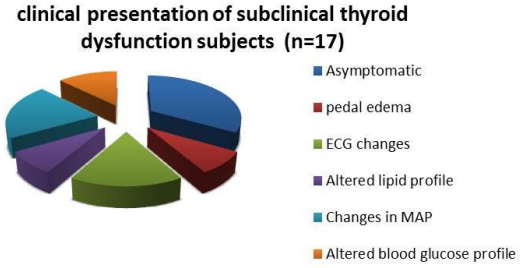


Figure - 3: Clinical presentation of the cases with subclinical thyroid disorders.



Based on ETA guidelines [11], patients with subclinical hypothyroidism were asked to repeat the thyroid profile along with thyroid peroxidase antibodies after 3 months. None of the patients progressed to overt hypothyroidism in the first quarter, however one patient (8.3%) was positive for thyroid peroxidase antibodies with TSH= 9µIU/ml and normal T3, FT4. All patients were put on yearly follow up at the end of the study period.

Risk versus benefit assessment was done in subclinical hyperthyroid patients. Since all the patients had borderline low TSH levels they were put on follow up after confirming the absence of co-morbidities such as atrial fibrillation and osteoporosis.

Discussion

Subclinical thyroid dysfunctions are diagnosed when peripheral thyroid hormone levels are within normal reference laboratory range but serum TSH levels are deranged. Thyroid hormone homeostasis is regulated as part of a negative feedback control circuit within the hypothalamic-pituitary-thyroid axis [12, 13].

The incidence of subclinical hypothyroidism was 3% in comparison studies by Deshmukh, et al. [3] showed a prevalence of 11.5% in Mumbai. A study by the National Health and Nutrition Examination Survey [14] (NHANES III) of an unselected U.S population found subclinical hypothyroidism in 4.3% of the reference population.

Present study is in agreement with studies by Hoogendoorn, et al. [15] and Somwaru, et al. [16], which show that advancing age has direct correlation with the cases. Most of the studies [17, 18] show an increased female preponderance similar to our study. Therefore variables such as sex, age, race/ethnicity and geographic location clearly influence the presence of subclinical hypothyroidism in a given population.

Coming to subclinical hyperthyroidism, the present study gives an incidence of 1.25%. Studies by Marqusee, et al. [19] have shown a prevalence range of 0.7 and 12.4%. Similar to subclinical hypothyroidism findings, advancing age and female sex seems to have a higher association with subclinical hyperthyroidism.

The criteria for treatment of subclinical thyroid disorders are controversial and hence an individualized judgment is mandatory in order to evaluate the grade and the clinical consequences of the condition in a given patient [20, 21]. Present study based its treatment regimen on ETA recommendations [11, 22]. Factors such as absence or presence of anti-thyroid antibodies, cardiovascular risk factors and other co-morbidities influence treatment decisions. Minimal variations of TSH along with absence of other overt risk factors do not necessarily merit treatment, in such situations active surveillance is advised.

Conclusion

The study highlights the association of various variables such as age, sex and geography with the epidemiology of subclinical thyroid disorders. Screening of high-risk cases helps in early identification and thereby reduces the associated mortality and morbidity. Treatment of subclinical thyroid disorders in the present scenario is based on the evidence that subclinical thyroid dysfunctions may be associated with progression to overt disease. Hence individualized treatment and regular follow up is strongly recommended in the management of subclinical thyroid disorders.

Limitations of the study

Since the study period lasted for a year the follow up of the cases could not be documented in this study.

References

1. Douglas S. Ross subclinical hypothyroidism. In: Braverman LE, Utiger RD, editors. Werner and Ingbar's The Thyroid: A fundamental and clinical text. 8th ed. Philadelphia: Lippincott Williams and Wilkins; 2000, p. 1001–6.
2. George R. Wilson, R. Whit Curry, JR. Subclinical Thyroid Disease. American Family Physician, October 2005; 72(8): 1517-24.
3. Deshmukh V, Behl A, Iyer V, Joshi H, Dholye JP, and Varthakavi PK. Prevalence, clinical and biochemical profile of subclinical hypothyroidism in normal population in Mumbai. Indian J Endocrinol Metab., 2013 May-Jun; 17(3): 454–459.
4. Surks MI, Ortiz E, Daniels GH, Sawin CT, Col NF, Cobin RH, et al. Subclinical thyroid disease: scientific review and guidelines for diagnosis and management. JAMA, 2004; 291: 228-38.
5. Persani L, Ferretti E, Borgato S, Faglia G, Beck-Peccoz P. Circulating thyrotropin bioactivity in sporadic central hypothyroidism. J Clin Endocrinol Metab., 2000; 85(10): 3631–5.
6. Abdullatif HD, Ashraf AP. Reversible subclinical hypothyroidism in the

presence of adrenal insufficiency. Endocr Pract., 2006; 12(5): 572–5.
7. Stryker TD, Molitch ME. Reversible hyperthyrotropinemia, hyperthyroxinemia, and hyperprolactinemia due to adrenal insufficiency. Am J Med., 1985; 79(2): 271–6.
8. Mark Chung Wai, Yu Xian Loo, Zhi Min Poon. Subclinical thyroid disorders: Clinical Significance and when to treat? Proceedings of Singapore Healthcare, 2014; 23(3): 226-240.
9. Correia N, Mullally S, Cooke G, Tun TK, Phelan N, Feeney J, et al. Evidence for a specific defect in hippocampal memory in overt and subclinical hypothyroidism. J Clin Endocrinol Metab., 2009; 94(10): 3789–97.
10. Cooper DS, Biondi B. Subclinical thyroid disease. Lancet, 2012; 379(9821): 1142–54.
11. Simon H.S. Pearce, Georg Brabant, Leonidas H. Duntas, Fabio Monzani, Robin P. Peeters, Salman Razvi, Jean-Louis Wemeau. 2013 ETA Guideline: Management of Subclinical Hypothyroidism. Eur Thyroid J., 2013; 2: 215–228.
12. Braverman LE, Cooper DS. Werner & Ingbar's The Thyroid: A Fundamental and Clinical Text, 10th edn. Philadelphia, PA: Lippincott Williams & Wilkins, 2013.
13. Dietrich JW, Landgrafe G, Fotiadou EH. TSH and thyrotropic agonists: key actors in thyroid homeostasis. J Thyroid Res., 2012; 2012: 351864.
14. Hollowell JG, Staehling NW, Flanders WD, Hannon WH, Gunter EW, Spencer CA, et al. Serum TSH, T4, and thyroid antibodies in the United States population (1988 to 1994): National Health and Nutrition Examination Survey (NHANES III). J Clin Endocrinol Metab., 2002; 87(2): 489–99.
15. Hoogendoorn EH, Hermus AR, de Vegt F, et al. Thyroid function and prevalence of anti-thyroperoxidase antibodies in a population with borderline sufficient iodine intake: influences of age and sex. Clin Chem., 2006*;* 52*:* 104–11.
16. Somwaru LL, Rariy CM, Arnold AM, Cappola AR. The natural history of subclinical hypothyroidism in the elderly: the cardiovascular health study. J Clin Endocrinol Metab 2012*;* 97*:* 1962–9.
17. Bensenor IM, Goulart AC, Lotufo PA, et al. Prevalence of thyroid disorders among older people: results from the Sao Paulo Ageing & Health Study. Cad Saude Publica, 2011*;* 27*:* 155–61.
18. Asvold BO, Bjoro T, Vatten LJ. Associations of TSH levels within the reference range with future blood pressure and lipid concentrations: 11-year follow-up of the HUNT study. Eur J Endocrinol., 2013*;* 169*:* 73–82.
19. Marqusee E, Haden ST, Utiger RD. Subclinical thyrotoxicosis. Endocrinol Metab Clin North Am., 1998; 27(1): 37–49.
20. Surks MI, Ortiz E, Daniels GH, Sawin CT, Col NF, Cobin RH, et al. Subclinical thyroid disease: scientific review and guidelines for diagnosis and management. JAMA, 2004; 291(2): 228-38.
21. Gharib H, Tuttle RM, Baskin HJ, Fish LH, Singer PA, McDermott MT. Subclinical thyroid dysfunction: a joint statement on management from the American Association of Clinical Endocrinologists, the American Thyroid Association, and the Endocrine Society. J Clin Endocrinol Metab., 2005; 90(1): 581-5.
22. Biondi B, Palmieri EA, Klain M, Schlumberger M, Filetti S, Lombardi G. Subclinical hyperthyroidism: clinical features and treatment options. Eur J Endocrinol., 2005; 152(1): 1-9.